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Review Article

# Cardiovascular screening of athletes

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A B S T R A C T

The sudden death of an athlete causes grave concern among the medical, sporting, and lay communities, considering athletes epitomise the healthiest segment of society. Most decedents are asymptomatic, therefore screening to identify vulnerable individuals seems appropriate, particularly since most aetiologies can be detected during life and several therapeutic interventions can be implemented to minimise the risk. Given the diversity of conditions implicated in exercise related SCD, no single test will detect all disorders. Furthermore, the low incidence of SCD allows for a cost-effective approach using the simplest and most readily available tools. The 12-lead electrocardiogram has emerged as the most effective tool for detecting electrical diseases and raising suspicion of cardiomyopathy. The international recommendations for ECG interpretation allows for physiological remodelling, substantially reducing false positive rates. Nonetheless, the challenge remains, since the ECG will fail to identify up to 20 % of diseases implicated in young sudden cardiac death and is of limited value in middle-aged and older athletes, in whom atherosclerotic coronary artery disease dominates. Therefore, mitigation of risk extends beyond screening to encompass timely resuscitation, universal defibrillator access, and education in cardiac awareness. The future of screening for cardiovascular disease in athletes is likely to combine traditional evaluation with artificial intelligence, including the use of wearable monitoring, and equal access for effective screening worldwide.

## 1. Introduction

Before considering the role and design of cardiovascular screening in athletes, it is essential to understand the rationale for such programmes. Screening is predicated on the recognition that sudden cardiac death (SCD), while infrequent, is the most catastrophic event in sport, and that early identification of underlying structural, electrical, or genetic conditions, whether predisposing to malignant arrhythmias or to progressive heart failure, offers the potential to prevent tragic outcomes and alter disease trajectories.

Sudden cardiac death in young athletes, although rare, affects between 1 in 15,000 to 1 in 100,000 athletes and remains one of the most devastating outcomes in sports medicine [1,2]. These events, often occurring in otherwise healthy adolescents and young adults during peak exertion, are not only emotionally traumatic but also clinically perplexing. Each case represents a profound loss of life years and casts a long shadow across families, teams, and sporting communities, especially when high-profile athletes are affected.

The epidemiological profile of SCD in athletes is characterised by a mean age of death around 19 years, with a marked male predominance (male-to-female ratio of 9:1) and significantly increased risk among athletes of African and Afro-Caribbean origin (black athletes), where the incidence may be up to five times higher than in their white counterparts [1]. Certain sports, particularly those involving high-intensity, stop-start dynamics such as football, basketball, and soccer, appear to carry a greater intrinsic risk. Strikingly, more than 80 % of individuals who succumb to SCD are asymptomatic prior to the event, highlighting the limitations of symptom-based screening and the need for more proactive strategies [2].

This review aims to examine current evidence surrounding the epidemiology, causes, and mechanisms of SCD in both young and older athletes, with a particular focus on the role of cardiovascular screening. It will explore the strengths and limitations of existing screening protocols, including history, examination, and electrocardiography (ECG), as well as novel risk indicators such as ethnic considerations and post-infection myocardial changes. Additionally, the review will extend its scope to address coronary artery disease (CAD)-related SCD in master athletes, where the underlying pathophysiology and optimal screening approaches differ considerably.

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## 2. Causes and complexity of sudden cardiac death in athletes

The aetiology of SCD in young athletes is multifactorial and complex, encompassing a wide array of structural, electrical, and congenital cardiac abnormalities. These include both inherited cardiomyopathies such as hypertrophic cardiomyopathy (HCM), arrhythmogenic right ventricular cardiomyopathy (ARVC), and dilated cardiomyopathy (DCM), as well as congenital coronary artery anomalies, aortopathies such as Marfan syndrome, valvular disorders, acquired myocarditis, and primary electrical diseases including channelopathies and Wolff–Parkinson–White syndrome [2].

Data from autopsy-based registries and national surveillance studies, such as those reported by Maron et al. highlighted cardiomyopathies accounting for the largest proportion of exertion-related SCD in athletes, with HCM alone responsible for approximately 36 % of cases. Congenital coronary artery anomalies follow, implicated in roughly 17 % of deaths, particularly in younger athletes under the age of 25. Additional causes include myocarditis (6 %), idiopathic left ventricular hypertrophy (8 %), arrhythmic syndromes and valvular heart diseases such as mitral valve prolapse and aortic stenosis [1]. This highlights the diverse spectrum of inherited, congenital, and electrical abnormalities, underlining the difficulty to for screening for SCD and that no single test alone can identify all these abnormalities. The underlying causes of SCD in athletes are heterogeneous and vary according to geography, age, and population risk. Whereas in US registries, hypertrophic cardiomyopathy (HCM) accounts for up to one-third of cases, studies in Italian cohort's report arrhythmogenic right ventricular cardiomyopathy (ARVC) as the most common cause of SCD in young athletes, representing 16–23 % of all cases [3,4].

A UK regional autopsy study involving 357 consecutive athletes who died suddenly (mean age 29 ± 11 years; 92 % male; 69 % competitive) revealed further insights: 42 % of deaths were attributed to sudden arrhythmic death syndrome (SADS), a of exclusion when the heart is structurally normal but presumed to reflect underlying electrical disease. Arrhythmogenic right ventricular cardiomyopathy accounted for 16 % of cases, idiopathic left ventricular hypertrophy or fibrosis 13 %, HCM 12 %, myocarditis 6 %, and coronary artery anomalies and atheroma combined for 7 %. Only 2 % of deaths were from dilated cardiomyopathy, and 13 % from other causes [5] (Fig. 1).

Crucially, this study confirmed that the heart appeared structurally normal in 42 % of SCD cases. Our experience of screening first-degree relatives of decedents from SADS has identified an underlying ion channel disorder in 42 % of cases [6]. This finding strongly suggests that electrical disorders may play a more prominent role in the pathogenesis of SCD in athletes than previously recognised. Highlighting the potential of identifying such disorders early through screening may prevent SCD. A sudden death of an athlete is unique in the sense that it is often a visible event which resonates beyond the sporting community and is amplified by the publicity and media that surrounds it. Combined with the number of life years lost in a young individual, from quiescent yet sinister detectable disease, has resulted in and fuelled several preventive initiatives, including mandatory pre-participation evaluations in organised sport, voluntary community screening programmes, and cascade testing of relatives following a confirmed diagnosis in an index case.

Whilst no single strategy has been devised to eliminate risk completely, early identification through targeted screening alongside athlete education, symptom vigilance, and widespread access to automated external defibrillators forms a layered approach aimed at reducing the burden of SCD in the young athletic population [7].

## 3. Current screening strategies in place for athletes

The aetiology of SCD in young athletes encompasses a broad spectrum of inherited cardiomyopathies, congenital cardiac anomalies, primary electrical disorders and acquired diseases. This heterogeneity presents a fundamental challenge: as no single screening modality can reliably identify all individuals at risk. Furthermore, given the relatively low incidence of SCD, any proposed screening programme must balance sensitivity and specificity with considerations of cost-effectiveness, accessibility and scalability.

Internationally, two principal models have emerged. The American

| | Corrado et 2003 | Maron et 2003 | Harmon et 2015 | Finocchiaro et 2016 |
|---|---|---|---|---|
| HCM | 2% | 36% | 8% | 6% |
| SUD/SAD | - | - | 25% | 42% |
| ARVC | 23% | 4% | 5% | 13% |
| CAD | 19% | 3% | 9% | 2% |
| Anomalous CAD | 14% | 17% | 11% | 5% |
| Myocarditis | 10% | 6% | 9% | 2% |
| Ion Channel disorders | - | 4% | - | - |
| LAD Bridge | 4% | 3% | - | - |
| Aortic dissection/rupture | 2% | 3% | 5% | - |
| Possible HCM / Idiopathic LVH | - | 8% | - | 16% |
| Mitral valve prolapse | 12% | 4% | - | - |
| WPW/conduction disorders | 10% | 2% | 3% | - |
| DCM | 2% | 2% | 3% | 1% |
| Others | 2% | 5% | 5% | 12% |

Not documented
0-2%
3-6%
7-14%
15-20%
>20%

**Fig. 1. Heat Map Depicting Causes of Sudden Cardiac Death in Athletes Across Four Landmark Studies** Abbreviations: ARVC = arrhythmogenic right ventricular cardiomyopathy; CAD = coronary artery disease; DCM = dilated cardiomyopathy; HCM = hypertrophic cardiomyopathy; LAD = left anterior descending; LVH = left ventricle hypertrophy; SAD = sudden arrhythmic death; SUD = sudden unexplained death; WPW = wolff- Parkinson-white.

Heart Association (AHA) endorses a 14-point evaluation centred on personal and family history combined with physical examination [8], reflecting concerns regarding cost and the potential for false positives. In contrast, the European Society of Cardiology (ESC) advocates the inclusion of a 12-lead ECG, recognising its superior diagnostic yield in detecting electrical and structural cardiac disease [9]. Divergence in these strategies reflects differing interpretations of the trade-off between sensitivity, specificity, and resource allocation. Ultimately, responsibility for implementation lies with governing sporting bodies, whose policies vary according to regional healthcare infrastructure and cultural attitudes towards risk tolerance in sport.

ECG screening has demonstrated a sensitivity of up to 94 % for the detection of high-risk cardiovascular conditions, far surpassing the diagnostic performance of history (20 %) and physical examination alone (9 %). This distinction is clinically critical, as the majority of athletes harbouring potentially lethal conditions are asymptomatic and display unremarkable findings on examination. Consequently, reliance on history and physical assessment alone risks leaving the vast majority of at-risk individuals unidentified [8,10,11].

Beyond its superiority in detecting electrical disorders such as channelopathies, the 12-lead ECG has also demonstrated strong utility in the identification of cardiomyopathic conditions, most notably hypertrophic cardiomyopathy (HCM). Evidence from the Veneto region of Italy, where 33,735 competitive athletes underwent systematic evaluation, revealed 22 cases of HCM. Importantly, nearly three-quarters of these diagnoses were prompted by abnormal ECG findings rather than by clinical history or auscultatory signs such as a cardiac murmur. These data reinforce the role of ECG as a first-line screening tool, capable of detecting silent but potentially life-threatening cardiomyopathies that would otherwise escape conventional clinical assessment [11].

Past reservations relating to the routine utility of the 12-lead ECG during pre-participation screening in young athletes are primarily based on the high false positives from the original 2010 European Society of Cardiology recommendations for ECG interpretation [12], especially in black athletes. The 2017 International Recommendations for ECG Interpretation in Athletes represented a major step in enhancing diagnostic precision. By refining the criteria to account for age and ethnicity and demoting investigation of non-specific ECG patterns such as axis deviation and voltage criteria for atrial enlargement, these guidelines substantially reduced the false positive rate to 3 % while maintaining a high sensitivity (92 %) for clinically significant disease. As a result, the current positive predictive value of an abnormal ECG is approximately 17 %, indicating nearly one in five athletes with an abnormal ECG will harbour a serious cardiac condition [13].

## 4. Specific diagnostic challenges with ECG screening

The ECG is inexpensive, universally available as a bedside investigation, and uniquely capable of detecting both electrical disorders and structural cardiomyopathies with electrical expression. Although it is the cornerstone of screening among elite sporting organisations worldwide, it does have its limitations. Several important causes of SCD, such as anomalous coronary arteries, myocarditis [14], commotio cordis, and ion channelopathies, such as catecholaminergic polymorphic ventricular tachycardia and the concealed form of Brugada syndrome, may escape detection on resting ECG.

Additionally, incomplete or evolving phenotypic expression of cardiomyopathies can lead to false negatives. This limitation was underscored by a large British study of 11,000 adolescent footballers affiliated with the English Football Association (FA), in which ECG and echocardiography were used as initial tools. While 93 % were cleared at baseline, 7 % required additional evaluation, and 0.4 % were ultimately diagnosed with serious cardiac disease, predominantly cardiomyopathy. Notably, the ECG proved to be the most effective first-line modality, with a diagnostic yield of 86 %, far exceeding that of history (7 %), physical examination (4 %), and echocardiography (29 %). Over a 10-year follow-up period, there were 8 deaths from cardiac disease, of which only 2 were detected during the initial screening episode. The mean period from initial screening to death from cardiomyopathy was over 6 years, indicating that a negative test in adolescence does not exclude the risk of cardiomyopathy in later life [15].

An Italian longitudinal study involving 15,127 adolescent athletes over an 11-year period demonstrated that repeated evaluations increased diagnostic yield more than threefold, particularly for conditions such as myocardial fibrosis and scar tissue that evolve gradually. Each athlete underwent a mean of four screening assessments, with 0.4 % ultimately diagnosed with serious cardiac disease during follow-up [16].

Therefore, cardiac screening should be regarded not as a single static event but as an ongoing process of risk refinement throughout an athlete's career. The English FA mandates screening with an ECG and echocardiogram at the age of 16. Following the publication of the results of their screening programme, the association extended the screening process to 2-yearly evaluations after the age of 16 years, including a health questionnaire and a 12-lead ECG, on the premise that evolving abnormalities on the ECG would identify underlying structural diseases.

Acquired conditions, such as atherosclerotic coronary artery disease or transient electrolyte imbalances, also fall outside the detection scope of routine athlete screening protocols. Indeed, ECG screening will fail to identify between 16 % and 20 % of cases of SCD. Inter-observer variability may also contribute to false positive results and the costs associated with ECG screening. Our institution conducted a study comparing the results of ECG interpretation of 400 ECGs between experienced sports cardiologists and cardiologists without experience in sports cardiology. We found that cardiologists who routinely screen athletes are more likely to agree on ECG interpretation than their less-experienced counterparts. However, even amongst experienced cardiologists, there is only moderate inter-observer agreement [17]. This suggests that while experience is useful, further training and possibly accreditation are required for physicians involved in ECG screening of athletes [18]. In addition to lower inter-observed agreement rates, inexperienced cardiologists were 5 times more likely request echocardiograms, 6 times more likely to request an exercise stress test and Holter and 12 times more likely to request CMR following ECG screening. Such variation is unsurprising given that there are no standardised pathways for investigating athletes with ECG abnormalities [17].

Within this broader context, several conditions illustrate the unique diagnostic challenges faced by sports cardiologists.

### *4.1. Arrhythmogenic right ventricular cardiomyopathy*

Arrhythmogenic right ventricular cardiomyopathy exemplifies the difficulties of detecting disease due to its heterogeneous presentation, which often overlaps with normal athletic adaptation. ECG abnormalities occur in approximately 80 % of cases of ARVC, but some of these, such as anterior T-wave inversion, are common findings in healthy children, females, black athletes, and endurance athletes. However, anterior T-wave inversion in isolation rarely represents ARVC in our experience [19].

### *4.2. Ethnic variances and T-wave inversion*

As athlete populations become increasingly diverse, the interpretation of ECG findings must evolve to accommodate racial variation in cardiac phenotype. Mixed-race athletes, such as those with one white European and one black or Afro-Caribbean parent, are a growing demographic. A large cohort study comparing 1000 Black, 1000 white, and 1000 mixed-race adolescent football players found that mixed-race athletes more closely resembled black athletes in terms of ECG and structural cardiac features. These included a greater prevalence of anterior T wave inversion, increased left ventricular (LV) wall thickness, and larger cavity sizes [14]. Accordingly, current recommendations advise that ECG and echocardiography in mixed-race athletes should be interpreted using criteria validated for black athletes.

Specific repolarisation anomalies such as lateral T-wave inversion should be considered abnormal irrespective of ethnicity, even though they are more common in black athletes [20]. Sheikh et al. investigated 50 black and 50 white athletes with TWI; 21 athletes were diagnosed with a clinical disorder, over 90 % of white athletes and all black athletes diagnosed showed lateral TWI, with diagnostic yields of almost 60 % in white athletes and almost 20 % in black athletes. Furthermore, 12 % with lateral TWI had identifiable pathogenic variants linked to cardiomyopathy [21]. In a study by Schnelle, deep lateral TWI in two contiguous leads had a diagnostic yield of 45 % for cardiomyopathy or myocarditis [22].

### *4.3. Novel ECG markers: low voltage complexes and PVC morphology*

Beyond conventional criteria, emerging ECG markers are increasingly being recognised, such as low QRS voltages in the limb leads, which are found in approximately 25 % of patients with arrhythmogenic cardiomyopathy or non-ischaemic scar, have demonstrated diagnostic value. A recent Italian study involving 2300 athletes (median age 18) reported that 1 % had low limb-lead voltages. Among these, 8 % had myocardial scar, frequently in conjunction with premature ventricular complexes (PVCs) [23]. Furthermore, PVC morphology can provide further precision; PVCs with a right bundle branch block (RBBB) morphology and superior axis are particularly suggestive of underlying fibrosis. Additionally, a PVC burden with a right bundle branch block morphology and a superior or intermediate axis that exceeds 500 beats per day, provides the likelihood of myocardial scar in approximately 20 % [24]; if the burden of PVCs of this morphology increases with exercise, this prevalence rises to nearly 50 % [25]. These findings suggest that QRS amplitude and PVC morphology could be incorporated into future ECG interpretation guidelines [15].

### *4.4. ECG screening Post-COVID: Implications for athletic cardiac surveillance*

The COVID-19 pandemic has added a new dimension to cardiovascular surveillance in athletes, particularly due to its recognised association with myocarditis and pericardial inflammation, both of which can heighten susceptibility to exercise-induced arrhythmias. While a single resting ECG has limited sensitivity for detecting myocarditis, the value of longitudinal assessment has become increasingly evident. Comparison of pre- and post-infection tracings enhances diagnostic accuracy, illustrating the importance of baseline ECGs within athlete health records.

In a cohort of 511 elite footballers (mean age 22), 3 % developed new ECG abnormalities following SARS-CoV-2 infection. The most frequent changes included low QRS voltages and T-wave flattening or inversion in the inferior and lateral leads. Of the 17 athletes affected, 15 were confirmed to have active pericardial inflammation, acute myocarditis, or residual myocardial injury on advanced imaging. Serial ECG comparison achieved a sensitivity of 88 % and specificity of 99 % for detecting COVID-related cardiac sequelae, highlighting the clinical value of pre-infection reference tracings. These findings underscore that, in the post-pandemic era, serial ECG monitoring may be indispensable for safeguarding athletes and guiding safe return-to-play decisions following a severe viral infection [26].

### *4.5. Screening middle-aged and older athletes*

While SCD in sport is often associated with young competitive athletes, epidemiological data show that the majority of events actually occur in middle-aged recreational athletes, particularly men between the ages of 45 and 55 [27]. In this demographic, the dominant underlying pathology is atherosclerotic coronary artery disease (CAD), a condition frequently missed by resting electrocardiography (ECG).

Intense physical exertion can acutely precipitate plaque rupture and ventricular arrhythmias, leading to myocardial infarction (MI) or SCD. The risk of MI during exercise is estimated to be six times greater than at rest, and the risk of sudden death up to sixteen times higher. However, the long-term benefits of regular physical activity are substantial. Individuals with the highest levels of habitual exercise have a 50-fold lower risk of exertion-related MI compared with sedentary counterparts [28–30].

Given these dynamics, effective screening in older athletes must go beyond ECG and focus on integrated cardiovascular risk profiling. Clinical evaluation should include assessment of symptoms such as chest pain or dyspnoea, baseline physical activity levels, and established risk prediction tools such as the SCORE or Framingham models. Particular attention should be paid to high-risk features, including diabetes mellitus, markedly elevated blood pressure or cholesterol, a family history of premature cardiovascular disease, or elevated body mass index [31].

Asymptomatic individuals with low calculated risk and good exercise tolerance may be cleared without additional testing [32]. In contrast, those with high or very high estimated cardiovascular risk, sedentary lifestyles, or intentions to engage in vigorous physical activity should undergo further assessment. This often includes a maximal exercise stress test, which provides functional information on myocardial ischaemia, aerobic capacity, and arrhythmogenic potential. Depending on the findings, further imaging, such as myocardial perfusion scans or coronary CT angiography (CTCA), may be warranted [32].

## 5. Mitigating risk beyond cardiac screening

The 12-lead ECG is not foolproof and fails to identify several chronic diseases implicated in exercise-related sudden cardiac death, especially atherosclerotic coronary artery disease. Furthermore, some deaths are secondary to the stresses of exercise, such as excessive heat or electrolyte disturbance, which any screening measure cannot identify. Several studies have shown that early Cardiopulmonary Resuscitation and the application of an automated external defibrillator can improve survival rates by up to 5-fold [33]. Whereas survival from sudden cardiac arrest in the community is in the range of 10 %, survival rates in sporting events equipped with defibrillators are in the range of 24–49 % [34], and particularly high in some marathons [35]. Such success requires the initiation of active cardiopulmonary resuscitation within 1 min and the application of an external defibrillator within 2 minutes and 20 seconds (Fig. 2).

Proper education and awareness can significantly contribute to the overall health and safety of athletes. Individuals who exercise should be advised about the cardinal symptoms of cardiac disease. Vigorous exercise is not recommended during febrile episodes, lower respiratory tract infections, and diarrheal illnesses. Furthermore, athletes should be forewarned about the use of performance-enhancing agents, such as anabolic steroids, which are more accessible and implicated in pathological left ventricular hypertrophy and accelerated coronary atherosclerosis [36] (Fig. 3).

## 6. The role of genetic testing in athlete evaluation

While advances in molecular diagnostics and emerging studies involving polygenic risk scores are transforming the understanding of inherited cardiac diseases, the role of **genetic** testing in pre-participation screening remains limited **in the current era, due** to low diagnostic yield, uncertainties related to variants of undetermined significance and factors such as cost are just some of the barriers. However, genetic testing may assist in **resolving indeterminate or borderline phenotypes**. Even in such cases, the ethical, psychological implications of disclosure, insurability, and athletic eligibility, necessitate careful counselling, specialist oversight and discussion with all the stakeholders. Its **greatest value lies in cascade testing**, where a

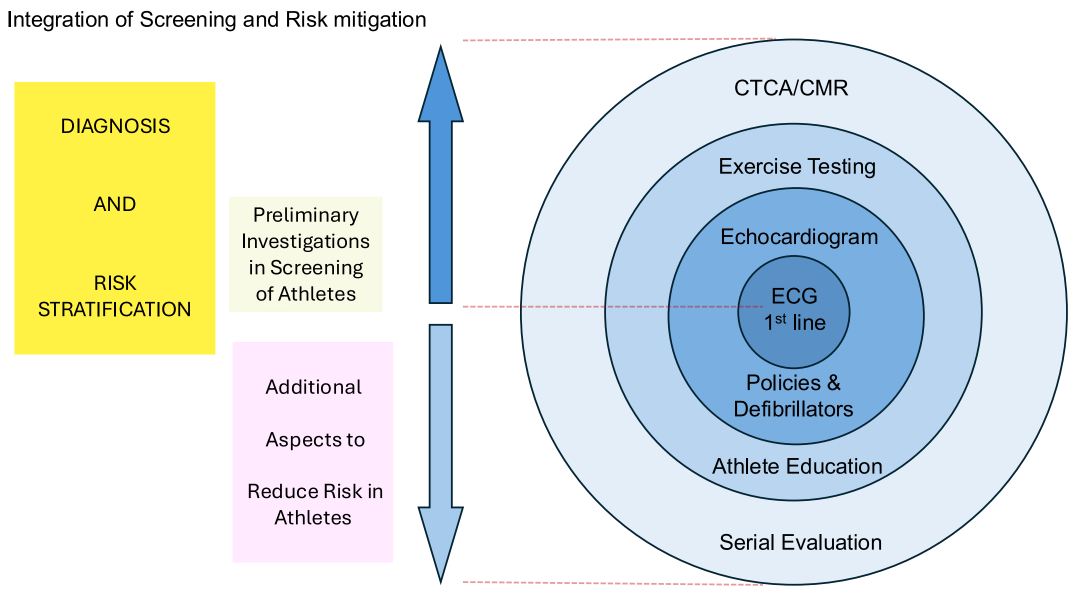


**Fig. 2. Integration of Screening and Risk mitigation** Abbreviations: ECG = electrocardiogram; CMR = cardiovascular magnetic resonance; CTCA = computed tomography coronary angiogram.

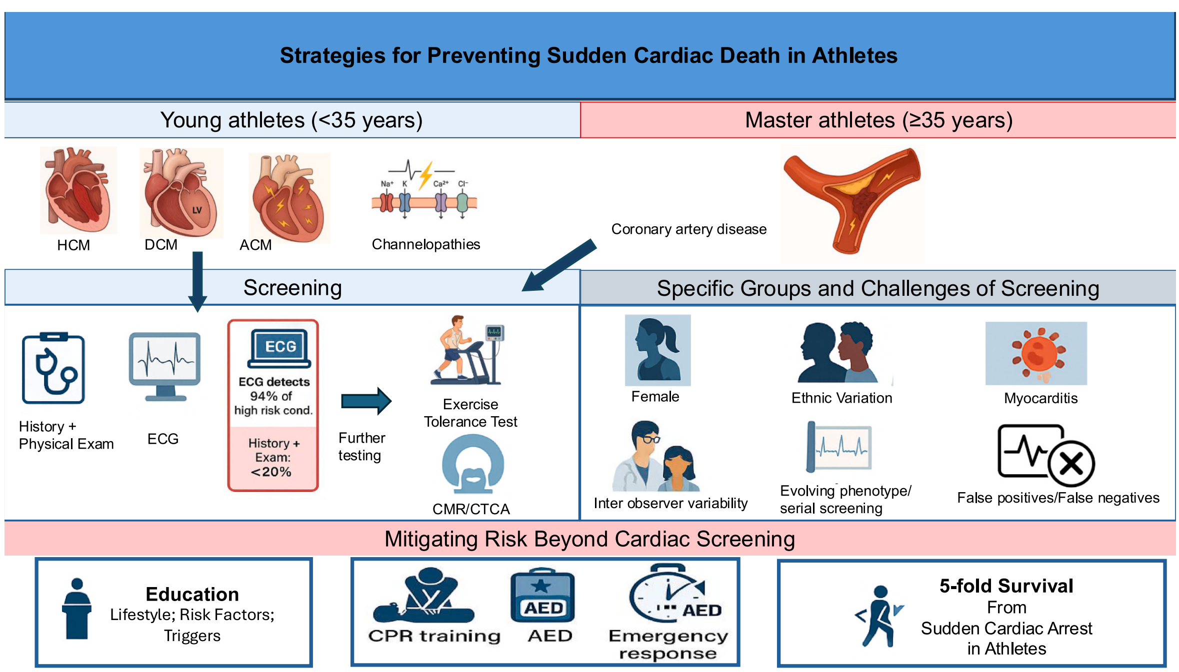


**Fig. 3. Central illustration: Screening Athletes from Aetiology to Screening to Challenges of Screening and Specific Groups.** Abbreviations: ACM = arrhythmogenic cardiomyopathy; AED = automated external defibrillator; COND = conditions; CMR = . Cardiovascular magnetic resonance; CPR = cardiopulmonary resuscitation; CTCA = computer tomography coronary angiogram; DCM = dilated cardiomyopathy; ECG = electrocardiogram; HCM = hypertrophic cardiomyopathy.

pathogenic or likely pathogenic variant has already been identified in a family member of an athlete, enabling early detection and focused surveillance of at-risk athletes. Thus, while genetic testing is invaluable in familial evaluation, its integration into population-level pre-participation screening in athletes for SCD is impractical [37].

## 7. Future directions

Future strategies must be multi-layered. Genetic testing has value in familial SCD but is limited by incomplete penetrance and uncertain variant interpretation. AI-enhanced ECG analysis offers promise in reducing inter-observer variability and improving accuracy across diverse populations. Wearables provide opportunities for continuous arrhythmia monitoring, bridging static screening with real-time surveillance. Policy must also evolve screening must be equitable across all sex, race, and socioeconomic statuses. Female athletes remain under-researched, and tailored recommendations are urgently needed. Moreover, the global sporting community must reach consensus regarding serial screening intervals, myocarditis return-to-play thresholds, and how to integrate novel markers into standard practice.

## 8. Conclusion

Sudden cardiac death in athletes, though rare, remains a profoundly consequential event with disproportionate societal and emotional impact. Its prevention is complicated by the heterogeneity of underlying pathology, which differs substantially between younger athletes. Screening strategies must be cost-effective and balanced against the vast number of exercising individuals compared to a relatively small number that succumb to a sudden cardiac arrest. The ECG is a cheap and readily available screening tool. The development and refinement of international ECG interpretation guidelines have significantly enhanced diagnostic specificity in young athletes while preserving sensitivity. However, expert interpretation is essential and may be lacking in some regions. Deaths may occur despite a normal electrocardiogram; therefore, adequate cardiopulmonary resuscitation and rapid use of an automated external cardioverter defibrillator are essential in minimising risk.

## Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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