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Sudden Cardiac Arrest in Athletes: Current Perspectives on Prevention and Emergency - A Narrative Review

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Abstract

Background: Sudden cardiac arrest (SCA) in athletes is a leading cause of exercise-related mortality driven by underlying cardiac substrates and exertional triggers.

Aim: The primary objective of this narrative review is to summarize and evaluate current evidence regarding the etiology, epidemiology, pathophysiology, and prevention strategies of SCA in athletes, while highlighting essential protocols for on-field emergency management.

Material and methods: This review synthesizes data from global registries and clinical studies to analyze SCA incidence, screening models, and survival outcomes.

Results: SCA incidence ranges from 1:40,000 to 1:80,000 athlete-years, with higher risks in male (5.55-fold) and Black athletes (nearly 3-fold). Primary causes include hypertrophic cardiomyopathy (36% of US fatalities) and coronary anomalies (12%–14%), triggered by exercise-induced adrenergic surges. The 2017 International Criteria for ECG have reduced false-positives to ~3%. Witnessed SCA with defibrillation within 3 minutes yields an 68%–89% survival rate, vastly outperforming the general population (8%–10%).

Conclusions: Preventing SCA mortality requires optimized screening, rehearsed venue Emergency Action Plans, immediate AED access, and genetic cascade screening for inherited conditions.

Keywords: Sudden Cardiac Arrest (SCA), Competitive Athletes, Sports Cardiology, Cardiovascular Abnormalities.

1. Introduction

Sudden cardiac arrest (SCA) in athletes is defined as a critical, life-threatening electrophysiological event characterized by the abrupt termination of cardiac electrical and mechanical activity, leading to immediate circulatory collapse and unresponsiveness [21]. These events typically result from an underlying susceptible substrate, specifically structural cardiac abnormalities such as hypertrophic cardiomyopathy (HCM) and arrhythmogenic cardiomyopathy (ACM), or primary electrical diseases, including inherited channelopathies like long QT syndrome and Brugada syndrome [1, 26, 27]. A central diagnostic dilemma in sports cardiology is the accurate differentiation between physiological "athlete's heart"-characterized by benign exercise-induced cardiac remodeling (EICR)-and pathological disease, as structural features such as left ventricular hypertrophy and electrocardiographic patterns like T-wave inversions create significant overlap between the two states [34, 35]. While pre-participation screening (PPS) aims to identify at-risk individuals, the standard 14-point history and physical examination (H&P) exhibits a documented low sensitivity of 10%–20% in detecting silent cardiac conditions [18, 26]. Consequently, the clinical significance of rapid on-field recognition of SCA features-including agonal gasps, seizure-like activity, and eyes wide open with a fixed gaze-is paramount, as survival is highly dependent on the speed of the medical response [2, 13, 16].

The primary objective of this narrative review is to summarize and evaluate current evidence regarding the etiology, epidemiology, pathophysiology, and prevention strategies of SCA in athletes, while highlighting essential protocols for on-field emergency management.

2. Materials and Methods

2.1. Literature Search and Study Selection

This study is a narrative literature review concerning the epidemiology, underlying etiologies, pathophysiology, and prevention strategies of sudden cardiac arrest (SCA) in competitive athletes. Publications were analyzed in order to base the review on contemporary original studies, comprehensive reviews, and current scientific statements addressing cardiovascular abnormalities, screening models, and field-side emergency management in sports cardiology.

The literature search was conducted in the PubMed, Scopus, and Web of Science databases. Keywords and their combinations included, among others: “Sudden Cardiac Arrest”, “Competitive Athletes”, “Sports Cardiology”, “Cardiovascular Abnormalities”. In addition, the references cited in publications considered key to the topic were also examined.

The review included English-language publications directly related to exertional SCA or underlying cardiac substrates, involving competitive athletes or highly physically active individuals, and addressing diagnostic, electrophysiological, or emergency-response outcomes. Original studies, narrative and systematic reviews, meta-analyses, and contemporary consensus statements from major cardiovascular societies were included. Publications not directly related to the topic, studies conducted outside athletic or relevant young populations, as well as isolated case reports and editorial comments, were excluded.

The analysis was qualitative in nature and consisted of a critical synthesis of the data, taking into account demographic and racial disparities, structural and electrical substrates, diagnostic utility of screening criteria, and the efficacy of on-field emergency action plans. Due to the heterogeneity of the registry data, regional variations in incidence, and evolving electrocardiographic interpretation criteria, no quantitative meta-analysis was performed.

3. Etiology of Sudden Cardiac Arrest in Athletes

Sudden cardiac arrest (SCA) in competitive athletes is a heterogeneous event caused by various structural, electrical, and acquired cardiac conditions [13]. Historically, hypertrophic cardiomyopathy (HCM) was identified as the leading cause of sudden cardiac death (SCD) in

United States athletes, accounting for approximately 36% of fatalities [1]. Pathophysiologically, HCM is characterized by unexplained left ventricular hypertrophy and is frequently linked to pathogenic variants in sarcomere protein genes, most commonly MYH7 and MYBPC3 [18, 22, 27]. Another significant structural etiology is arrhythmogenic cardiomyopathy (ACM), which involves the progressive fibrofatty replacement of the myocardium, primarily affecting the right ventricle but often involving the left [25, 26, 27]. This condition is notably prevalent in European cohorts, specifically in the Veneto region of Italy, where it accounts for approximately 20% of SCA cases [1, 26]. Congenital coronary artery anomalies (CCAA), particularly those with an interarterial course between the aorta and pulmonary artery, are the second most common cause of SCD in athletes, identified in 12% to 14% of cases [5, 17, 26].

Primary electrical diseases, or channelopathies, occur in structurally normal hearts and include inherited syndromes such as Long QT syndrome (LQTS), Brugada syndrome, and catecholaminergic polymorphic ventricular tachycardia (CPVT) [1, 3, 18]. LQTS, affecting approximately 1 in 2,000 individuals, is driven by mutations in genes like KCNQ1 (LQT1) and KCNH2 (LQT2), which disrupt cardiac repolarization and predispose athletes to polymorphic ventricular tachycardia [17, 27]. CPVT is triggered by adrenergic surges during exercise and is linked to mutations in the RYR2 or CASQ2 genes [17, 22, 27]. Furthermore, autopsy-negative sudden unexplained death (AN-SUD) has emerged as a predominant finding in modern registries, representing 42% of sudden deaths in United Kingdom athletes and up to 31% in other populations [1, 28]. Finally, commotio cordis accounts for up to 20% of SCD in young athletes and results from blunt, non-penetrating chest trauma during a critical 15-millisecond window of the cardiac cycle [3].

4. Epidemiology of Sudden Cardiac Arrest in Athletes

Modern surveillance identifies an overall incidence of sudden cardiac arrest or death (SCA/D) in athletes ranging from 1:40,000 to 1:80,000 athlete-years (AY) [28]. Significant geographic variations exist across global registries; for instance, the cumulative incidence rate in regional and national European data is reported at 0.98 per 100,000 AY, while focused studies on competitive North American athletes aged 14 to 25 indicate a higher rate of 1.91 per 100,000 AY [10]. In Denmark, studies indicate an incidence of approximately 1.21 per 100,000 person-years among the young population, whereas specific national incidence statistics for Poland remain less detailed in current literature. Specific sport disciplines also demonstrate

varying risk profiles; soccer remains the leading discipline for SCA events in European registries, representing approximately 46% of recorded sports-related arrests, whereas basketball and American football account for over 50% of all reported cases in the United States [1].

Substantial and pronounced demographic disparities exist in the incidence and underlying causes of SCA across sex and racial groups. Quantitative analysis indicates that male competitive athletes face an approximately 5.55-fold higher relative risk of SCA compared to female athletes, with incidences estimated at 1.42 per 100,000 AY and 0.32 per 100,000 AY, respectively. The underlying etiologies for these events demonstrate highly sex-specific distributions: hypertrophic cardiomyopathy (HCM) is the predominant cause identified in 45.12% of male cases, whereas congenital coronary artery anomalies are the most frequent etiology in females, accounting for 33.04% of arrests [24]. Physiological mechanisms contributing to the lower incidence in women include biological differences in exercise-induced cardiac remodeling (EICR); female athletes typically exhibit lower left ventricular (LV) wall thickness and mass compared to males, even when adjusted for body size [12]. Additionally, hormonal protection provided by estrogen in pre-menopausal women may contribute to a significantly lower prevalence of coronary artery disease (CAD) in younger female athletes [24, 29].

Racial disparities are equally pronounced, with Black athletes exhibiting a nearly three-fold increased risk of SCA compared to their white counterparts (1:21,491 vs. 1:68,354 AY) [1, 13]. Several physiological, structural, and environmental factors drive this heightened vulnerability [1]. Athletes of African ancestry often exhibit greater baseline LV wall thickness than those of European ancestry, with some individuals presenting thresholds that overlap with structural cardiomyopathies like HCM [12, 24]. Furthermore, abnormal T-wave inversions (TWI) are found in up to 13% of Black athletes compared to only 1.5% of white athletes, severely complicating diagnostic differentiation during pre-participation screening [34, 35]. In the specific context of basketball, it has been postulated that the profound hemodynamic and adrenergic strain of the sport, coupled with a higher prevalence of a "marfanoid habitus" within this athletic population, further increases susceptibility to catastrophic cardiac events [1].

5. Pathophysiology and Pathology of Sudden Cardiac Arrest

5. 1. Electrophysiological Mechanisms and Triggering Factors

Sudden cardiac arrest (SCA) in athletes primarily results from the interaction between a susceptible cardiac substrate and acute triggers during physical exertion [1, 22]. During exercise, the concurrent activation of the sympathetic nervous system and suppression of the parasympathetic system leads to profound changes in cardiac ion channel dynamics. Increased circulating noradrenaline binds to β-adrenergic receptors on cardiomyocytes, enhancing the inward calcium current ($I_{CaL)}$ through L-type Ca 2+ channels. This mechanism leads to the loading of the sarcoplasmic reticulum with calcium, which may result in spontaneous calcium leakage and subsequent triggered activity. While noradrenaline also activates slow delayed rectifier potassium channels (I_{Ks}) to balance the action potential duration, an imbalance in these currents can predispose the myocardium to electrical instability [12]. Consequently, acute exercise acts as a trigger for malignant arrhythmias, including ventricular tachycardia (VT) and ventricular fibrillation (VF), which are the leading causes of circulatory collapse [1, 2, 21, 23].

5. 2. Adrenergic Modulation and Ion Channel Dynamics

In athletes with inherited channelopathies, catecholamine surges during exercise directly modulate mutated ion channels, disrupting normal repolarization [12, 17, 26]. For instance, Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT) is characterized by pathogenic variants in the ryanodine receptor 2 (*RYR2*) or calsequestrin 2 (*CASQ2*) genes, which cause calcium handling abnormalities during adrenergic stress [12, 17, 27]. In Long QT Syndrome Type 1 (LQT1), mutations in the *KCNQ1* gene impair the *IKs* current, preventing the necessary shortening of the QT interval during exercise-induced tachycardia [12, 26, 27]. Furthermore, acute strenuous exercise has been associated with transient electrical instability, manifested as increased premature ventricular complexes (PVCs) and *QTc* prolongation [12, 25]. These alterations create a pro-arrhythmogenic environment where abnormal cardiac impulse formation can degenerate into sustained, life-threatening ventricular arrhythmias [12].

5. 3. Structural Substrates: Myocardial Fibrosis and Fatty Infiltration

Structural remodeling provides a permanent substrate for reentry-based arrhythmias in competitive athletes. Myocardial fibrosis, a common feature in hypertrophic cardiomyopathy (HCM) and arrhythmogenic cardiomyopathy (ACM), acts as a barrier to normal electrical conduction, causing conduction slowing and block. Fibrosis-related uncoupling of myocardial

tissue increases the probability of reentrant circuits and triggered arrhythmias [12]. In ACM, the myocardium undergoes progressive fibrofatty replacement, primarily in the right ventricle, due to mutations in desmosomal protein genes such as plakophilin-2 (*PKP2*) [17, 18, 26]. High-intensity endurance exercise has been shown to accelerate this disease progression and increase the risk of VAs [12, 18]. Additionally, a localized pattern of mid-myocardial or epicardial scar, known as non-ischaemic LV scar (NILVS), has been identified in some highly trained athletes, serving as an occult substrate for sudden circulatory collapse [12, 26].

5. 4. Molecular Mechanisms of Mechanical Stretch and Genetic Predisposition

Mechanical stretch of the myocardium, both acute and chronic, contributes to the triggering of arrhythmias through the activation of mechano-sensitive ion channels. This mechano-electric feedback allows excess sodium and calcium entry into cardiac cells, predisposing them to electrical instability. Chronic hemodynamic stress also activates gene expression in cardiomyocytes leading to hypertrophic remodeling via factors such as angiotensin II, endothelin-1, and basic fibroblast growth factor [12]. Genetic biomarkers play a critical role, with sarcomere protein gene mutations (e.g., *MYH7* and *MYBPC3*) underlying the disordered alignment of myocardial fibers and collagen deposition seen in HCM [17, 18]. Furthermore, emerging research indicates that a high polygenic risk score (PRS) for left ventricular end-systolic volume index is associated with reduced ejection fraction and increased PVC frequency in a subset of elite endurance athletes [12, 34]. Finally, acute exertional SCA may involve plaque disruption in athletes with coronary artery disease, where shear forces induced by high-intensity activity trigger plaque rupture and myocardial ischaemia [22, 29].

6. General Clinical Presentation of Cardiac Warning Signs

Although sudden cardiac arrest (SCA) often occurs without warning, approximately 25% to 50% of individuals experience prodromal symptoms prior to the event [21]. Retrospective analyses indicate that up to 29% of athletes who suffer sudden cardiac death (SCD) during competition had documented cardiac symptoms in the weeks preceding the arrest [17, 25]. Exertional syncope is a paramount clinical red flag, with collegiate data demonstrating that a history of syncope increases the odds of SCA by a factor of 41.98 [19]. Sudden syncope occurring during exercise without a prodrome is highly concerning for life-threatening arrhythmias associated with conditions such as hypertrophic cardiomyopathy (HCM) [30]. Exertional chest pain or pressure is a significant warning sign that may indicate underlying coronary artery anomalies or myocardial ischemia [25, 30]. Unexplained or excessive dyspnea

during physical activity, disproportionate to the level of effort, must be evaluated as a potential precursor to circulatory collapse [6, 22, 30]. Palpitations, specifically those characterized as abrupt, sustained, or frequent, are strongly suggestive of underlying electrical instability or primary arrhythmia syndromes [18, 22]. Systemic warning signs also include exertional lightheadedness, dizziness, and profound, unexplained fatigue [4, 22]. A positive family history of premature SCD in first-degree relatives-defined as occurring under age 35 or 60 depending on the criteria-serves as a critical objective red flag for inherited cardiac conditions [25, 32]. Despite these documented warning signs, approximately 80% of athletes remain completely asymptomatic until the onset of SCA [8, 23].

7. Pre-Participation Screening (PPS)

The American Model, advocated by the American Heart Association (AHA) and the American College of Cardiology (ACC), utilizes a targeted 14-point questionnaire and physical examination aimed at identifying high-risk individuals [1, 18, 22, 26]. While this method is considered pragmatic and cost-effective, retrospective and prospective analyses indicate a documented low sensitivity ranging from 10% to 20% in detecting silent cardiac conditions [1, 5, 18, 26, 30]. In contrast, the European Model, supported by the European Society of Cardiology (ESC) and the International Olympic Committee (IOC), mandates the inclusion of a 12-lead electrocardiogram (ECG) as a primary screening tool [1, 30]. Proponents of this model cite a landmark Italian study where the implementation of mandatory ECG screening led to a 90% decline in sudden cardiac death (SCD) rates over 25 years [1, 26, 34]. However, universal ECG screening faces challenges regarding healthcare infrastructure costs and historically high false-positive rates, which reached up to 46% in some cohorts prior to criteria refinement [1, 26]. The evolution of ECG Interpretation Criteria, transitioning from the 2010 ESC criteria to the Seattle Criteria (2013) and the current International Criteria (2017), was specifically designed to reduce false-positive rates by accurately differentiating benign physiological adaptations, known as "athlete's heart," from pathological substrates [1, 18, 30]. Utilizing the International Criteria has been shown to reduce the false-positive rate to approximately 3% while maintaining high diagnostic sensitivity [1, 26].

8. On-Field Emergency Response and Chain of Survival

Recognition of sudden cardiac arrest (SCA) on the field is frequently hindered by the misinterpretation of clinical features such as agonal gasps (abdominal breathing) or seizure-

like activity, including myoclonic jerks [1, 3, 13]. Video analysis reveals that over 70% of bystanders fail to recognize SCA, often wasting critical time by attempting to open the airway to prevent "tongue swallowing" instead of initiating immediate chest compressions [1, 2, 13, 16]. To mitigate these delays, structured Emergency Action Plans (EAPs) are recommended for all sports organizations and schools, requiring venue-specific facility maps, designated EAP coordinators, and predetermined emergency medical services (EMS) transportation routes [1, 3, 13, 31]. Effective EAPs must be rehearsed and reviewed at least annually with all potential responders, including coaches and athletic trainers, to ensure a coordinated response [1, 3, 11, 13, 20, 31]. The "Chain of Survival" emphasizes that the single most important determinant of survival is reducing the collapse-to-defibrillation interval to under three minutes [1, 13, 15]. Statistical data indicate that survival rates decrease by approximately 7% to 10% for every minute that defibrillation is delayed [2, 3, 11]. High schools with established automated external defibrillator (AED) programs have demonstrated survival rates as high as 89% when witnessed SCA occurs on campus and prompt CPR is provided [6, 11, 13, 16].

9. Secondary Prevention and Return-to-Play (RTP) Challenges

Clinical management of athletes with implantable cardioverter-defibrillators (ICDs) has evolved significantly, moving from a historical stance of absolute restriction to more liberal return-to-play (RTP) criteria supported by multinational registry data [4, 18, 28, 30]. Appropriate device programming is paramount, utilizing high-rate detection zones and specialized sensing algorithms to prevent inappropriate shocks triggered by physiological exercise-induced tachycardia or T-wave changes [17, 28]. Contemporary sports cardiology emphasizes a Shared Decision-Making (SDM) framework, which moves away from medical paternalism to integrate the athlete's individual values and risk tolerance with clinical data [18, 25, 30, 33]. The SDM process typically involves multiple phases: diagnostic confirmation and risk stratification, education of the athlete and family on specific recurrence risks (e.g., <1%-2% annual risk of arrhythmia), and consensus implementation involving nonmedical stakeholders such as coaches and school administrators [18, 33]. Physicians face significant ethical, legal, and clinical complexities when clearing athletes for competition, particularly as the legal landscape regarding liability in the event of an exertional cardiac arrest remains largely untested in many jurisdictions [18, 26, 30]. A multidisciplinary approach involving sports cardiologists, electrophysiologists, and team physicians is recommended to ensure comprehensive evaluation and longitudinal monitoring during the RTP process [18, 33].

10. Comparative Analysis of Survival Rates: Athletes vs. General Population

Survival outcomes following sudden cardiac arrest (SCA) in the general population are historically poor, with survival to hospital discharge for out-of-hospital cardiac arrest (OHCA) ranging between 8% and 10% [2, 8, 15]. In contrast, survival from exercise-related SCA in young competitive athletes in the United States is estimated at approximately 48% to 49% [8, 11, 15, 20, 29]. Prospective studies specifically evaluating high school campus-based arrests report even higher survival rates, reaching between 68% and 89% [2, 8, 15, 16, 20]. This significant disparity is primarily driven by the fact that 93% to 96% of SCA events in competitive sports are witnessed, allowing for the immediate initiation of the "Chain of Survival" [8, 9, 15].

The single most critical determinant of these improved outcomes is the collapse-to-defibrillation interval, as survival probability declines by 7% to 10% for every minute defibrillation is delayed [1, 3, 11, 13, 23]. When an on-site automated external defibrillator (AED) is deployed, survival rates among athletes increase to 85%–89%, compared to significantly lower rates when relying solely on traditional emergency medical services [3, 8, 11, 15, 20, 31]. Furthermore, the presence of a certified athletic trainer (AT) on-site is associated with a survival rate of 83% [8, 15, 20]. Electrophysiologically, athletes are more likely to present with shockable rhythms, with ventricular fibrillation (VF) identified as the initial rhythm in 59% to 98% of sports-related SCA cases [1, 2, 7]. Despite these encouraging trends, racial disparities persist; Black and minority athletes exhibit significantly lower survival proportions (31%–33%) compared to white athletes (60%–76%), a gap potentially linked to systemic differences in school-based emergency resources [8, 15].

11. Clinical Utility of Genetic Testing and Cascade Familial Screening

In athletes presenting with a clear disease phenotype, such as hypertrophic cardiomyopathy (HCM) or arrhythmogenic cardiomyopathy (ACM), genetic testing is fundamental for diagnostic confirmation, risk stratification, and the initiation of preventive strategies [12, 27]. This process transitions from a diagnostic tool in the index athlete (proband) to a preventive strategy for the entire family through the protocol of "cascade screening" [6, 27]. Because most inherited cardiac conditions (ICCs) follow an autosomal dominant inheritance pattern, first-degree relatives-including parents, siblings, and children-carry a 50% probability of harboring the same pathogenic variant [17, 27]. Targeted genetic testing of these relatives allows for the early identification of "genotype-positive/phenotype-negative" individuals who carry the mutation but currently show no structural or electrical abnormalities on transthoracic

echocardiography (TTE), cardiac magnetic resonance (CMR), or 12-lead electrocardiogram (ECG) [22, 27].

The identification of pathogenic variants in sarcomere protein genes (e.g., *MYH7, MYBPC3* in HCM), desmosomal protein genes (e.g., *PKP2* in ACM), or ion channel genes (e.g., *KCNQ1, KCNH2* in LQTS; *RYR2, CASQ2* in CPVT) provides actionable data for long-term clinical surveillance [18, 22, 27]. The clinical management of these silent mutation carriers is critical, particularly in the context of ACM, where pathogenic variants in *PKP2* are associated with a high risk of disease acceleration [12, 18, 27]. Systematic evidence indicates that high-intensity endurance training in *PKP2* carriers can actively promote fibrofatty myocardial infiltration and heighten the risk of life-threatening ventricular arrhythmias, even in the absence of baseline symptoms or overt phenotypic disease. Consequently, the discovery of such variants may necessitate more aggressive exercise modification or even disqualification from competitive endurance sports to prevent premature phenotypic conversion and sudden circulatory collapse [18, 26, 27]. In the context of acquired inflammatory substrates, contemporary diagnosis of myocarditis in young athletes relies heavily on Cardiac Magnetic Resonance (CMR) imaging to identify myocardial edema and late gadolinium enhancement, which serve as critical markers for long-term arrhythmic risk [14].

12. Conclusions

Reducing SCA-related mortality in competitive athletes requires a multi-layered approach. While pre-participation screening using the 2017 International Criteria significantly minimizes false-positive rates, no screening model can completely eliminate risk. Therefore, secondary prevention through established, annually rehearsed venue-specific Emergency Action Plans (EAPs) and immediate access to on-site AEDs remains the most critical determinant of survival. Furthermore, modern sports cardiology must increasingly integrate genetic testing and cascade family screening to identify silent mutation carriers-particularly in conditions like ACM where high-intensity exercise accelerates disease progression-while fostering a shared decision-making paradigm for return-to-play frameworks.

Disclosure

The authors report no disclosures.

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References

1. Malik A, Hanson J, Han J, Dolezal B, Bradfield JS, Boyle NG, et al. Sudden cardiac arrest in athletes and strategies to optimize preparedness. Clin Cardiol. 2023;46(9):1059-1071. https://doi.org/10.1002/clc.24095
2. Panhuyzen-Goedkoop NM, Wellens HJ, Piek JJ. Early recognition of sudden cardiac arrest in athletes during sports activity. Neth Heart J. 2018;26(1):21-25. https://doi.org/10.1007/s12471-017-1061-5
3. Rothmier JD, Drezner JA. The role of automated external defibrillators in athletics. Sports Health. 2009;1(1):16-20. https://doi.org/10.1177/1941738108326979
4. Vecchiato M, Egger F, Palermi S. Sporting Careers After ICD Implantation in Elite Athletes. J Cardiovasc Dev Dis. 2026;13(2):97. https://doi.org/10.3390/jcdd13020097

5. Peterson DF, Kucera K, Thomas LC, et al. Aetiology and incidence of sudden cardiac arrest and death in young competitive athletes in the USA: a 4-year prospective study. Br J Sports Med. 2021;55(21):1196-1203. https://doi.org/10.1136/bjsports-2020-102666
6. Graziano F, Schiavon M, Cipriani A, et al. Causes of sudden cardiac arrest and death and the diagnostic yield of sport preparticipation screening in children. Br J Sports Med. 2024;58(5):255-260. https://doi.org/10.1136/bjsports-2023-107357
7. Kramer EB, Dvorak J, Schmied C, Meyer T. F-MARC: promoting the prevention and management of sudden cardiac arrest in football. Br J Sports Med. 2015;49(9):597-598. https://doi.org/10.1136/bjsports-2015-094764
8. Drezner JA, Peterson DF, Siebert DM, et al. Survival After Exercise-Related Sudden Cardiac Arrest in Young Athletes: Can We Do Better? Sports Health. 2019;11(1):91-98. https://doi.org/10.1177/1941738118799084
9. Edwards JJ, Compton C, Chatrath N, et al. International Criteria for Reporting Study Quality for Sudden Cardiac Arrest/Death Tool. J Am Heart Assoc. 2024;13(11):e033723. https://doi.org/10.1161/JAHA.123.033723
10. Lear A, Patel N, Mullen C, Simonson M, Leone V, Koshiaris C, et al. Incidence of Sudden Cardiac Arrest and Death in Young Athletes and Military Members: A Systematic Review and Meta-Analysis. J Athl Train. 2022;57(5):431-443. https://doi.org/10.4085/1062-6050-0748.20
11. Mason Z, Watson AM, Drezner JA. Emergency Preparedness for Sudden Cardiac Arrest in Amateur Athletic Union Basketball Teams: An Opportunity to Improve Outcomes in Higher Risk Athletes. Clin J Sport Med. 2022;32(6):617-619. https://doi.org/10.1097/JSM.0000000000001062
12. D'Ambrosio P, Claessen G, Kistler PM, Heidbuchel H, Kalman JM, La Gerche A. Ventricular arrhythmias in association with athletic cardiac remodelling. Europace. 2024;26(12):euae279. https://doi.org/10.1093/europace/euae279
13. Siebert DM, Drezner JA. Sudden cardiac arrest on the field of play: turning tragedy into a survivable event. Neth Heart J. 2018;26(3):115-119. https://doi.org/10.1007/s12471-018-1084-6
14. Żmigrodzka A, Przepióra A, Orłowska M, Kozłowska J, Sanocka M, Wielogórska A, et al. Sudden Cardiac Death (SCD) in Young Athletes – A Review of Current Guidelines and Key Studies. Qual Sport. 2026;54:70269. https://doi.org/10.12775/QS.2026.54.70269

15. Schattenkerk J, Kucera K, Peterson DF, Huggins RA, Drezner JA. Socioeconomic factors and outcomes from exercise-related sudden cardiac arrest in high school student-athletes in the USA. Br J Sports Med. 2022;56(3):138-143. https://doi.org/10.1136/bjsports-2021-104486
16. Ungaro S, Truglio TS, Zorzi A, et al. Sports-related sudden cardiac arrest: a video analysis of presenting features, management and outcomes. Br J Sports Med. 2026;60(1):20-27. https://doi.org/10.1136/bjsports-2025-110275
17. Baswaraj D, Flaker G. Syncope in Athletes: A Prelude to Sudden Cardiac Death? Mo Med. 2024;121(1):52-59.
18. Kim JH, Baggish AL, Levine BD, et al. Clinical Considerations for Competitive Sports Participation for Athletes With Cardiovascular Abnormalities: A Scientific Statement From the American Heart Association and American College of Cardiology. J Am Coll Cardiol. 2025;85(10):1059-1108. https://doi.org/10.1016/j.jacc.2024.12.025
19. Yano S, Katsumata Y, Muramoto Y, Kinoda A, Kimura T, Sato K, et al. Characteristics of Sudden Cardiac Arrest in Young Athletes: A Web-Based Survey of Athletes in Japanese College Sports. Cardiol Res Pract. 2025;2025:1265728. https://doi.org/10.1155/crp/1265728
20. Petek BJ, Churchill TW, Moulson N, et al. Survival Outcomes After Sudden Cardiac Arrest in Young Competitive Athletes From the United States. J Am Coll Cardiol. 2025;85(17):1682-1692. https://doi.org/10.1016/j.jacc.2025.03.006
21. Chhabra J, Voong T, Barnes G, Gaal W, Bratton A. Sideline Management of Sudden Cardiac Arrest. Video J Sports Med. 2025;5(2):26350254251329104. https://doi.org/10.1177/26350254251329104
22. Prakash K, Swarnakari KM, Bai M, et al. Sudden Cardiac Arrest in Athletes: A Primary Level of Prevention. Cureus. 2022;14(10):e30517. https://doi.org/10.7759/cureus.30517
23. Bassi MD, Farina JM, Bombau J, et al. Sudden Cardiac Arrest in Basketball and Soccer Stadiums, the Role of Automated External Defibrillators: A Review. Arrhythm Electrophysiol Rev. 2023;12:e03. https://doi.org/10.15420/aer.2022.30
24. Li L, Le Douairon Lahaye S, Ding S, Schnell F. Sex Differences in the Incidence of Sudden Cardiac Arrest/Death in Competitive Athletes: A Systematic Review and Meta-analysis. Sports Med. 2025;55(3):697-712. https://doi.org/10.1007/s40279-024-02163-5

25. Fanous Y, Dorian P. The prevention and management of sudden cardiac arrest in athletes. CMAJ. 2019;191(28):E787-E791. https://doi.org/10.1503/cmaj.190166
26. Abdallah AW, Nguyen D, Odeh O, et al. Cardiac Screening in Young Athletes: The Role of Diagnostics in Preventing Sudden Cardiac Death and Exploring Clearance Protocols. J Clin Med. 2026;15(5):1895. https://doi.org/10.3390/jcm15051895
27. Gray B, Semsarian C. Utility of genetic testing in athletes. Clin Cardiol. 2020;43(8):915-920. https://doi.org/10.1002/clc.23289
28. Carrington M, Providência R, Chahal CAA, et al. Cardiopulmonary Resuscitation and Defibrillator Use in Sports. Front Cardiovasc Med. 2022;9:819609. https://doi.org/10.3389/fcvm.2022.819609
29. Bohm P, Meyer T, Narayanan K, et al. Sports-related sudden cardiac arrest in young adults. Europace. 2023;25(2):627-633. https://doi.org/10.1093/europace/euac172
30. Siebert DM, Harmon KG. Looking Under the Hood: Screening for Cardiac Abnormalities in Young Athletes. Curr Rev Musculoskelet Med. 2025;19(1):2. https://doi.org/10.1007/s12178-025-09995-2
31. Edge CC, Collins JM, Avey MH, et al. On-Field Management: Cardiac Event. Video J Sports Med. 2025;5(2):26350254251326950. https://doi.org/10.1177/26350254251326950
32. Davis AJ, Orchard JW, Driscoll T, Orchard JJ. Chief medical officers' perceptions of athlete cardiac screening. JSAMS Plus. 2026;7:100138. https://doi.org/10.1016/j.jsampl.2026.100138
33. Konduri A, Lynn MM, Ezekian JE, Gaitonde M, Levine BD, Hansen K. Adolescent Athlete Returns to Competitive Basketball After Cardiac Arrest and Diagnosis of Probable Hypertrophic Cardiomyopathy. JACC Case Rep. 2026;31(12):107036. https://doi.org/10.1016/j.jaccas.2026.107036
34. Hsieh PN, Shen S, Chukwurah MI, et al. Athlete's Heart Revisited: Historical, Clinical, and Molecular Perspectives. Circ Res. 2025;137(2):231-254. https://doi.org/10.1161/CIRCRESAHA.125.325638
35. Finocchiaro G, Zorzi A, Abela M, et al. Abnormal electrocardiogram findings in athletes. Eur Heart J. 2026;47(2):152-169. https://doi.org/10.1093/eurheartj/ehaf646