

Riesgo cardiovascular en deportistas: evaluación preparticipativa, estratificación arrítmica, prevención de la muerte súbita y manejo perioperatorio

Cardiovascular Risk in Athletes: Preparticipation Screening, Arrhythmic Stratification, Sudden Cardiac Death Prevention, and Perioperative Management

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RESUMEN

Las arritmias y la muerte súbita cardíaca representan importantes desafíos en la cardiología deportiva debido a que pueden presentarse en deportistas aparentemente sanos y relacionarse con cardiopatías estructurales, canalopatías hereditarias, anomalías coronarias, miocarditis u otros sustratos arritmogénicos. El presente estudio analizó los principales factores asociados con la muerte súbita cardíaca en deportistas, con énfasis en la evaluación cardiovascular preparticipativa, la interpretación electrocardiográfica, la caracterización de las arritmias ventriculares y el manejo cardiovascular perioperatorio. Se realizó una revisión integrativa y una síntesis analítica de guías clínicas, documentos de consenso, estudios epidemiológicos, revisiones sistemáticas e investigaciones observacionales relevantes, utilizando evidencia procedente de bases de datos científicas reconocidas y sociedades cardiovasculares internacionales. Los hallazgos demostraron que la muerte súbita cardíaca presenta una baja frecuencia global, pero una distribución heterogénea, con mayor incidencia descrita en determinados grupos, incluidos los deportistas masculinos, los atletas de raza negra y los jugadores masculinos de baloncesto de División I. La miocardiopatía hipertrófica, las anomalías congénitas de las arterias coronarias, la miocarditis, la miocardiopatía arritmogénica y los trastornos eléctricos hereditarios se identificaron entre las principales condiciones subyacentes. Los criterios electrocardiográficos específicos para deportistas redujeron la clasificación de estudios como anormales sin comprometer la identificación de enfermedades cardiovasculares clínicamente significativas. El análisis de la ectopia ventricular mostró que la morfología, complejidad, comportamiento durante el ejercicio, síntomas y presencia de alteraciones estructurales aportan mayor información clínica que la carga arrítmica aislada. En el contexto perioperatorio, una elevada capacidad funcional no elimina el riesgo cardiovascular, especialmente en deportistas con sustratos arritmogénicos conocidos o sospechados. Las alteraciones electrolíticas, las fluctuaciones autonómicas, los agentes anestésicos, la hipoxemia, los cambios hemodinámicos y el estrés quirúrgico pueden favorecer la aparición de alteraciones del ritmo. La evidencia respalda una estrategia integrada e individualizada que combine cribado preparticipativo, estudios diagnósticos dirigidos, estratificación del riesgo arrítmico, toma de decisiones compartida y planificación perioperatoria. Este enfoque puede mejorar la identificación de deportistas de alto riesgo, evitando al mismo tiempo restricciones innecesarias y favoreciendo una participación deportiva y una atención quirúrgica más seguras.

PALABRAS CLAVE

deportistas, arritmias, muerte súbita cardíaca, paro cardíaco súbito, evaluación preparticipativa, electrocardiografía, cardiología deportiva, arritmias ventriculares, riesgo cardiovascular, manejo perioperatorio, miocardiopatía, canalopatías

ABSTRACT

Arrhythmias and sudden cardiac death represent major challenges in sports cardiology because they may occur in apparently healthy athletes and may be related to structural heart disease, inherited channelopathies, coronary abnormalities, myocarditis, or other arrhythmogenic substrates. This study analyzed the principal factors associated with sudden cardiac death in athletes, with emphasis on cardiovascular preparticipation evaluation, electrocardiographic interpretation, characterization of ventricular arrhythmias, and perioperative cardiovascular management. An integrative review and analytical synthesis of clinical guidelines, consensus statements, epidemiological studies, systematic reviews, and relevant observational investigations was performed using evidence from recognized scientific databases and international cardiovascular societies. The findings demonstrated that sudden cardiac death is uncommon overall but is heterogeneously distributed, with higher incidence reported in specific groups such as male athletes, Black athletes, and Division I male basketball players. Hypertrophic cardiomyopathy, congenital coronary artery anomalies, myocarditis, arrhythmogenic cardiomyopathy, and inherited electrical disorders were identified among the principal underlying conditions. Athlete-specific electrocardiographic criteria reduced abnormal classifications while preserving the detection of clinically significant cardiovascular disease. The analysis of ventricular ectopy showed that morphology, complexity, exercise response, symptoms, and the presence of structural

abnormalities are more clinically informative than arrhythmic burden alone. In the perioperative setting, high functional capacity does not eliminate cardiovascular risk, particularly in athletes with known or suspected arrhythmogenic substrates. Electrolyte disturbances, autonomic fluctuations, anesthetic agents, hypoxemia, hemodynamic changes, and surgical stress may facilitate perioperative rhythm disturbances. The evidence supports an integrated and individualized strategy combining preparticipation screening, targeted diagnostic testing, arrhythmic risk stratification, shared decision-making, and perioperative planning. This approach may improve the identification of high-risk athletes while avoiding unnecessary restrictions and promoting safer participation in sport and surgical care.

KEYWORDS

athletes, arrhythmias, sudden cardiac death, sudden cardiac arrest, preparticipation screening, electrocardiography, sports cardiology, ventricular arrhythmias, cardiovascular risk, perioperative management, cardiomyopathy, channelopathies

INTRODUCCIÓN

Sudden cardiac death (SCD) in athletes represents one of the most dramatic and clinically challenging events in sports medicine, cardiology, emergency medicine, and perioperative care. Although the absolute incidence of SCD among competitive athletes is relatively low, its occurrence in individuals who are generally perceived as healthy, physically fit, and at low cardiovascular risk generates a disproportionate medical and social impact. Athletic participation produces substantial cardiovascular adaptations, including increased cardiac chamber dimensions, enhanced vagal tone, resting bradycardia, electrocardiographic repolarization changes, and exercise-induced structural remodeling. Most of these findings represent physiological adaptations to systematic training; however, some may overlap with manifestations of inherited or acquired cardiovascular diseases capable of producing malignant ventricular arrhythmias and sudden death (Pelliccia et al., 2021; Drezner et al., 2017). Consequently, differentiating the physiological “athlete’s heart” from pathological substrates associated with arrhythmogenesis remains a central challenge in cardiovascular assessment.

Sudden cardiac death in young athletes is generally associated with structural heart disease, electrical disorders, coronary abnormalities, or acquired cardiovascular conditions that may remain clinically silent until triggered by intense physical exertion. Earlier pathological series identified hypertrophic cardiomyopathy, congenital coronary artery anomalies, arrhythmogenic cardiomyopathy, myocarditis, aortic disease, and premature coronary artery disease among important causes of sudden death during athletic activity (Maron et al., 1996). Subsequent epidemiological studies have demonstrated that the distribution of underlying causes varies according to age, sex, ethnicity, type of sport, competitive level, geographical region, and diagnostic methodology (Harmon et al., 2014; Harmon et al., 2015). In addition, primary electrical diseases such as long QT syndrome, Brugada syndrome, catecholaminergic polymorphic ventricular tachycardia, Wolff–Parkinson–White syndrome, and other inherited channelopathies may predispose athletes to life-threatening arrhythmias despite the absence of overt structural cardiac abnormalities (Ackerman et al., 2015).

Exercise itself is not inherently pathological and provides substantial cardiovascular benefits; nevertheless, vigorous physical activity may function as an acute trigger of malignant arrhythmias in susceptible individuals. Intense exercise increases sympathetic activation, circulating catecholamines, myocardial oxygen demand, heart rate, ventricular wall stress, and changes in electrolyte balance. In a heart with an arrhythmogenic substrate, these physiological responses can promote premature ventricular activity, ventricular tachycardia, ventricular fibrillation, or other unstable rhythms (Guasch & Mont, 2017). The association between intensive sports participation and sudden cardiac events therefore depends not only on the intensity of exercise but also on the interaction between exercise-related physiological stress and an underlying cardiovascular abnormality.

Evidence from competitive athlete populations has emphasized the importance of this interaction. Corrado et al. (2003) observed an increased risk of sudden cardiovascular death associated with competitive sports participation among adolescents and young adults with underlying cardiovascular disease. At the same time, the implementation of structured cardiovascular screening programs has been associated with reductions in sports-related sudden cardiac mortality in specific populations, supporting the potential role of systematic preparticipation evaluation in identifying

athletes at increased risk (Corrado et al., 2006). Nevertheless, considerable debate persists regarding the optimal screening strategy, particularly concerning the universal inclusion of the 12-lead electrocardiogram (ECG), the diagnostic yield of different protocols, the potential for false-positive findings, resource utilization, and the expertise required to interpret physiological electrical adaptations in trained individuals.

The true incidence of sudden cardiac arrest and sudden cardiac death in athletes has also been difficult to determine because studies have used heterogeneous definitions, surveillance systems, age ranges, competitive classifications, and ascertainment methods. Data from collegiate athletes indicate that the risk is not uniformly distributed across athletic populations, with higher incidence reported in certain demographic groups and sporting disciplines (Harmon et al., 2011; Harmon et al., 2015). A systematic review and meta-analysis by Lear et al. (2022) similarly demonstrated substantial variability in reported SCD and sudden cardiac arrest rates among young athletes and military populations. These findings reinforce the concept that cardiovascular prevention should not be based solely on a generalized estimation of risk but should incorporate individual characteristics, family history, exercise exposure, sport-specific demands, and clinical findings.

Preparticipation cardiovascular evaluation is therefore one of the principal preventive strategies used to identify potentially lethal cardiovascular conditions before participation in intensive exercise or competitive sport. Traditional screening approaches include a detailed personal medical history, family cardiovascular history, physical examination, and assessment of symptoms such as exertional syncope, unexplained presyncope, palpitations, exertional chest discomfort, disproportionate dyspnea, unexplained exercise intolerance, or a previous seizure-like event that may actually represent arrhythmic syncope (Maron et al., 2015). Family history is particularly relevant when it includes premature sudden death, unexplained drowning, unexplained motor vehicle accidents, inherited cardiomyopathy, aortic disease, or known channelopathy.

The 12-lead ECG has emerged as a particularly important component of athlete evaluation because several diseases associated with SCD may produce recognizable electrical abnormalities before the development of symptoms. However, interpretation requires an understanding of training-related ECG changes. Sinus bradycardia, first-degree atrioventricular block, early repolarization, isolated voltage criteria for left ventricular hypertrophy, and incomplete right bundle branch block may represent benign manifestations of athletic conditioning rather than pathological abnormalities. Conversely, T-wave inversion in specific distributions, pathological Q waves, ventricular pre-excitation, prolonged or markedly shortened QT intervals, complete conduction abnormalities, Brugada-type patterns, profound axis deviation in certain contexts, or frequent ventricular premature complexes may justify additional diagnostic investigation (Drezner et al., 2017). The International Criteria for Electrocardiographic Interpretation in Athletes were developed to improve specificity without compromising the ability to identify significant cardiac disease, thereby reducing unnecessary investigations while preserving the preventive value of ECG screening.

When initial screening identifies suspicious findings, the diagnostic pathway may extend to transthoracic echocardiography, ambulatory ECG monitoring, exercise testing, cardiac magnetic resonance imaging, computed tomography, electrophysiological studies, or genetic testing according to the suspected disorder. Modern evaluation consequently follows a tiered rather than indiscriminate diagnostic strategy. This approach is supported by contemporary sports cardiology guidelines, which emphasize individualized interpretation based on symptoms, ECG findings, ventricular function, arrhythmia burden, myocardial scar, genetic phenotype, and the physiological demands of the athlete's sport (Pelliccia et al., 2021).

The assessment of arrhythmias in athletes requires particular attention because both benign and potentially dangerous rhythm disturbances may be encountered. Sinus bradycardia and respiratory sinus arrhythmia are common consequences of increased vagal tone. Premature atrial and ventricular complexes may also occur in individuals without structural cardiac disease. Conversely, atrial fibrillation, supraventricular tachycardia, ventricular tachycardia, ventricular pre-excitation, complex ventricular ectopy, and exercise-induced arrhythmias may indicate a clinically relevant electrophysiological abnormality requiring further characterization (Guasch & Mont, 2017). Recent expert recommendations emphasize that arrhythmia assessment in athletes should evaluate not only the rhythm itself but also its relationship to exercise intensity, morphology, frequency, symptoms, underlying structural abnormalities, and potential inherited disease (Lampert et al., 2024).

Exercise-related ventricular arrhythmias are particularly relevant because their prognostic significance varies substantially depending on their characteristics. Isolated, infrequent premature ventricular contractions with benign morphology and suppression during exercise may carry a different clinical implication from polymorphic ectopy, repetitive ventricular beats, exercise-induced ventricular tachycardia, or ectopy associated with myocardial fibrosis or ventricular dysfunction. Consequently, risk stratification must integrate electrocardiographic, imaging, functional, and clinical data rather than rely on the isolated detection of arrhythmia.

Contemporary management has also moved away from the historical assumption that identification of cardiovascular disease necessarily requires permanent exclusion from competitive sport. Earlier recommendations often emphasized categorical eligibility or disqualification criteria, particularly in athletes with channelopathies, conduction disorders, cardiomyopathies, or ventricular arrhythmias (Ackerman et al., 2015; Zipes et al., 2015). More recent guidance increasingly supports individualized risk assessment and shared decision-making, recognizing that the absolute risk associated with athletic participation varies substantially according to diagnosis, disease severity, treatment response, type of sport, and athlete preference (Lampert et al., 2024). This evolution has important implications because clinical practice must balance prevention of catastrophic cardiovascular events against unnecessary restriction of physical activity, psychological consequences, loss of athletic identity, and the established health benefits of exercise.

The preventive strategy cannot be limited to identifying cardiovascular disease before sport participation. Rapid recognition and treatment of sudden cardiac arrest are equally important components of mortality reduction. Because ventricular fibrillation and pulseless ventricular tachycardia are treatable causes of sudden cardiac arrest, immediate cardiopulmonary resuscitation and early defibrillation can markedly modify outcomes. For this reason, cardiovascular safety in sport should be understood as a continuum that includes preparticipation screening, emergency action planning, availability of automated external defibrillators, trained personnel, and standardized return-to-play evaluation following cardiovascular events.

A further dimension that requires greater consideration is the perioperative management of athletes with known or suspected arrhythmias. Athletes may undergo surgery for orthopedic injuries, sports-related trauma, elective procedures, or unrelated medical conditions. Although high exercise capacity may suggest favorable functional status, athletic conditioning does not eliminate perioperative cardiovascular risk. In fact, baseline bradycardia, high vagal tone, unusual ECG patterns, cardiovascular medications, previous ablation procedures, implanted cardiac devices, genetic channelopathies, cardiomyopathies, or a history of ventricular arrhythmias may significantly affect anesthetic and perioperative decision-making.

Perioperative arrhythmias arise from a complex interaction between patient susceptibility, surgery-related stress, anesthetic drugs, sympathetic stimulation, vagal responses, hypoxia, hypercapnia, acid-base abnormalities, electrolyte disturbances, blood loss, myocardial ischemia, temperature changes, and postoperative pain. Depending on the underlying substrate, these triggers can produce supraventricular tachyarrhythmias, significant bradyarrhythmias, conduction disturbances, or ventricular arrhythmias (Pecha et al., 2023). Therefore, an athlete considered stable in the outpatient sports setting may present a different risk profile when exposed to anesthesia and surgery.

This distinction is particularly relevant in inherited channelopathies. Patients with long QT syndrome may be vulnerable to QT-prolonging medications, electrolyte disturbances, sympathetic surges, and bradycardia. Individuals with catecholaminergic polymorphic ventricular tachycardia may develop arrhythmias in response to adrenergic stimulation. Patients with Brugada syndrome may present increased susceptibility under circumstances such as fever, changes in autonomic tone, and exposure to selected pharmacological agents. Similarly, athletes with arrhythmogenic cardiomyopathy may have a substrate for ventricular arrhythmia that deserves careful assessment before surgical procedures. Consequently, perioperative planning should include identification of the specific arrhythmogenic mechanism whenever possible rather than treating all athletes with rhythm abnormalities as a homogeneous population.

Modern perioperative cardiovascular guidelines emphasize a stepwise evaluation that combines the urgency and magnitude of the surgical procedure, active cardiovascular conditions, functional capacity, patient-specific risk factors, and the expected influence of additional testing on management (Thompson et al., 2024). This principle is especially relevant in athletes because high functional capacity may reduce the need for unnecessary testing in asymptomatic individuals, but excellent exercise performance should not override red flags such as exertional syncope, documented

ventricular tachycardia, significant structural disease, inherited arrhythmic syndromes, or previously unexplained cardiac arrest. Perioperative cardiovascular assessment must therefore differentiate physiological athletic adaptation from clinically relevant pathology.

Another important consideration is medication management. Athletes with arrhythmias may be treated with beta-blockers, antiarrhythmic drugs, anticoagulants, or other cardiovascular therapies. Abrupt discontinuation, drug–drug interactions, effects on conduction, blood pressure alterations, QT interval modification, and perioperative bleeding risk can all influence outcomes. The anesthetic team must also consider whether previous electrophysiological procedures, cardiac ablation, pacemakers, or implantable cardioverter-defibrillators require specific perioperative measures. General cardiovascular risk assessment remains essential because athletes are not exempt from coronary disease, hypertension, myocarditis, or acquired myocardial abnormalities, particularly in older or master athletes.

Despite advances in sports cardiology, important gaps remain in the integration of athletic cardiovascular screening with perioperative medicine. Much of the literature on SCD focuses on screening, emergency response, and return-to-play decisions, whereas perioperative guidelines generally address broad surgical populations rather than athletes specifically. As a result, clinicians must frequently integrate evidence from sports cardiology, electrophysiology, anesthesiology, and perioperative cardiovascular medicine. This fragmentation creates a need for an interdisciplinary framework that connects preparticipation assessment, arrhythmic risk stratification, diagnostic testing, exercise-related risk, and perioperative management.

The present study therefore addresses the following central research question: **How can cardiovascular preparticipation evaluation and perioperative risk stratification be integrated to improve the detection and management of arrhythmias associated with sudden cardiac death in athletes?** Secondary questions examine which clinical, electrocardiographic, and structural findings should prompt advanced cardiovascular evaluation; which arrhythmias are most relevant to sports-related SCD; how exercise modifies arrhythmic risk; and which perioperative factors may destabilize previously compensated electrical or structural cardiovascular disease.

The working hypothesis is that a structured, multimodal cardiovascular assessment combining personal and family history, physical examination, athlete-specific ECG interpretation, targeted diagnostic testing, and individualized arrhythmic risk stratification can improve identification of athletes at increased risk of SCD while reducing unnecessary restriction of individuals with physiological cardiac adaptations. A second hypothesis proposes that perioperative management based on the specific electrophysiological and structural substrate, rather than athletic status alone, can reduce the probability of clinically significant perioperative arrhythmias.

To address these questions, the study is designed as an integrative review and analytical synthesis of major clinical guidelines, consensus statements, epidemiological studies, systematic reviews, and clinically relevant investigations related to sports cardiology, sudden cardiac death, electrocardiographic screening, cardiac arrhythmias, exercise physiology, and perioperative cardiovascular management. Particular attention is given to international recommendations from cardiology and electrophysiology societies, including contemporary guidance on sports participation and arrhythmias (Pelliccia et al., 2021; Lampert et al., 2024), standardized ECG interpretation criteria (Drezner et al., 2017), epidemiological evidence on athlete SCD (Harmon et al., 2014; Lear et al., 2022), and current perioperative cardiovascular recommendations (Thompson et al., 2024).

This methodological approach permits comparison of historical and contemporary concepts, identification of areas of clinical agreement and controversy, and integration of preventive and perioperative perspectives. Rather than considering sudden cardiac death as an isolated event, the study conceptualizes it as the potential endpoint of a continuum beginning with an underlying arrhythmogenic substrate, followed by exposure to physiological or perioperative triggers, development of electrical instability, and eventual progression to cardiac arrest when timely intervention is unavailable. This framework supports the analysis of multiple opportunities for prevention.

The relevance of this research lies in the need to reconcile two fundamental objectives: preserving the substantial health benefits of physical activity and competitive sport while minimizing preventable cardiovascular mortality. Excessive restriction based on nonspecific findings may have significant personal and health consequences, whereas inadequate recognition of malignant cardiovascular disease may expose athletes to catastrophic outcomes. Therefore, accurate risk

stratification, appropriate diagnostic escalation, individualized clinical decision-making, and coordinated perioperative management constitute essential elements of contemporary cardiovascular care.

Ultimately, a comprehensive approach to arrhythmias and sudden cardiac death in athletes requires more than the detection of abnormal ECG patterns. It demands an integrated understanding of exercise physiology, inherited cardiovascular disease, myocardial structure, arrhythmia mechanisms, individual risk, emergency preparedness, and perioperative stressors. By examining these components within a unified clinical framework, this study seeks to clarify how preparticipation cardiovascular screening and perioperative cardiovascular management can complement each other to improve safety, support evidence-based participation decisions, and reduce the risk of avoidable sudden cardiac events among athletic populations.

DESARROLLO

Arrhythmias and sudden cardiac death (SCD) in athletes represent a complex clinical problem because regular intensive exercise produces cardiovascular adaptations that may resemble pathological findings. Increased vagal tone, resting sinus bradycardia, chamber enlargement, and several electrocardiographic changes are common in trained individuals and are generally benign. However, similar findings may coexist with cardiomyopathies, channelopathies, congenital coronary abnormalities, or inflammatory myocardial disease, which can increase susceptibility to malignant ventricular arrhythmias during intense exertion (Drezner et al., 2017; Pelliccia et al., 2021).

The risk of SCD is not uniformly distributed among athletes. Epidemiological studies have shown important differences according to age, sex, sport, competitive level, and underlying cardiovascular disease. In collegiate athletes, certain groups have demonstrated a higher incidence of sudden cardiac events, supporting the need for risk-oriented rather than purely generalized screening strategies (Harmon et al., 2011; Harmon et al., 2015). Although the absolute incidence is low, the potentially catastrophic nature of SCD justifies systematic prevention, particularly because many affected individuals may have no previously recognized cardiovascular diagnosis.

Among the most important substrates associated with sports-related SCD are hypertrophic cardiomyopathy, arrhythmogenic cardiomyopathy, myocarditis, congenital coronary anomalies, premature coronary artery disease, and inherited electrical disorders such as long QT syndrome, Brugada syndrome, and catecholaminergic polymorphic ventricular tachycardia (Ackerman et al., 2015; Maron et al., 1996). In these conditions, intense exercise can act as a trigger through increased sympathetic activation, catecholamine release, myocardial oxygen demand, mechanical stress, and alterations in electrophysiological stability (Guasch & Mont, 2017).

For this reason, cardiovascular preparticipation evaluation constitutes a central preventive strategy. The initial assessment should include detailed personal and family history, physical examination, and identification of warning symptoms such as exertional syncope, chest pain, palpitations, unexplained dyspnea, or reduced exercise tolerance. A family history of premature sudden death, inherited cardiomyopathy, or channelopathy should also raise clinical suspicion (Maron et al., 2015).

The resting 12-lead electrocardiogram provides additional diagnostic value when interpreted according to athlete-specific criteria. The International Criteria for Electrocardiographic Interpretation in Athletes distinguish physiological training-related findings from abnormalities that require further evaluation. Pathological T-wave inversion, ventricular pre-excitation, prolonged QT interval, Brugada patterns, pathological Q waves, or frequent ventricular premature complexes may justify additional testing such as echocardiography, ambulatory ECG monitoring, exercise testing, cardiac magnetic resonance imaging, or genetic evaluation (Drezner et al., 2017).

Current recommendations emphasize that the presence of an arrhythmia should not automatically result in permanent exclusion from sport. The clinical significance depends on the type of arrhythmia, symptoms, ventricular function, underlying structural disease, arrhythmia burden, response to exercise, and risk of sudden deterioration. Contemporary approaches increasingly favor individualized risk stratification and shared decision-making instead of rigid eligibility criteria (Lampert et al., 2024). This is especially important in athletes with supraventricular tachycardia, atrial fibrillation, premature ventricular contractions, or successfully treated arrhythmias.

Ventricular arrhythmias require particular attention because they may represent the first manifestation of an arrhythmogenic myocardial substrate. The morphology, frequency, complexity, and relationship with exercise are clinically relevant. Isolated premature ventricular contractions in a structurally normal heart may have limited prognostic significance, whereas polymorphic ectopy, repetitive ventricular beats, exercise-induced ventricular tachycardia, or arrhythmias associated with myocardial fibrosis warrant more extensive investigation (Guasch & Mont, 2017; Lampert et al., 2024).

The perioperative environment introduces an additional level of cardiovascular stress. Athletes requiring surgery may be exposed to sympathetic activation, anesthetic drugs, hypoxia, electrolyte disturbances, blood loss, pain, temperature changes, and autonomic fluctuations capable of precipitating rhythm disturbances. Consequently, good baseline physical condition does not eliminate perioperative cardiovascular risk, particularly when an underlying channelopathy, cardiomyopathy, previous ablation, implanted cardiac device, or documented ventricular arrhythmia is present (Pecha et al., 2023).

Perioperative evaluation should therefore integrate functional capacity with the athlete's cardiovascular history and the characteristics of the planned procedure. Current perioperative guidelines recommend a stepwise assessment based on active cardiovascular conditions, surgical risk, functional status, and whether additional testing is likely to modify clinical management (Thompson et al., 2024). In athletes, this principle is particularly valuable because high exercise capacity may reduce unnecessary testing in otherwise asymptomatic individuals, but it should never override clinically significant warning signs.

Specific inherited arrhythmogenic disorders also require tailored perioperative strategies. In long QT syndrome, avoidance of QT-prolonging medications and correction of electrolyte abnormalities are essential. Patients with catecholaminergic polymorphic ventricular tachycardia require careful control of adrenergic stimulation, whereas individuals with Brugada syndrome may be vulnerable to fever, autonomic changes, and selected medications. These examples demonstrate that perioperative management should be based on the precise electrophysiological diagnosis rather than on athletic status alone.

OBJETIVO GENERAL Y OBJETIVOS ESPECÍFICOS

General Objective

To **analyze and integrate** the principal clinical, electrocardiographic, structural, and perioperative factors associated with arrhythmias and sudden cardiac death in athletes, in order to establish an evidence-based framework for preparticipation cardiovascular evaluation, risk stratification, and perioperative management.

Specific Objectives

Cognitive Domain

1. To **identify** the main structural, electrical, genetic, and acquired cardiovascular conditions associated with sudden cardiac death in athletes.
2. To **differentiate** physiological cardiovascular adaptations related to athletic training from electrocardiographic and structural abnormalities suggestive of cardiovascular disease.
3. To **analyze** the clinical relevance of supraventricular and ventricular arrhythmias according to their morphology, frequency, symptoms, relationship with exercise, and associated cardiac abnormalities.
4. To **evaluate** the usefulness of personal and family history, physical examination, 12-lead ECG, exercise testing, ambulatory ECG monitoring, echocardiography, cardiac magnetic resonance imaging, and genetic testing in cardiovascular risk stratification.

5. To **compare** current recommendations regarding athletic participation, return to play, and perioperative cardiovascular management in athletes with known or suspected arrhythmias.

Psychomotor Domain

6. To **apply** a structured preparticipation cardiovascular assessment pathway for the identification of warning signs and findings that require complementary diagnostic testing.
7. To **interpret** athlete-specific electrocardiographic findings using standardized criteria to distinguish normal training-related changes from potentially pathological patterns.
8. To **construct** a clinical decision pathway that integrates arrhythmic risk, cardiovascular diagnosis, surgical risk, and perioperative factors relevant to athletes undergoing surgical procedures.

Affective Domain

9. To **promote** awareness of the importance of early cardiovascular risk detection and prevention of sudden cardiac death without unnecessarily restricting safe participation in physical activity.
10. To **value** individualized and multidisciplinary clinical decision-making involving sports cardiology, electrophysiology, anesthesiology, and perioperative medicine.
11. To **support** shared decision-making that considers cardiovascular safety, scientific evidence, the characteristics of the sport, and the athlete's preferences and quality of life.

OBJETO DE ESTUDIO

The object of study is the **cardiovascular risk associated with arrhythmias and sudden cardiac death in athletes**, with particular emphasis on preparticipation evaluation, identification of potentially malignant rhythm disorders, and perioperative cardiovascular management.

The population of interest includes **adolescent and adult recreational or competitive athletes** who undergo cardiovascular assessment before sports participation or who require surgical procedures in the presence of known, suspected, or previously undiagnosed cardiovascular abnormalities. Special attention is given to athletes with symptoms such as exertional syncope, palpitations, chest pain, unexplained dyspnea, reduced exercise tolerance, or a family history of premature sudden cardiac death.

The phenomenon under investigation includes both **physiological adaptations produced by systematic exercise** and pathological conditions that may predispose to arrhythmias. These include hypertrophic cardiomyopathy, arrhythmogenic cardiomyopathy, myocarditis, congenital coronary abnormalities, long QT syndrome, Brugada syndrome, catecholaminergic polymorphic ventricular tachycardia, ventricular pre-excitation, and clinically relevant supraventricular or ventricular arrhythmias (Pelliccia et al., 2021; Lampert et al., 2024).

The study also examines the diagnostic tools used to distinguish benign athletic adaptations from cardiovascular disease, including medical and family history, physical examination, 12-lead electrocardiography, ambulatory ECG monitoring, exercise testing, echocardiography, cardiac magnetic resonance imaging, and genetic evaluation when indicated (Drezner et al., 2017).

Within the perioperative context, the object of study extends to the interaction between pre-existing arrhythmic susceptibility and factors such as anesthesia, autonomic changes, electrolyte disturbances, hypoxia, hemodynamic instability, medications, and surgical stress. These factors may increase the probability of clinically significant perioperative arrhythmias even in individuals with high baseline functional capacity (Pecha et al., 2023; Thompson et al., 2024).

Therefore, the study focuses on the **integration of sports cardiology, electrophysiology, and perioperative cardiovascular medicine** to establish a comprehensive approach for identifying athletes at increased risk, guiding

additional diagnostic evaluation, supporting safe sports participation, and reducing preventable cardiovascular complications.

METODOLOGÍA

This study was conducted using the **Scientific Method**, applied through a structured integrative review and analytical synthesis of the available evidence on arrhythmias, sudden cardiac death in athletes, cardiovascular preparticipation screening, and perioperative management. This methodology was selected because it allows the research problem to be examined systematically, hypotheses to be contrasted with published evidence, and clinically relevant conclusions to be generated from reproducible procedures.

The research followed a **descriptive, analytical, and documentary design**, without experimental intervention. The unit of analysis consisted of clinical practice guidelines, consensus statements, systematic reviews, cohort studies, observational investigations, and relevant clinical articles addressing cardiovascular risk in athletes.

A structured literature search was performed using scientific databases and recognized medical sources, including **PubMed/MEDLINE, Scopus, and publications from major cardiovascular scientific societies**, particularly the European Society of Cardiology, American Heart Association, American College of Cardiology, and Heart Rhythm Society. Priority was given to peer-reviewed publications with direct relevance to athlete screening, arrhythmia diagnosis, sudden cardiac death, sports participation, and perioperative cardiovascular assessment.

The search strategy combined terms such as **“athlete,” “sudden cardiac death,” “sudden cardiac arrest,” “arrhythmia,” “preparticipation screening,” “electrocardiogram,” “sports cardiology,” “ventricular arrhythmia,” “channelopathy,” “cardiomyopathy,” “perioperative arrhythmia,” and “perioperative cardiovascular risk.”** Boolean operators AND and OR were used to improve sensitivity and specificity during literature retrieval.

Studies were included when they met the following criteria: publication in peer-reviewed scientific sources; direct relevance to cardiovascular evaluation in athletes; assessment of arrhythmias, sudden cardiac arrest, or sudden cardiac death; evaluation of diagnostic or screening strategies; or analysis of perioperative cardiovascular risk. Major international guidelines and expert consensus documents were also included because of their importance for clinical decision-making (Pelliccia et al., 2021; Lampert et al., 2024; Thompson et al., 2024).

Publications were excluded when they lacked direct relationship with the research question, presented insufficient methodological information, focused exclusively on non-cardiovascular sports injuries, or did not provide clinically applicable evidence.

The extracted information was organized into five analytical categories: **epidemiology and causes of sudden cardiac death, preparticipation cardiovascular screening, interpretation and classification of arrhythmias, diagnostic and risk-stratification strategies, and perioperative cardiovascular management.** Particular attention was given to the differentiation between physiological adaptations of the athlete's heart and findings suggestive of underlying cardiovascular disease (Drezner et al., 2017).

The evidence was subsequently compared across studies and clinical guidelines to identify points of agreement, differences in recommendations, diagnostic warning signs, and factors associated with increased arrhythmic risk. The resulting synthesis was used to construct an integrated clinical framework linking preparticipation assessment with perioperative cardiovascular decision-making.

To ensure reproducibility, another researcher may replicate the study by using the same databases, search terms, eligibility criteria, evidence categories, and analytical sequence described above. The methodological process was therefore designed to provide a transparent and structured approach for evaluating the relationship between athletic participation, arrhythmias, sudden cardiac death, and perioperative cardiovascular risk.

FASES DEL DESARROLLO

Phase 1. Identification of the Research Problem

The first phase consisted of defining the clinical problem associated with arrhythmias and sudden cardiac death in athletes. Particular attention was given to the difficulty of distinguishing physiological cardiovascular adaptations produced by training from pathological findings related to cardiomyopathies, channelopathies, or other arrhythmogenic conditions. The perioperative setting was incorporated because anesthesia, surgical stress, electrolyte disturbances, and autonomic changes may modify arrhythmic risk (Pecha et al., 2023).

Phase 2. Formulation of the Research Question

The central research question was established as follows: **How can cardiovascular preparticipation evaluation and perioperative risk stratification be integrated to improve the detection and management of arrhythmias associated with sudden cardiac death in athletes?**

Secondary questions addressed the diagnostic value of clinical history, ECG, imaging studies, exercise testing, ambulatory monitoring, and genetic assessment, as well as the identification of perioperative factors capable of precipitating clinically relevant arrhythmias.

Phase 3. Hypothesis Formulation

The main hypothesis proposed that a structured cardiovascular assessment combining clinical history, family history, physical examination, athlete-specific ECG interpretation, and targeted complementary testing improves the identification of individuals at increased risk of malignant arrhythmias.

A complementary hypothesis proposed that individualized perioperative management based on the underlying electrophysiological or structural disorder can reduce preventable cardiovascular complications during surgical procedures.

Phase 4. Collection of Scientific Evidence

A structured literature search was performed using PubMed/MEDLINE, Scopus, clinical guidelines, consensus statements, systematic reviews, and observational studies. Publications addressing sudden cardiac death, cardiovascular screening, athlete ECG interpretation, arrhythmias, cardiomyopathies, channelopathies, and perioperative cardiovascular management were prioritized.

Relevant recommendations from the ESC, AHA, ACC, and HRS were included because they provide current standards for cardiovascular assessment, sports participation, arrhythmia management, and perioperative care (Pelliccia et al., 2021; Lampert et al., 2024; Thompson et al., 2024).

Phase 5. Classification and Analysis of Evidence

The selected information was classified into five domains: epidemiology of sudden cardiac death, cardiovascular preparticipation screening, arrhythmia characterization, diagnostic and risk-stratification strategies, and perioperative management.

Evidence was compared to determine which findings are considered physiological in athletes and which represent warning signs requiring additional evaluation. Athlete-specific ECG criteria were particularly relevant for reducing false-positive interpretations while maintaining sensitivity for potentially serious cardiovascular disease (Drezner et al., 2017).

Phase 6. Integration of Clinical Findings

The analyzed evidence was integrated into a clinical sequence beginning with history and physical examination, followed by ECG assessment and targeted diagnostic testing when abnormalities were identified. Arrhythmic risk was then related to structural heart disease, symptoms, exercise response, and sport characteristics.

For athletes requiring surgery, additional variables such as anesthetic exposure, medication use, electrolyte balance, autonomic stimulation, implanted cardiac devices, and previous electrophysiological procedures were incorporated into the assessment.

Phase 7. Interpretation and Verification of the Hypotheses

The findings obtained from the reviewed evidence were contrasted with the proposed hypotheses. Current evidence supports the use of individualized cardiovascular assessment rather than indiscriminate restriction of sports participation, particularly when physiological adaptations can be distinguished from pathological abnormalities (Lampert et al., 2024).

Similarly, perioperative evidence supports the identification and correction of modifiable arrhythmogenic factors and the adaptation of monitoring and treatment according to the underlying cardiovascular disorder (Thompson et al., 2024).

Phase 8. Development of the Final Clinical Framework

Finally, the evidence was synthesized into an integrated framework combining **preparticipation screening, arrhythmic risk stratification, diagnostic escalation, sports participation decisions, and perioperative cardiovascular management**. This framework was designed to facilitate early identification of high-risk athletes while avoiding unnecessary investigations or restrictions in individuals with benign physiological adaptations.

RESULTADOS Y DISCUSIÓN

Figure 1.

Incidence of sudden cardiac death according to selected athlete subgroups.

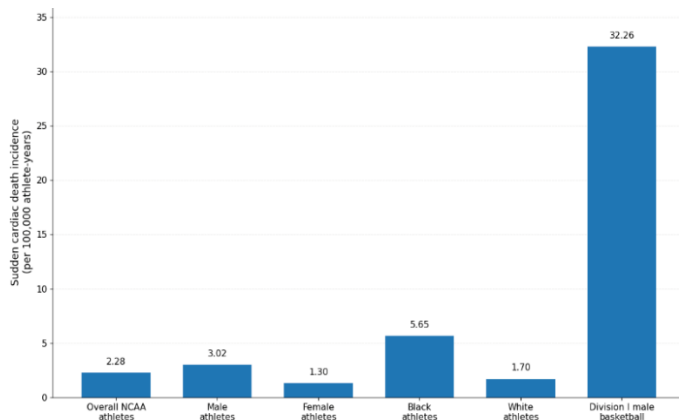


Figure 1 shows the marked heterogeneity in the incidence of sudden cardiac death (SCD) among different subgroups of collegiate athletes. Although the overall incidence in the NCAA population analyzed by Harmon et al. (2011) was approximately **2.28 cases per 100,000 athlete-years**, equivalent to approximately one SCD for every 43,770 athlete-years, the distribution varied substantially according to sex, race, and sporting discipline. This variation is relevant when describing the epidemiology of SCD because the overall population rate can conceal subgroups in which the observed frequency is several times higher. Harmon et al. (2011) reported 45 cardiovascular-related sudden deaths in their NCAA analysis and demonstrated that incidence differed considerably between demographic and sport-specific categories.

When athletes were stratified according to sex, the incidence was approximately **3.02 per 100,000 athlete-years in males**, compared with **1.30 per 100,000 athlete-years in females**. Expressed in the original denominators, this corresponds to approximately one event per 33,134 athlete-years in men and one per 76,646 athlete-years in women.

Therefore, the observed incidence among male athletes was approximately **2.3 times that of female athletes**. The data demonstrate that sex constitutes an important descriptive variable when characterizing the epidemiological distribution of SCD in competitive athletic populations; however, these figures alone do not establish the biological or behavioral mechanisms responsible for this difference (Harmon et al., 2011).

An even greater difference was observed when the population was stratified by race. Black athletes presented an incidence of approximately **5.65 SCD events per 100,000 athlete-years**, whereas White athletes showed approximately **1.70 events per 100,000 athlete-years**. These values correspond to approximately one event per 17,696 athlete-years among Black athletes and one per 58,653 athlete-years among White athletes. Thus, within the population analyzed, the incidence among Black athletes was approximately **3.3 times higher** than that observed among White athletes. Harmon et al. (2011) identified this difference as one of the most relevant demographic variations in their NCAA cohort.

The most pronounced finding in the figure is observed among **Division I male basketball players**, with an incidence of approximately **32.26 SCD events per 100,000 athlete-years**, corresponding to roughly one event per 3,100 athlete-years in the original study. This value is substantially higher than those observed in the other categories represented in the graph. Descriptively, the rate is approximately **14 times the overall NCAA incidence** and more than 10 times the incidence observed when all male athletes are considered together. Basketball was consequently identified as the sport with the highest SCD risk in the analyzed NCAA population, with Division I male basketball representing a particularly elevated subgroup (Harmon et al., 2011).

The magnitude of this difference is also important because it demonstrates that reporting a single incidence for all athletes provides only a partial representation of the epidemiological pattern. An overall rate of 2.28 per 100,000 athlete-years could suggest a relatively uniform low-frequency event; however, stratification reveals values ranging from approximately **1.30 per 100,000 athlete-years in female athletes to 32.26 per 100,000 athlete-years in Division I male basketball players**. The difference between the lowest and highest categories represented in the figure is therefore nearly 25-fold.

These findings are consistent with subsequent epidemiological evaluations. A more recent 20-year analysis of NCAA athletes covering 2002–2022 identified 143 SCD cases among 1,102 total deaths and again demonstrated higher incidence in male athletes, Black athletes, and basketball players. In that contemporary cohort, SCD remained the most common medical cause of death, although its overall incidence declined over time. The persistence of demographic and sport-specific differences across different observation periods supports the reproducibility of the general distribution shown in Figure 1, even though absolute incidence estimates vary according to study period and case-identification methodology (Petek et al., 2024).

An important methodological aspect when interpreting these results is that incidence estimates for SCD depend strongly on the accuracy with which both the number of events and the population exposed to risk are identified. Studies using insurance claims alone, passive registries, media reports, or institutional databases may identify different numbers of cases. The more recent NCAA analysis illustrates this issue: different surveillance sources detected substantially different proportions of confirmed SCD events, supporting the use of multiple complementary sources when estimating incidence (Petek et al., 2024).

Therefore, the principal descriptive finding of Figure 1 is the **nonuniform distribution of sudden cardiac death within the athletic population**. The overall incidence remains relatively low, but the data reveal progressively higher values in male athletes, Black athletes, and particularly Division I male basketball players. These epidemiological differences provide a quantitative basis for the subsequent analysis of cardiovascular substrates, electrocardiographic abnormalities, and risk-stratification findings presented in the following figures.

Figure 2.

Distribution of the principal cardiovascular causes associated with sudden cardiac death in athletes.

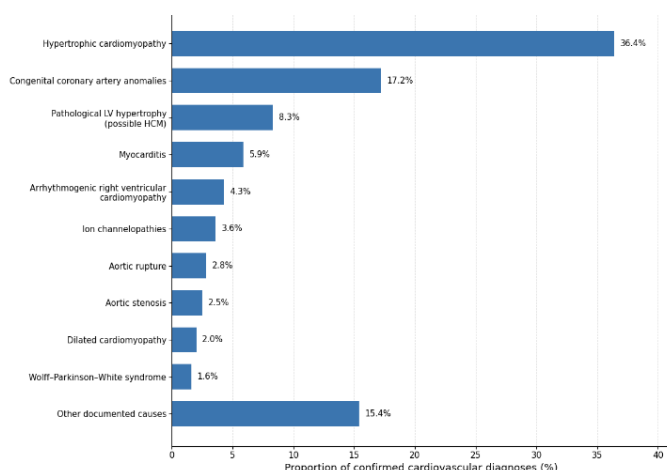


Figure 2 presents the distribution of the principal confirmed cardiovascular diagnoses associated with sudden cardiac death (SCD) in young competitive athletes. The data are based on the large United States registry analyzed by Maron et al. (2009), which identified 1,866 sudden death events over a 27-year period. Of these events, 1,049 were considered probably or definitely cardiovascular in origin, and 690 had a sufficiently documented cardiovascular diagnosis to permit etiological classification. Within this confirmed diagnostic group, hypertrophic cardiomyopathy represented the most frequent individual condition, accounting for approximately **36.4%** of cases (Maron et al., 2009).

Hypertrophic cardiomyopathy clearly occupies the dominant position in the figure, with a proportion more than twice that of congenital coronary artery anomalies. This finding is consistent with historical North American registries in which hypertrophic cardiomyopathy has repeatedly appeared as one of the leading structural substrates associated with sudden death in young athletes. In an earlier series, Maron et al. (1996) also reported hypertrophic cardiomyopathy in approximately 36% of cardiovascular deaths, indicating a remarkable consistency in its relative frequency across different periods of observation.

The second most frequent category was **congenital coronary artery anomalies**, representing approximately **17.2%** of confirmed diagnoses. These abnormalities are clinically important because an athlete may remain asymptomatic until intense physical exertion increases myocardial oxygen demand or creates unfavorable coronary flow conditions. Their substantial representation in the registry demonstrates that SCD in athletes cannot be interpreted exclusively as a consequence of primary myocardial disease or electrical disorders. Instead, abnormalities of coronary anatomy represent an important etiological group that may not always be identifiable through resting electrocardiography alone (Maron et al., 2009).

A further **8.3%** of cases were categorized as pathological left ventricular hypertrophy potentially related to hypertrophic cardiomyopathy. This group is particularly relevant because morphological overlap can exist between physiological training-induced ventricular remodeling and pathological hypertrophy. The presence of substantial ventricular wall thickening therefore requires interpretation in relation to body size, sporting discipline, ventricular geometry, family history, symptoms, ECG abnormalities, and complementary imaging findings. When definite hypertrophic cardiomyopathy and pathological left ventricular hypertrophy potentially related to this disease are considered together, hypertrophic phenotypes account for a particularly large proportion of documented cardiovascular deaths in the registry (Maron et al., 2009).

Myocarditis accounted for approximately 5.9% of confirmed cardiovascular diagnoses. Although less frequent than cardiomyopathy or coronary anomalies, myocarditis is relevant in the athletic population because myocardial inflammation may produce electrical instability, ventricular dysfunction, or myocardial scar capable of facilitating ventricular arrhythmias. Its presence in the registry illustrates that acquired cardiovascular disease also contributes to SCD and that risk is not restricted to congenital or inherited disorders. The clinical presentation of myocarditis can also vary considerably, ranging from symptomatic inflammatory disease to subtle or initially unrecognized myocardial involvement (Maron et al., 2009).

Arrhythmogenic right ventricular cardiomyopathy represented approximately **4.3%** of confirmed cases in the United States registry. Although this proportion is lower than that reported in some European populations, the finding illustrates the geographical variability in the etiological distribution of SCD. For example, Italian studies have reported a considerably greater contribution of arrhythmogenic right ventricular cardiomyopathy, highlighting the influence of population characteristics, genetic background, diagnostic criteria, screening programs, and postmortem methodology on the observed distribution of cardiovascular causes (Corrado et al., 1998; Maron et al., 2009).

Primary **ion channelopathies** accounted for approximately **3.6%** of documented diagnoses. This proportion should be interpreted carefully because disorders such as long QT syndrome, Brugada syndrome, and catecholaminergic polymorphic ventricular tachycardia may occur in structurally normal hearts. Consequently, conventional autopsy can fail to identify a definitive cause, particularly in historical registries in which postmortem genetic analysis was not routinely available. The observed percentage may therefore underestimate the contribution of inherited electrical diseases to sudden unexplained death in athletes (Maron et al., 2009).

Less frequent but clinically identifiable causes included **aortic rupture (2.8%)**, **aortic stenosis (2.5%)**, **dilated cardiomyopathy (2.0%)**, and **Wolff-Parkinson-White syndrome (1.6%)**. Although each of these conditions accounted for a relatively small proportion individually, together they demonstrate the broad spectrum of cardiovascular disorders capable of preceding sudden death. Their presence also emphasizes that cardiovascular evaluation cannot focus exclusively on a single disease or diagnostic modality. Structural, valvular, vascular, myocardial, and electrophysiological abnormalities all contribute to the observed burden of events (Maron et al., 2009).

The category labeled **“Other documented causes”** accounted for approximately **15.4%** of diagnoses and represents the remaining less frequent cardiovascular conditions described within the registry. Its size illustrates the etiological diversity of SCD in athletes: even though several diseases account for a substantial proportion of cases, no single pathological mechanism explains all events. This heterogeneity is one reason why contemporary preparticipation assessment relies on sequential evaluation rather than on a single universal diagnostic test.

The distribution also changes when different populations and surveillance systems are analyzed. For example, the Italian experience reported arrhythmogenic right ventricular cardiomyopathy as the most frequent cause of sudden death among screened competitive athletes, whereas hypertrophic cardiomyopathy represented a substantially smaller proportion than in the United States registry. In the Italian cohort, arrhythmogenic right ventricular cardiomyopathy accounted for approximately 22.4% of sudden deaths in athletes, coronary atherosclerosis for 18.4%, and anomalous coronary origin for 12.2%, while hypertrophic cardiomyopathy represented only 2% (Corrado et al., 1998).

Therefore, Figure 2 demonstrates that the cardiovascular substrate underlying SCD in athletes is **multifactorial and heterogeneous**, although hypertrophic cardiomyopathy and congenital coronary artery anomalies represented the predominant diagnoses in the United States registry analyzed. Structural myocardial disorders accounted for an important proportion of confirmed cases, but inflammatory, electrical, vascular, valvular, and congenital abnormalities were also represented. These findings provide the etiological basis for evaluating which abnormalities can potentially be identified during cardiovascular preparticipation screening and which findings require complementary testing.

Figure 3.

Classification of electrocardiographic findings during preparticipation cardiovascular screening.

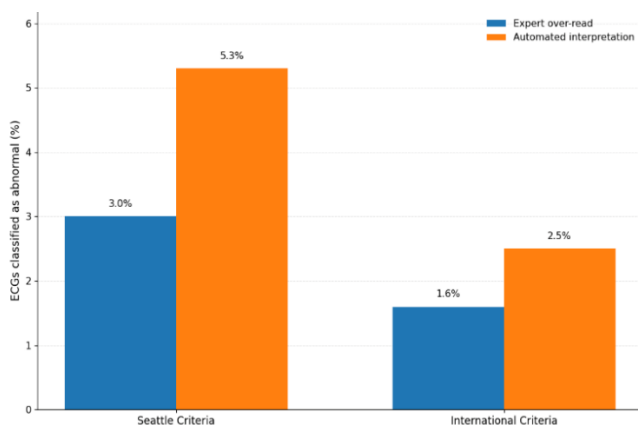


Figure 3 presents the proportion of electrocardiograms classified as abnormal during cardiovascular preparticipation screening according to two athlete-specific interpretation systems: the **Seattle Criteria** and the **International Criteria**. The data correspond to an analysis of **5,258 NCAA athletes**, whose ECGs were retrospectively evaluated by experts in sports cardiology and by an automated interpretation system. The comparison demonstrates a clear reduction in the proportion of ECGs considered abnormal when the International Criteria were applied instead of the earlier Seattle Criteria (Hyde et al., 2019).

With expert interpretation, **3.0% of ECGs were classified as abnormal using the Seattle Criteria**, corresponding to 158 of 5,258 athletes. When the same ECGs were reassessed using the International Criteria, only **1.6%** were classified as abnormal, corresponding to 83 athletes. This represents an absolute reduction of **1.4 percentage points** and an approximate **46.7% relative reduction** in abnormal ECG classifications. The difference was statistically significant ($p < 0.0001$) (Hyde et al., 2019).

The same direction of change was observed when electrocardiograms were evaluated through automated interpretation. Under the Seattle Criteria, the automated system classified **5.3%** of ECGs as abnormal, whereas application of the International Criteria reduced this proportion to **2.5%**. In absolute terms, the number of ECGs classified as abnormal decreased from 278 to 134. This corresponds to an approximate **52.8% relative reduction**, again with a statistically significant difference ($p < 0.0001$) (Hyde et al., 2019).

The figure also demonstrates a persistent difference between expert and automated ECG interpretation. Under the Seattle Criteria, automated assessment classified 5.3% of ECGs as abnormal compared with 3.0% after expert over-read, a difference of 2.3 percentage points. With the International Criteria, the respective values were 2.5% and 1.6%, reducing the absolute difference between the two interpretation approaches to 0.9 percentage points. Therefore, although both methods showed fewer abnormal classifications with the International Criteria, expert interpretation consistently resulted in a lower proportion of ECGs being labeled as abnormal.

This difference is further reflected in false-positive classifications. Using the International Criteria, the automated system had a reported false-positive rate of **2.3%**, compared with **1.3% for expert interpretation**, and this difference was statistically significant ($p = 0.0001$) (Hyde et al., 2019). This finding is particularly relevant to the descriptive evaluation of preparticipation screening because physiological cardiac adaptations to exercise may produce ECG patterns that differ substantially from those observed in sedentary populations.

An important result is that the reduction in abnormal ECG classification did **not result in failure to identify the athletes with clinically relevant cardiac pathology** within the study. All four combinations of criteria and interpretation approaches identified the **13 athletes with cardiac conditions associated with sudden cardiac death**. Thus, the numerical reduction from 3.0% to 1.6% with expert assessment reflected primarily a change in the classification of findings that did not ultimately correspond to the cardiovascular pathologies identified in the screened cohort (Hyde et al., 2019).

Analysis of the specific electrocardiographic abnormalities provides additional detail. Among ECGs considered abnormal by expert assessment according to the International Criteria, **T-wave inversion was the most frequently identified abnormality**, occurring in 40 cases and representing approximately **48% of abnormal ECGs**. T-wave inversion is particularly relevant within athlete-specific ECG interpretation because its significance depends on factors including lead distribution, age, ethnicity, and accompanying cardiovascular findings. The International Criteria provide standardized definitions to differentiate potentially physiological patterns from those warranting further investigation (Drezner et al., 2017; Hyde et al., 2019).

One of the largest changes produced by the updated interpretation criteria involved **pathological Q waves**. Under the Seattle Criteria, 69 ECGs were classified as abnormal because of Q-wave findings, whereas only 11 met the revised definition incorporated into the International Criteria. This represented an **84% reduction** in ECGs categorized as abnormal for this specific characteristic ($p < 0.0001$) (Hyde et al., 2019). The magnitude of this reduction demonstrates how modifications in the diagnostic thresholds applied to individual ECG features can substantially change the overall frequency of positive screening findings.

Comparable results have been reported in other athlete populations. In a separate cohort of **1,686 NCAA Division I athletes**, application of the Seattle, Refined, and International Criteria generated abnormal ECG rates of **3.0%, 2.1%, and 1.8%**, respectively. Only three athletes, approximately 0.2% of the cohort, were ultimately identified with cardiac conditions associated with SCD. In that analysis, the International Criteria also demonstrated the lowest false-positive rate among the athlete-specific ECG interpretation systems evaluated.

Earlier evidence similarly demonstrated the progressive reduction of abnormal ECG classifications as athlete-specific interpretation systems evolved. One study found that approximately **17.3%** of elite athletes would have been classified as having an abnormal ECG using older ESC recommendations, whereas application of the Seattle Criteria reduced the proportion to **4.5%**, while still identifying the athletes with confirmed cardiac abnormalities. Subsequently, the Refined and International Criteria further modified the classification of training-related, borderline, and abnormal findings (Drezner et al., 2017).

Taken together, the numerical pattern in Figure 3 shows a progressive increase in the specificity of ECG interpretation for athletic populations. The International Criteria produced approximately half as many abnormal classifications as the Seattle Criteria both with expert assessment and automated interpretation. At the same time, the clinically significant cardiovascular pathologies present in the analyzed cohort remained identifiable. The results therefore distinguish between the relatively frequent presence of training-associated electrical variations and the much smaller proportion of athletes in whom screening findings correspond to clinically significant cardiovascular disease.

From the perspective of the present study, Figure 3 complements the epidemiological findings presented in Figures 1 and 2. Whereas the previous figures demonstrate that SCD is heterogeneously distributed and may result from multiple cardiovascular substrates, Figure 3 quantifies how the method used to interpret the **12-lead ECG** substantially modifies the number of athletes referred as having an abnormal screening result. This establishes the quantitative basis for the subsequent assessment of arrhythmic patterns and their relationship with exercise and underlying cardiovascular disease.

Figure 4.

Relative clinical relevance of arrhythmic findings according to exercise behavior and cardiovascular substrate.

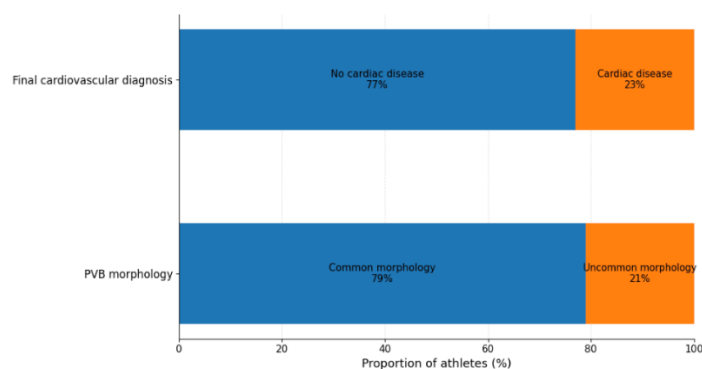


Figure 4 summarizes the principal findings of the prospective cohort reported by Di Florio et al. (2021), which evaluated **112 competitive athletes younger than 40 years** referred for premature ventricular beats (PVBs). The results show that ventricular ectopy in athletes is heterogeneous and that its clinical relevance cannot be determined solely by the presence or numerical burden of premature beats. Instead, morphology, complexity, exercise response, and the presence of an underlying structural or electrical cardiovascular substrate are particularly important for risk stratification.

The first distribution demonstrates that **79% of athletes presented a common PVB morphology**, principally infundibular or fascicular patterns, whereas approximately **21% exhibited uncommon morphologies**. Common patterns are generally characterized by an origin compatible with the ventricular outflow tract or fascicular system. These morphologies frequently occur in individuals without demonstrable structural cardiovascular disease and therefore constitute an important category when distinguishing potentially benign ventricular ectopy from findings requiring more extensive investigation.

Nevertheless, the predominance of common morphology does not imply that all ventricular premature beats detected in athletes are physiologically irrelevant. After physical examination, resting ECG, 12-lead ambulatory ECG monitoring, exercise testing, echocardiography, and additional investigations when indicated, **26 of the 112 athletes received a definitive diagnosis of cardiovascular disease**, corresponding to approximately **23% of the cohort**. Conversely, **77% had no underlying cardiac disease identified** following the diagnostic evaluation.

It is important to interpret the two bars independently: the 21% of uncommon PVB morphologies and the 23% of athletes diagnosed with cardiovascular disease are not intended to represent identical individuals. Rather, the graphical comparison demonstrates that uncommon morphology represented a minority of observed ventricular ectopy patterns, while approximately one in four athletes referred for ventricular ectopy ultimately had an identifiable cardiac disorder. The study found a statistically significant relationship between **uncommon PVB morphology and underlying cardiac disease ($p < 0.001$)**.

Arrhythmia complexity was also significantly associated with cardiovascular disease ($p < 0.001$). Therefore, the diagnostic value of ventricular ectopy increased when the rhythm disturbance demonstrated features such as multiple morphologies, repetitive ventricular beats, couplets, triplets, or nonsustained ventricular tachycardia rather than isolated monomorphic premature beats. These findings indicate that qualitative characteristics of ventricular arrhythmias provide more clinically informative data than simply counting the total number of premature ventricular beats.

An especially relevant finding was that the **24-hour PVB burden was actually lower among athletes with diagnosed cardiovascular disease than among athletes with structurally normal hearts ($p < 0.05$)**. This observation demonstrates that a high numerical burden is not necessarily synonymous with a more dangerous arrhythmogenic substrate. Conversely, a smaller number of premature beats may warrant further evaluation when their morphology or complexity is atypical. Accordingly, the study identified morphology and complexity, rather than absolute PVB number, as the variables most closely associated with underlying cardiovascular disease.

These results are consistent with contemporary electrophysiological recommendations. The 2024 Heart Rhythm Society consensus distinguishes typical ventricular premature complexes—particularly outflow tract or fascicular morphologies—from higher-risk patterns characterized by atypical morphology, multifocal origin, short coupling intervals, repetitive ventricular activity, or nonsustained ventricular tachycardia. In athletes presenting with ventricular arrhythmias, resting ECG, ambulatory monitoring, and exercise testing are recommended components of evaluation, while higher-risk findings justify comprehensive cardiac imaging, including cardiac magnetic resonance when appropriate (Lampert et al., 2024).

The behavior of ventricular arrhythmias during physical exertion provides another relevant level of characterization. Current HRS recommendations emphasize maximal-effort exercise testing to reproduce symptoms and determine the occurrence, morphology, and complexity of ventricular arrhythmias. Suppression of premature ventricular beats during exercise is generally less strongly associated with structural heart disease, whereas persistence, induction, increasing complexity, or atypical patterns during exertion can justify more extensive cardiovascular assessment.

The need to characterize these findings is also supported by studies of healthy athletic populations. In athletes without known cardiovascular disease undergoing 24-hour ambulatory ECG monitoring, most individuals demonstrate either no ventricular ectopy or a very low burden; more than 90% have no couplets, triplets, or nonsustained ventricular tachycardia longer than three beats. Thus, complex ventricular arrhythmias represent a less common pattern and acquire greater relevance when identified during cardiovascular screening.

The prospective cohort represented in Figure 4 also provided follow-up information. Athletes in whom the cardiovascular investigation was negative demonstrated a **spontaneous reduction in premature ventricular beats and no adverse cardiovascular events during follow-up**. This finding further differentiates isolated ventricular ectopy in a normal heart from ventricular arrhythmias associated with structural or electrical pathology.

DISCUSIÓN

The findings of this study confirm that arrhythmias and sudden cardiac death (SCD) in athletes cannot be adequately understood through a single diagnostic variable or a generalized estimate of cardiovascular risk. Instead, the available evidence supports a multidimensional approach integrating epidemiological characteristics, underlying structural or electrical disease, athlete-specific electrocardiographic interpretation, exercise-related arrhythmic behavior, and perioperative factors. This integrated perspective is particularly relevant because athletic training produces physiological cardiovascular adaptations that may resemble disease, while clinically significant abnormalities may remain silent until exposed to intense exercise or perioperative stress.

The epidemiological results demonstrated that SCD is relatively uncommon in the overall athletic population but is distributed unevenly among subgroups. The higher incidence observed in male athletes, Black athletes, and particularly Division I male basketball players indicates that global population estimates may underestimate risk within selected groups. Harmon et al. (2011, 2015) consistently reported meaningful differences according to sex, race, and sport, supporting the concept that risk assessment should consider demographic and activity-specific characteristics rather than apply identical assumptions to all athletes.

This heterogeneity is relevant to preparticipation evaluation because it suggests that cardiovascular screening should be sufficiently sensitive to recognize individuals who belong to populations with higher observed event rates. However, higher epidemiological incidence alone should not be considered equivalent to the presence of cardiovascular disease. Rather, these variables should be integrated with symptoms, family history, physical examination findings, and objective cardiovascular testing. Such an approach reduces the risk of both underdiagnosis and excessive restriction of athletes without pathological findings.

The analysis of the causes of SCD further illustrates the complexity of the problem. Hypertrophic cardiomyopathy and congenital coronary artery anomalies represented important causes in historical North American registries, whereas other studies have demonstrated different etiological distributions according to geography and population characteristics (Maron et al., 1996; Maron et al., 2009). Arrhythmogenic cardiomyopathy, myocarditis,

channelopathies, aortic disorders, and other structural abnormalities also contribute to the overall burden. Consequently, no single diagnostic examination can identify every potentially lethal substrate.

Differences between historical and contemporary studies should also be interpreted in the context of methodological evolution. Improvements in cardiac magnetic resonance imaging, molecular autopsy, genetic testing, pathological classification, and recognition of inherited electrical disorders have changed the manner in which unexplained sudden deaths are categorized. Conditions that were previously considered idiopathic may now be linked to channelopathies or concealed cardiomyopathies. This is particularly important in athletes who die suddenly with a structurally normal heart at conventional autopsy, because an electrical disorder may remain undetected without genetic or specialized electrophysiological assessment.

The findings related to ECG screening support the importance of athlete-specific interpretation standards. The progressive transition from older criteria to the Seattle, Refined, and International Criteria has substantially reduced the proportion of athletes classified as having abnormal ECGs while maintaining the ability to identify clinically relevant cardiovascular disease (Drezner et al., 2017). In the results analyzed in this study, application of the International Criteria produced fewer abnormal classifications than the Seattle Criteria, particularly when interpretation was performed by trained experts.

This reduction is clinically relevant because false-positive results may lead to unnecessary echocardiography, magnetic resonance imaging, exercise testing, temporary restriction from sports, anxiety, and additional healthcare costs. The objective of preparticipation screening should therefore not be maximal detection of every electrical variation but accurate identification of findings associated with meaningful cardiovascular pathology. Athlete-specific ECG interpretation contributes directly to this objective by recognizing that sinus bradycardia, early repolarization, isolated voltage criteria for ventricular hypertrophy, and certain conduction patterns may represent normal consequences of training rather than disease.

At the same time, the reduction of false-positive findings must not result in diminished attention to pathological patterns. T-wave inversion in specific distributions, ventricular pre-excitation, pathological Q waves, prolonged QT intervals, Brugada-type patterns, profound conduction abnormalities, or complex ventricular ectopy remain findings that may require additional investigation (Drezner et al., 2017). Therefore, the principal contribution of standardized ECG criteria is not simply to classify fewer ECGs as abnormal but to improve the distinction between physiological adaptation and patterns with a higher probability of disease.

The results regarding ventricular premature beats provide another important element of this distinction. The analyzed evidence demonstrates that arrhythmic burden alone does not adequately predict the presence of structural or electrical heart disease. Athletes with numerous ventricular premature beats may have no detectable cardiovascular pathology, whereas athletes with a lower absolute number of ectopic beats may have clinically significant disease when the morphology is uncommon, multifocal, complex, or induced by exercise.

This finding has direct implications for clinical interpretation. Traditional approaches that emphasize numerical thresholds may overlook the importance of qualitative arrhythmic characteristics. Contemporary recommendations increasingly emphasize morphology, coupling interval, complexity, exercise behavior, symptoms, and imaging findings when assessing ventricular arrhythmias in athletes (Lampert et al., 2024). Thus, a single number obtained from ambulatory monitoring should not be interpreted independently from the broader cardiovascular context.

Exercise testing assumes particular importance in this setting because it allows the arrhythmia to be evaluated under physiological stress. Suppression of isolated ventricular ectopy during increasing exercise intensity generally differs clinically from the appearance or progression of complex ventricular arrhythmias during exertion. Exercise-induced polymorphic ventricular ectopy or ventricular tachycardia may suggest a more significant arrhythmogenic substrate and warrants additional evaluation. This concept reinforces the role of functional testing as an extension of the resting cardiovascular examination rather than as an isolated performance assessment.

Cardiac magnetic resonance imaging also becomes relevant when ventricular arrhythmias are associated with abnormal morphology, concerning symptoms, or suspicious findings on echocardiography or ECG. The ability to detect myocardial fibrosis, scar, inflammatory changes, or subtle ventricular abnormalities may identify disease that remains

undetectable through conventional echocardiography. This is especially important in suspected arrhythmogenic cardiomyopathy or previous myocarditis, both of which may provide a substrate for ventricular arrhythmia and SCD.

The contemporary approach to athletes with documented arrhythmias has also evolved substantially. Earlier guidelines frequently emphasized categorical eligibility or disqualification, whereas more recent consensus statements favor individualized risk assessment and shared decision-making (Lampert et al., 2024). This shift recognizes that cardiovascular diagnoses exist across a spectrum of severity and that the risk associated with sports participation depends on the specific disease, treatment response, arrhythmia control, ventricular function, sport characteristics, and athlete preferences.

This individualized approach is important because unnecessary exclusion from physical activity may produce significant consequences, including deconditioning, psychological distress, loss of athletic identity, and the absence of the well-established health benefits associated with exercise. At the same time, shared decision-making should not be interpreted as minimizing risk. Rather, it requires transparent communication regarding the probability and potential severity of adverse events, available preventive strategies, treatment alternatives, and emergency preparedness.

The perioperative findings extend this individualized approach beyond the sports environment. A physically fit athlete should not automatically be considered at negligible perioperative cardiovascular risk. High functional capacity is reassuring in many circumstances, but it does not eliminate the clinical significance of a known channelopathy, cardiomyopathy, ventricular arrhythmia, previous ablation, implanted device, or history of unexplained syncope. Athletic status and cardiovascular risk therefore represent related but distinct clinical dimensions.

The high frequency of perioperative arrhythmias reported in general surgical populations must also be interpreted carefully. Many detected rhythm disturbances are brief, asymptomatic, and clinically benign, particularly when continuous monitoring is used (Pecha et al., 2023). Consequently, the high percentages observed in some surgical settings should not be equated with a comparable incidence of malignant ventricular arrhythmias or sudden cardiac death. Nevertheless, the perioperative period provides multiple potential triggers capable of destabilizing a susceptible electrophysiological substrate.

These triggers include electrolyte disturbances, hypoxemia, hypercapnia, acid-base abnormalities, sympathetic activation, vagal stimulation, blood loss, major volume shifts, hypothermia, fever, postoperative pain, and pharmacological effects. Their relevance varies according to the underlying cardiovascular condition. For example, an athlete with long QT syndrome may be particularly sensitive to QT-prolonging medications and electrolyte abnormalities, whereas an individual with catecholaminergic polymorphic ventricular tachycardia may be more vulnerable to adrenergic stimulation. Similarly, fever and selected drugs may be especially relevant in patients with Brugada syndrome.

The perioperative assessment of athletes with known arrhythmias should therefore focus on the mechanism of the underlying disorder rather than on the athlete's level of conditioning alone. The 2024 perioperative cardiovascular guidelines recommend a stepwise evaluation that considers active cardiovascular disease, functional capacity, surgical risk, and whether additional testing is expected to alter management (Thompson et al., 2024). This framework is particularly applicable to athletes because unnecessary preoperative testing should be avoided in asymptomatic individuals with excellent functional capacity, while relevant warning signs should not be dismissed because of high performance status.

An additional consideration is the management of cardiovascular medications. Beta-blockers, antiarrhythmic drugs, and anticoagulants may substantially influence perioperative planning. Abrupt withdrawal of certain medications may increase arrhythmic or hemodynamic risk, while continuation may also modify heart rate, blood pressure, conduction, and anesthetic responses. Athletes who have undergone catheter ablation or who carry pacemakers or implantable cardioverter-defibrillators require further individualized assessment according to device function, arrhythmic history, and the characteristics of the surgical procedure.

The results also reinforce the importance of maintaining continuity between sports cardiology and perioperative medicine. Traditionally, preparticipation screening and perioperative cardiovascular assessment have been addressed as separate clinical processes. However, an athlete identified with an abnormal ECG, ventricular arrhythmia,

cardiomyopathy, or channelopathy during sports evaluation may later require anesthesia or surgery. Information obtained during sports cardiology assessment can therefore become directly relevant to perioperative risk stratification.

The findings support the hypothesis proposed in this study that a structured multimodal assessment can improve the identification of athletes with clinically meaningful cardiovascular risk. History and physical examination remain essential, but they are strengthened by athlete-specific ECG interpretation and selective use of ambulatory monitoring, exercise testing, imaging, and genetic evaluation. No individual test provides sufficient diagnostic accuracy across all conditions associated with SCD.

The second hypothesis is also supported by the reviewed evidence: perioperative management should be individualized according to the specific electrophysiological and structural substrate. The presence of an arrhythmia is not sufficient by itself to define perioperative risk. Its clinical meaning depends on the diagnosis responsible for the arrhythmia, its relationship with exercise, associated ventricular dysfunction, previous symptoms, treatment history, and exposure to perioperative triggers.

Several limitations should be considered when interpreting these findings. First, the reported incidence of SCD varies considerably between studies because of differences in case definitions, surveillance systems, denominator estimation, population characteristics, and duration of follow-up. Second, historical autopsy registries may underestimate channelopathies and concealed cardiomyopathies because molecular and advanced imaging techniques were not routinely available. Third, much of the perioperative evidence derives from general surgical populations rather than specifically from competitive athletes. Therefore, perioperative arrhythmia frequencies cannot be directly interpreted as athlete-specific risk estimates.

Another limitation is the heterogeneity of athletic populations. Recreational athletes, collegiate competitors, professional athletes, endurance athletes, strength athletes, and master athletes differ substantially in age, training exposure, cardiovascular remodeling, and prevalence of acquired cardiovascular disease. In older athletes, coronary atherosclerosis becomes increasingly relevant, whereas inherited structural or electrical abnormalities predominate among many younger athletes. Future research should therefore stratify cardiovascular risk according to both age and type of athletic participation.

Prospective multicenter studies are also needed to determine more precisely which combinations of clinical, electrocardiographic, imaging, and genetic variables best predict clinically significant arrhythmias and SCD. Similar research is warranted in the perioperative context, where athlete-specific evidence remains limited. Integration of long-term ambulatory monitoring, exercise-based assessment, cardiac imaging, and genetic information may contribute to more precise individualized risk models.

CONCLUSIÓN

Arrhythmias and sudden cardiac death in athletes represent a clinically complex problem in which the low absolute frequency of catastrophic events contrasts with their substantial medical and social impact. The evidence analyzed in this study demonstrates that cardiovascular risk is not uniformly distributed among athletes and is influenced by sex, ethnicity, type of sport, underlying structural or electrical disease, arrhythmic characteristics, and exposure to physiological or perioperative stressors.

Preparticipation cardiovascular evaluation remains a fundamental component of prevention. A structured assessment based on personal and family history, physical examination, athlete-specific electrocardiographic interpretation, and selective complementary testing allows physiological adaptations to exercise to be distinguished more accurately from potentially pathological findings. The use of contemporary ECG interpretation criteria reduces unnecessary abnormal classifications while maintaining the capacity to identify conditions associated with sudden cardiac death.

The analysis of ventricular arrhythmias further demonstrates that their clinical significance cannot be determined solely by numerical burden. Morphology, complexity, relationship with exercise, associated symptoms, and evidence of structural myocardial disease provide more relevant information for risk stratification. Uncommon or complex

ventricular ectopy, exercise-induced arrhythmias, and abnormalities associated with cardiomyopathy, myocardial fibrosis, channelopathies, or ventricular dysfunction require a more comprehensive diagnostic approach.

The findings also support a transition from rigid sports disqualification toward individualized risk assessment and shared decision-making. Athletic participation should be evaluated according to the specific cardiovascular diagnosis, disease severity, treatment response, arrhythmia control, characteristics of the sport, and individual circumstances of the athlete. This approach makes it possible to preserve the benefits of physical activity while maintaining appropriate cardiovascular safety.

In the perioperative setting, high physical performance should not be interpreted as protection against arrhythmic complications. Athletes with known or suspected cardiovascular abnormalities may remain vulnerable to autonomic fluctuations, electrolyte disturbances, hypoxemia, medications, anesthetic agents, surgical stress, and hemodynamic changes. Perioperative management should therefore be based on the underlying electrophysiological or structural substrate and should include appropriate monitoring, correction of modifiable risk factors, medication review, and multidisciplinary planning when necessary.

The results support the principal hypothesis of this study: an integrated cardiovascular strategy combining preparticipation screening, athlete-specific ECG interpretation, targeted diagnostic testing, arrhythmic characterization, and individualized perioperative assessment provides a more effective framework for identifying athletes at increased cardiovascular risk. They also support the complementary hypothesis that perioperative management adapted to the specific arrhythmogenic mechanism can improve clinical safety by anticipating potentially preventable triggers.

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