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Cardiac Arrhythmias in Athletes and Their Impact on Health and Sports Participation: A Narrative Review

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Abstract:

Background: Sport and physical activity induce structural, functional, and electrical adaptations of the heart, known as the “athlete’s heart.” Although usually physiological, these changes may overlap with pathological conditions and increase susceptibility to arrhythmias, including sudden cardiac death (SCD). Endurance and mixed sports particularly affect cardiac remodeling and electrophysiology.
Aim: To summarize common cardiac arrhythmias in athletes, describe their mechanisms, and discuss clinical implications for diagnosis, management, and prevention of sudden cardiac events.

Materials and Methods: A literature review was conducted based on recent studies and metaanalyses on cardiac adaptations and arrhythmias in athletes. Key topics included sinus bradycardia, atrioventricular (AV) block, premature ventricular contractions (PVCs), atrial fibrillation (AF), and arrhythmias linked to SCD, such as long QT syndrome, Brugada syndrome, Wolff–Parkinson–White syndrome, and arrhythmogenic right ventricular cardiomyopathy.
Results: Sinus bradycardia and AV block are common in endurance athletes and are mainly related to increased vagal tone. PVCs are usually benign but require evaluation. AF is more prevalent in endurance athletes and is associated with atrial enlargement and fibrosis. Rare arrhythmias related to channelopathies and cardiomyopathies significantly contribute to SCD risk. Diagnosis includes ECG, echocardiography, Holter monitoring, exercise testing, and cardiac MRI. Management ranges from observation to pharmacological therapy or invasive procedures.
Conclusions: Regular monitoring is essential to distinguish physiological adaptations from pathological arrhythmias. Early diagnosis and individualized management enable safe sports participation and reduce SCD risk.
Key Words: Athlete’s heart, Cardiac arrhythmias, Sudden cardiac death, Atrial fibrillation, Cardiomyopathy.

1. Introduction

Sport and physical activity are fundamental elements influencing human health and well-being, with both daily activity and participation in competitive sports contributing to overall wellness. Each type of physical activity can have long-term effects on health throughout life (Blake et al. 2022). Recent research analyzing trends in youth sports participation in the United States between 2011 and 2019 revealed notable declining patterns in involvement in organized sport activities across age and gender groups. These findings reflect shifts in engagement over time and underscore the importance of monitoring athletic participation trends in public health research (Deng and Fan 2022). Nevertheless, many individuals continue to engage in competitive sports, which have a significant impact on the human body, including the cardiovascular system. Competitive sports can lead to various changes in cardiac function. In the scientific literature the term “athlete’s heart” is used to describe the structural and functional adaptations of the heart resulting from the continuous influence of intense physical exertion. (Yilmaz et al. 2013) It refers to the physiological adaptation of the heart to long-term, regular physical training. It is not a disease, but a normal structural, functional, and electrical remodeling of the heart in response to increased cardiovascular demand. These changes include: enlargement of the cardiac chambers, increased thickness of the ventricular walls, improved systolic and diastolic function, molecular and electrical remodeling of the myocardium. (15). Adaptation of the left ventricle largely depends on the type of sport practiced. Endurance sports typically induce eccentric hypertrophy, characterized by increased left ventricular end-diastolic volume (LVEDV), while strength (static) sports (e.g., weightlifting, wrestling) often result in concentric hypertrophy, with wall thickening but normal LVEDV due to pressure overload. Mixed sports show a combination of these adaptations. Functional adaptations include improved diastolic function, resting bradycardia, and increased heart rate variability (Maxwell and Oxborough 2025). Left ventricular changes may resemble hypertrophic cardiomyopathy or other pathological conditions, which can complicate the differentiation between physiological

adaptation and underlying cardiac disease. (Abulí et al. 2020) Endurance training also leads to increases in right ventricular (RV) cavity volume, inflow and outflow volumes, and overall RV volumes, whereas strength-trained athletes show RV structural parameters similar to those of sedentary controls. Additionally, endurance athletes exhibit greater RV wall thickness compared to both strength athletes and inactive individuals (Maxwell and Oxborough 2025). In endurance athletes, bi-atrial dilation is common and, in the presence of normal ventricular filling pressures, is considered a normal variant of the "athlete's heart." Some studies have demonstrated a significant correlation between left atrial volume and aerobic capacity. In strength-trained athletes, findings regarding atrial size are less consistent; some studies show significant atrial dilation, while others report minimal differences compared to sedentary controls, particularly when indexed to body surface area (BSA). The most common atrial arrhythmia observed in athletes is atrial fibrillation (AF), which, although relatively rare, occurs more frequently in younger athletes (<55 years) and in those practicing endurance or mixed sports. Atrial adaptation and remodeling may contribute to the development of AF, although the underlying mechanisms are complex and multifactorial (Maxwell and Oxborough 2025). The abovementioned changes may contribute to the development of various cardiac conditions, including a possible increased susceptibility to other arrhythmias (both ventricular and atrial), myocarditis and myocardial fibrosis, as well as difficulties in distinguishing physiological adaptations from pathological alterations. (La Gerche et al. 2022) The most common arrhythmias associated with competitive sports, which will be the focus of this paper, may result from mechanisms such as electrical and molecular cardiac remodeling, myocarditis, myocardial fibrosis (scarring), gene– exercise interactions, and extreme structural remodeling induced by long-term intensive training. (La Gerche et al. 2022) Monitoring cardiac adaptations to exercise is of critical importance, as athletes may be at risk of sudden cardiac death. To evaluate these adaptations, a variety of imaging modalities are employed, including 2D echocardiography, Doppler techniques (including flow Doppler and Doppler myocardial imaging, DMI), speckle-tracking echocardiography (STE), 3D echocardiography, cardiac magnetic resonance (CMR), cardiac computed tomography (CCT), and hemodynamic forces (HDFs) analysis. Each method provides valuable information on cardiac structure, function, and hemodynamics, enabling a comprehensive assessment of physiological changes in the athlete's heart. (Zholshybek et al. 2023) Sudden cardiac death may be caused by structural, electrical, and acquired abnormalities of the heart, such as myocarditis and atherosclerosis in older athletes. (Emery et al. 2018)
The aim of this review is to summarize the most common cardiac arrhythmias observed in athletes, describe their underlying mechanisms, and discuss the clinical implications for diagnosis and prevention of sudden cardiac events.

2. Research Materials and Methods

2.1 Data collection.

The present review was conducted using a comprehensive search of peer-reviewed scientific literature focusing on cardiac arrhythmias in athletes, structural and functional adaptations of the heart, and their clinical implications. Relevant articles were identified primarily through PubMed and PubMed Central (PMC) databases, covering publications from 2000 to 2024. A total of 33 studies were included in the analysis.
The inclusion criteria encompassed original research articles, meta-analyses, and reviews reporting on prevalence and mechanisms of sinus bradycardia, atrioventricular block, premature ventricular contractions, atrial fibrillation, and arrhythmias associated with sudden cardiac death, cardiac structural and electrophysiological adaptations to endurance,

strength, and mixed sports and diagnostic methods including ECG interpretation, echocardiography, cardiac magnetic resonance imaging and electrophysiological studies.
The studies analyzed included diverse populations of athletes, ranging in age from adolescents to adults over 55 years. Both male and female athletes were included.
Data extracted from the studies included type and intensity of sport, sex, age, cardiac structural parameters, prevalence and type of arrhythmias, diagnostic modalities, and clinical outcomes. Emphasis was placed on differentiating physiological adaptations of the athlete's heart from pathologic conditions predisposing to arrhythmias and SCD.

2.2 AI.

AI was utilized for two specific purposes in this research. Text analysis of clinical reasoning narratives to identify linguistic patterns associated with specific logical fallacies. Assistance in refining the academic English language of the manuscript, ensuring clarity, consistency, and adherence to scientific writing standards. AI were used for additional linguistic refinement of the research manuscript, ensuring proper English grammar, style, and clarity in the presentation of results. It is important to emphasize that all AI tools were used strictly as assistive instruments under human supervision. The final interpretation of results, classification of errors, and conclusions were determined by human experts in clinical medicine and formal logic. The AI tools served primarily to enhance efficiency in data processing, pattern recognition, and linguistic refinement, rather than replacing human judgment in the analytical process.

3. Reserch results

3.1 Bradycardia and AV block I

Prevalence

Sinus bradycardia occurs in up to 90% of endurance athletes. Sinus pauses >2 s occur in approximately 30% of athletes. (Al-Othman et al. 2024) The greater the duration and intensity of endurance training, the higher the likelihood of its occurrence. It most commonly affects long-distance runners, triathletes, and cyclists. (Grandys et al. 2025) Atrioventricular (AV) node block occurs in approximately 40% of athletes. Bradycardia and AV blocks are usually asymptomatic. In the presence of symptoms, further diagnostic evaluation should be undertaken. (Al-Othman et al. 2024)

Mechanisms

The traditional theory explaining the occurrence of bradycardia and atrioventricular (AV) node blocks in athletes is based on high parasympathetic tone—specifically increased vagal activity. (Grandys et al. 2025) However, in studies in which the autonomic nervous system was blocked using atropine and beta-blockers, trained athletes continued to exhibit lower resting heart rates compared to sedentary individuals, indicating the presence of another concomitant mechanism. (Finocchiaro et al. 2026) Recent studies demonstrate intrinsic electrical remodeling of the sinus node and AV node, independent of the autonomic nervous system, leading to a reduction in intrinsic heart rate and prolongation of conduction through the atrioventricular node. The mechanism is based on decreased expression of ion channels (HCN4—responsible for the "funny" current If, as well as L- and T-type calcium channels)

in sinus and AV nodal cells. (AlOthman et al. 2024) In a longitudinal study conducted in 157 former elite athletes who, during periods of intensive competition, had resting heart rates below 50 beats per minute, it was shown that after more than five years of detraining and cessation of competition, as many as 65% still had reduced heart rates, and in 18% the resting heart rate remained below 50 beats per minute. (Finocchiaro et al. 2026)
Clinical implications:

In asymptomatic athletes, sinus bradycardia (resting heart rate >30 beats/min) usually does not require further diagnostic evaluation, except in individuals >35 years of age with an abnormal heart rate response to exercise. (Finocchiaro et al. 2026) In older athletes, long-term training, circadian rhythm, and aging may lead to symptomatic bradyarrhythmias and the need for pacemaker implantation. Symptoms are most often more pronounced at night. (Al-Othman et al. 2024) In athletes with a heart rate <30 beats/min and limited chronotropic response, the following investigations are recommended: complete blood count and electrolytes, thyroid function tests, transthoracic echocardiography (TTE), maximal exercise testing, and 24–48 h ambulatory ECG monitoring including a training session to assess chronotropic reserve and exclude sinus pauses or atrioventricular disturbances. (Finocchiaro et al. 2026) Importantly, in a study involving 66 athletes, ECG changes consistent with bradycardia and atrioventricular blocks were observed only in individuals participating in endurance sports. (Malhotra et al. 2015) Bradycardia may be exacerbated by medications or energy deficiency. (Finocchiaro et al. 2026) In young athletes, assessment of anti-Ro/SSA antibodies may be considered to detect congenital or acquired AV block. Studies conducted by Pietro Enea Lazzerini et al. demonstrated that advanced atrioventricular block (AVB) may occur in young athletes, and in most cases its presence is associated with anti-Ro/SSA antibodies that inhibit L-type calcium channels. (Lazzerini et al. 2024) Treatment is based on pacemaker implantation, which is the gold standard, reduction of physical training intensity, which in some cases results in slowing of heart rate and conduction, and potential use of ivabradine, which blocks the "funny" current (If). (Al-Othman et al. 2024) Participation in sports does not require restriction in asymptomatic individuals with isolated AVB; however, monitoring of block progression is necessary, particularly when PR interval is ≥400 ms in adults and ≥300 ms in children/adolescents. (Finocchiaro et al. 2026)

3.2 Premature Ventricular Contractions (PVCs)

Prevalence

The results of a study conducted by Sofia E. Gomez et al. in athletes aged 14–35 years showed that PVCs were rare, occurring in 0.24% of cases. In 96% of these, the morphology was benign (Gomez et al. 2024). In other studies, PVCs have been reported in 5–10% of athletes, regardless of the type of training performed (Zorzi et al. 2023). In rare cases, they may be a marker of heart disease and an increased risk of sudden cardiac death during exercise (Emery and Kovacs 2018).

Mechanisms

Most PVCs in athletes are idiopathic and benign. The mechanism involves enhanced spontaneous activity of cardiomyocytes, which generate an electrical impulse earlier than the physiological sinus rhythm (Emery and Kovacs 2018). The electrical impulse may also circulate through two pathways with different refractoriness, leading to premature depolarization (reentry), or differences in refractoriness across various regions of the heart

may result in "R-onT" type PVCs, which are risky for ventricular fibrillation (e.g., in myocardial infarction, Brugada syndrome, LQTS) (Zorzi et al. 2023). They most commonly originate from the right ventricular outflow tract or from the fascicles of the left ventricle. Such beats often decrease or disappear during exercise, which is a favorable prognostic feature (Emery and Kovacs 2018). The most frequently observed pattern is left bundle branch block (LBBB). Only a small number of cases exhibit atypical morphology requiring further diagnostics (Gomez et al. 2024). Additional ventricular beats may also result from cardiac remodeling typical of the athlete's heart. Changes such as myocardial remodeling, alterations in the autonomic nervous system (increased vagal activity), and electrophysiological changes in cardiomyocytes can lead to the formation of ectopic foci. Heart disease should also be considered as a source of additional ventricular beats (Emery and Kovacs 2018). Other possible causes of PVCs may include prior myocarditis, the presence of false tendons in the LV, abnormalities of adrenergic innervation, electrolyte disturbances, and training-induced sinus bradycardia (Ben Halima et al. 2018).

Clinical implications

Premature ventricular beats in young athletes are rare and are mostly benign. However, the significance of careful analysis of PVC morphology on the ECG is extremely important, as it may help identify cases requiring further cardiological evaluation (Gomez et al. 2024). If ≥2 ventricular beats appear on the ECG, further diagnostics are recommended. The risk associated with PVCs primarily depends on several arrhythmic features: the number of beats (so-called burden), QRS complex morphology, arrhythmia complexity, and response to physical exercise. The most commonly used diagnostic tools include Holter ECG monitoring, exercise testing, and echocardiography. In some cases, cardiac magnetic resonance imaging is also necessary to detect myocardial changes that may not be visible on standard echocardiography (Emery and Kovacs 2018). Ventricular arrhythmias are the most common cause of sudden death in athletes, making thorough evaluation essential when deciding on sports eligibility. Athletes with structural heart disease and PVCs are restricted to low-intensity class IA sports (Ben Halima et al. 2018). Treatment is mainly symptomatic and surveillance-based, and decisions regarding sports participation should be based on a careful risk assessment and diagnostic findings. Antiarrhythmic drugs (except for beta-blockers in some situations) and ablation have not been shown to improve survival; they are primarily used in symptomatic cases or in athletes with a high PVC burden, especially when PVC-induced cardiomyopathy is present. Competitive sports are not prohibited if structural heart disease has been excluded, but in complex arrhythmias, the decision requires individualized assessment (Zorzi et al. 2023). **3.3 Atrial fibrillation**

Prevalence

Atrial fibrillation is one of the most common cardiac arrhythmias in the general population. It increases the risk of serious complications, such as stroke, heart failure, and cognitive dysfunction. (Stergiou and Duncan 2018) Since intense physical activity can lead to structural and electrical changes in the heart, physical exertion is considered a potential risk factor for AF. Epidemiological studies have shown that the prevalence of AF in athletes may be approximately 5–10%. (Lobo et al. 2023) A meta-analysis conducted by William Newman et al. demonstrated that athletes are more than twice as likely to develop atrial fibrillation compared to a control group. Athletes under 55 years of age had a higher relative

risk of developing AF than older athletes. Mixed sports (e.g., football or rugby) were associated with a greater risk of AF than endurance sports. (Newman et al. 2021)

Mechanisms

The mechanisms responsible for the development of arrhythmias in athletes may result from cardiac adaptations to prolonged physical exertion. The most important mechanism that can lead to episodes of atrial fibrillation is myocardial fibrosis. Prolonged intense exercise can cause micro-injuries to cardiomyocytes, increased collagen production, and remodeling of the extracellular matrix. This leads to increased myocardial stiffness, atrial enlargement, and disruption of electrical impulse conduction. Myocardial fibrosis promotes the formation of reentry circuits, which are crucial for the development of atrial fibrillation. (Lobo et al. 2023) Key factors contributing to atrial fibrillation in athletes also include atrial enlargement, structural remodeling and myocardial fibrosis, increased autonomic nervous system activity, chronic inflammation, and the presence of arrhythmogenic foci within the pulmonary veins. (Newman et al. 2021) Intense physical exertion may trigger the activation of pro-inflammatory cytokines, increase oxidative stress, and influence disturbances in calcium handling within cardiomyocytes. All of the above factors can lead to cardiac rhythm disturbances. (Lobo et al.
2023)

Clinical implications

The clinical significance of atrial fibrillation in athletes is not straightforward. Despite the higher prevalence of AF among athletes, they have a lower risk of stroke and cardiovascular disease compared to sedentary individuals. Many epidemiological studies have observed a Ushaped relationship between atrial fibrillation and physical activity, indicating that both lack of physical activity and very intense training are associated with a higher risk of arrhythmia. Only moderate activity appears to be protective. (Stergiou and Duncan 2018) The initial evaluation of an athlete with atrial fibrillation focuses on determining whether AF episodes are related to physical activity. This should begin with a detailed environmental history to exclude the use of doping substances and illicit drugs. Investigations should include a physical examination, ECG, and, in selected cases, echocardiography or magnetic resonance imaging to detect structural heart diseases such as dilated cardiomyopathy, hypertrophic cardiomyopathy, or arrhythmogenic right ventricular cardiomyopathy. Assessment of thyroid hormones and electrolytes is also recommended. In younger patients, congenital ion channel disorders and the presence of accessory conduction pathways should be considered. Exercise testing may be performed to assess the cardiac response to exertion and detect ischemia. (Estes and Madias 2017) During intense physical exertion, increases in markers indicative of cardiac injury, such as cardiac troponins, CK-MB, and natriuretic peptides, can be observed. These elevations are usually transient and return to normal after exercise. Markers associated with collagen remodeling may also be noted. Increases in both groups of markers support the theory of microinjury and myocardial fibrosis. (Lobo et al. 2023) The goal of AF management in athletes is not only to reduce or eliminate episodes of atrial fibrillation but also to enable athletes to continue physical activity. (Estes and Madias 2017) Athletes with paroxysmal AF often report a significant decline in exercise tolerance. Pharmacological treatment in this population is limited. Beta-blockers are poorly tolerated due to decreased physical performance during use. Because of multiple adverse effects, drugs such as sotalol or amiodarone are rarely used. Flecainide can reduce the frequency and duration of AF episodes; however, its use carries the risk of exercise-induced arrhythmias, syncope, and

1:1 atrioventricular conduction in the case of atrial flutter. According to recommendations, it should be used in combination with a βblocker, and patients taking this drug should temporarily refrain from sports until arrhythmia resolves and for a period corresponding to two half-lives of the medication. (Stergiou and Duncan 2018) Reducing training intensity may, in some cases, decrease the frequency of AF episodes. Catheter ablation is often used in athletes with AF and is one of the preferred treatment methods in this population. Studies suggest that its efficacy is comparable to that observed in non-athletes. The procedure allows a return to full physical activity without the need for longterm antiarrhythmic medication. (Lobo et al. 2023)

3.4 The most common arrhythmias associated with sudden cardiac death.

Cardiovascular diseases account for over 90% of sudden cardiac death cases in athletes. In individuals under 35 years of age, genetic conditions predominate, such as hypertrophic cardiomyopathy, which can lead to electrical instability of the heart and life-threatening ventricular arrhythmias. Other important causes include arrhythmogenic right ventricular cardiomyopathy and congenital coronary artery anomalies, which may induce myocardial ischemia and secondary rhythm disturbances during exertion. In the absence of structural changes at autopsy, channelopathies such as long QT syndrome, Brugada syndrome, or short QT syndrome should be considered. Wolff-Parkinson-White syndrome may also be a potential cause of sudden death. (Tlili et al. 2012) Acquired causes include, among others, commotio cordis (chest trauma inducing arrhythmia), myocarditis, environmental factors, and the use of performance-enhancing substances. (Emery and Kovacs 2018)

Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

In studies conducted in the American population, ARVC accounts for approximately 4% of SCD cases, whereas in studies of the Italian population, it accounts for about 23%. (D'Silva and Papadakis 2015) The disease involves progressive dystrophy of the right ventricular myocardium, with replacement of muscle tissue by fibrofatty tissue, leading to aneurysms and right ventricular weakening. In approximately 50% of cases, it is hereditary. The responsible genes encode proteins of cell junctions (plakoglobin, plakophilin, desmoglein, desmocollin, desmoplakin), resulting in remodeling of intercalated discs and an increased risk of arrhythmias. (Thiene et al. 2007) For diagnosis, ECG is essential, with approximately 85% of patients with ARVC showing T-wave inversion in leads V1–V3. A characteristic, though less frequent, electrocardiographic sign is epsilon waves, visible after the QRS complex and before the Twave, observed in about 30% of patients. In athletes with so-called "athlete's heart," similar changes may appear; however, in their case, they usually resolve during physical exertion, whereas in ARVC patients, they persist despite activity. (Walker et al. 2010), (Basu and Malhotra 2018) Clinically, palpitations, syncope, and ventricular tachycardias predominate. Sudden cardiac death may be the first manifestation. Progressive loss of right ventricular myocardium leads to heart failure. (Thiene et al. 2007) To reduce the risk of sudden cardiac death, an implantable cardioverter-defibrillator (ICD) is used, especially in patients with a positive family history. Beta-blockers may reduce the frequency of arrhythmias. Catheter ablation can achieve initial control of ventricular tachycardia in approximately 60–90% of patients; however, arrhythmias often recur as new arrhythmogenic foci develop over time. (Walker et al. 2010)

Long QT Syndrome

Long QT syndrome (LQTS) occurs in approximately 0.4% of athletes. It is characterized by a prolonged QT interval on ECG and susceptibility to life-threatening arrhythmias, primarily torsades de pointes (TdP), which can lead to syncope or sudden cardiac death, triggered by physical exertion, stress, or strong emotions. (Gomez et al. 2016) LQTS may be either acquired or genetic, resulting from various gene mutations. Three types of LQTS are summarized in the table below. (Walker et al. 2010) Patients with LQTS type 1 (LQTS1) exhibit the highest risk of cardiovascular events during physical activity, particularly swimming or diving. (Volpato et al. 2021)

Table 1. Types of Long QT Syndrome, Genetic Background, Arrhythmia Triggers, and Treatment. (Volpato et al. 2021)

LQTS Type	Gene	Chromosome	Electrophysiological Mechanism	Arrhythmia Trigger	Treatment
LQTS 1	KCNQ1	11	Impaired slow potassium current (IKs)	Physical exertion	Beta-blockers; high-risk patients – ICD
LQTS 2	KCNH2	7	Impaired rapid potassium current (IKr)	Stress, emotions, autonomic stimulation	Beta-blockers; high-risk patients – ICD
LQTS 3	SCN5A	3	Increased sodium channel activity	Rest or bradycardia	Sodium channel blockers; highrisk patients – ICD

The basis for diagnosis is ECG, with diagnostic values of the corrected QT interval (QTc) according to Bazett's formula set at ≥470 ms for male athletes and ≥480 ms for female athletes. (Walker et al. 2010) Congenital LQTS should be distinguished from its acquired form. Congenital LQTS is diagnosed when QTc in a standard 12-lead ECG is at least 480 ms, a pathogenic LQTS gene mutation is detected, or the LQTS diagnostic score exceeds 3. (Volpato et al. 2021) Genetic testing can also be performed, particularly in asymptomatic athletes with prolonged QTc or a family history of sudden cardiac death. (Walker et al. 2010) Pharmacological treatment includes beta-blockers (propranolol, nadolol), which are the mainstay therapy for all LQTS types. Additionally, in LQTS3, sodium channel blockers (mexiletine, flecainide, ranolazine) are used. QT-prolonging drugs, electrolyte disturbances, and dehydration should be avoided. Implantable cardioverter-defibrillator (ICD) placement

may be considered. (Gomez et al. 2016) Individuals with QTc above 500 ms or genetically confirmed LQTS with QTc exceeding 470 ms in men or 480 ms in women should avoid intense forms of sports, both recreational and competitive, even while on beta-blocker therapy. Patients with a history of cardiac arrest or arrhythmia-induced syncope should not participate in professional sports, even if an ICD is implanted. (Volpato et al. 2021)

Brugada Syndrome

Brugada syndrome is an autosomal dominant inherited disorder caused by a mutation in the SCN5A gene on chromosome 3, resulting in loss of function of sodium channels. (Walker et al. 2010) The main mechanism producing symptoms in athletes is high vagal tone, which slows the heart rate and increases the visibility of Brugada features on the ECG. The most high-risk period is the post-exercise phase, when sympathetic activity decreases and parasympathetic activity increases. This can lead to syncope and post-exertional symptoms. [32] The primary diagnostic tool is the ECG, which may show type 1 ST-segment elevation in the right precordial leads, followed by a negative T-wave. In some cases, ECG changes are not apparent, and a sodium channel blocker challenge is required. (Walker et al. 2010) (Petek et al. 2023) Physical activity does not worsen prognosis in patients with Brugada syndrome; it only accelerates diagnosis and does not increase the risk of sudden cardiac death. [32] In asymptomatic individuals with Brugada syndrome—including mutation carriers and athletes with only exercise-induced ECG changes—sports that significantly raise body temperature (e.g., intense competitions in heat or high humidity above 39°C) are not recommended. Athletes with an implanted cardioverter-defibrillator (ICD) who have previously experienced life-threatening arrhythmias or cardiac arrest may return to physical activity if they remain symptom-free for at least 3 months. (Volpato et al. 2021) ICD implantation can prevent sudden cardiac death. (Walker et al. 2010)

Wolff-Parkinson-White (WPW) Syndrome

In individuals with WPW syndrome, an accessory conduction pathway exists between the atria and ventricles, bypassing the AV node. This leads to premature ventricular activation and bidirectional impulse conduction. (Walker et al. 2010) Such a situation can result in arrhythmias, including supraventricular tachycardia (SVT), atrial fibrillation, or ventricular fibrillation. Physical exertion may enhance accessory conduction. While it does not always cause arrhythmias, a significant proportion of sudden cardiac deaths in WPW syndrome occur during exercise or emotional stress. (Volpato et al. 2021) WPW syndrome is primarily diagnosed based on ECG. A characteristic feature is the delta wave. An essential aspect of evaluation is risk assessment in athletes to identify those at risk for life-threatening arrhythmias and sudden cardiac death. Non-invasive investigations include 24–48-hour Holter ECG monitoring, exercise testing, and echocardiography. In cases of insufficient non-invasive testing or suspected high risk, electrophysiological studies may be performed. (Rao et al. 2014) The first-line treatment, especially for individuals at high risk of sudden cardiac death, is catheter ablation (radiofrequency ablation or cryoablation). If the athlete declines ablation or the procedure carries high risk, the possibility of returning to physical activity should be individually assessed, and class Ic antiarrhythmic drugs may be used.

After ablation, resumption of sports can be considered after approximately 1–3 months. (Volpato et al. 2021)

4. Discussion

The obtained results are consistent with the current state of knowledge, which indicates that cardiovascular adaptations in athletes constitute a continuum from physiological to potentially pathological changes. Similar conclusions have been presented in systematic reviews, which emphasize the difficulty in differentiating between "athlete's heart" and cardiomyopathies, particularly in the context of left ventricular hypertrophy and electrocardiographic changes (Walker et al. 2010). With regard to sinus bradycardia and atrioventricular blocks, the results of this study are consistent with previous findings demonstrating their high prevalence among endurance athletes. Traditionally, these changes have been explained by increased vagal tone; however, more recent reports also indicate a significant role of electrophysiological remodeling of the sinus node, including reduced expression of HCN4 channels (Flannery et al. 2021). Moreover, observations of persistent bradycardia after the end of an athletic career suggest a partially irreversible nature of these changes, which is consistent with the results of longitudinal studies (Finocchiaro et al. 2026). With respect to premature ventricular contractions, the obtained results confirm observations from other authors that most PVCs in young athletes are benign and idiopathic. At the same time, the literature emphasizes that certain arrhythmic features—such as high burden, atypical morphology, or lack of suppression during exercise— may indicate the presence of structural heart disease (Ben Halima et al. 2019). In the case of atrial fibrillation, the results of this review are consistent with meta-analyses indicating an increased risk of AF in athletes, particularly in endurance disciplines. Studies have demonstrated more than a twofold increase in the risk of AF in this population compared to physically inactive individuals (Newman et al. 2021). The mechanisms underlying this phenomenon, such as atrial enlargement, myocardial fibrosis, and autonomic disturbances, are widely described in the literature and confirm the multifactorial nature of this arrhythmia (D'Ambrosio et al. 2024). Additionally, the observed U-shaped relationship between the level of physical activity and the risk of AF suggests that both a lack of activity and extreme exercise may increase the risk of arrhythmias (Stergiou and Duncan 2018). In the context of sudden cardiac death, the obtained results are consistent with epidemiological data indicating the predominant role of genetic diseases in young athletes, such as cardiomyopathies and channelopathies (Tlili et al. 2012). In the absence of structural heart changes, particular importance is attributed to conditions such as long QT syndrome, Brugada syndrome, and Wolff–Parkinson–White syndrome, which may lead to life-threatening ventricular arrhythmias (Walker et al. 2010). In contrast, in older athletes, the literature clearly indicates a greater role of acquired diseases, especially atherosclerosis.
The limitations of this study are consistent with those indicated in other reviews. The heterogeneity of the studied populations, differences in arrhythmia definitions, and the predominance of observational studies make it difficult to draw unambiguous conclusions. Therefore, further prospective studies are necessary to better determine cause-and-effect relationships.

5. Conclusion

Intense physical exercise significantly affects the human body. It is beyond doubt that both endurance and strength sports lead to adaptive changes in the cardiovascular system, referred to in the literature as the "athlete's heart." These changes can be physiological, but

in some cases they may also lead to the development of pathology. This paper presents the most common arrhythmias that may result from changes occurring in the cardiovascular system during intense physical exertion. Although most rhythm disturbances, such as sinus bradycardia or atrioventricular block, are benign and reflect normal adaptation to exercise, there are also arrhythmias, such as atrial fibrillation and ventricular arrhythmias, which require careful clinical evaluation. A key challenge in athlete care is differentiating physiological changes from pathological ones and conducting appropriate diagnostics, enabling early detection of lifethreatening complications. Such diagnostics include, among others, electrocardiography (ECG), echocardiography, exercise testing, and cardiac magnetic resonance imaging. (Khan et al. 2016) Early identification of athletes at increased risk of serious arrhythmias or sudden cardiac death allows for the implementation of effective preventive and therapeutic strategies. Individualized clinical management and continuous monitoring are crucial to ensure patient safety while allowing continued physical activity.

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References

1. Blake, H. T., Buckley, J. D., Stenner, B. J., O'Connor, E. J., Burgess, S. A., & Crozier, A. J. (2022). Sport Participation and Subjective Outcomes of Health in Middle-Aged Men: A Scoping Review. American journal of men's health, 16(2), 15579883221084493. https://doi.org/10.1177/15579883221084493
2. Deng, Y., & Fan, A. (2022). Trends in sports participation in adolescents: Data from a large-scale sample in the US adolescents. Frontiers in public health, 10, 960098. https://doi.org/10.3389/fpubh.2022.960098
3. Maxwell, J. D., & Oxborough, D. (2025). The athletes heart-from acute stimulus to chronic adaptation. British medical bulletin, 153(1), ldae021. https://doi.org/10.1093/bmb/ldae021

4. Yilmaz, D. C., Buyukakilli, B., Gurgul, S., & Rencuzogullari, I. (2013). Adaptation of heart to training: a comparative study using echocardiography & impedance cardiography in male & female athletes. The Indian journal of medical research, 137(6), 1111–1120.

5. Gomez, S. E., Perez, M. V., Wheeler, M. T., Hadley, D., Hwang, C. E., Kussman, A., Kim, D. S., & Froelicher, V. (2024). Classification of Premature Ventricular Contractions in Athletes During Routine Preparticipation Exams. Circulation. Arrhythmia and electrophysiology, 17(9), e012835. https://doi.org/10.1161/CIRCEP.124.012835

6. Basu, J., & Malhotra, A. (2018). Interpreting the Athlete's ECG: Current State and Future Perspectives. Current treatment options in cardiovascular medicine, 20(12), 104. https://doi.org/10.1007/s11936-018-0693-0

7. Petek, B. J., Drezner, J. A., & Churchill, T. W. (2023). The International Criteria for Electrocardiogram Interpretation in Athletes: Common Pitfalls and Future Directions. Cardiology clinics, 41(1), 35–49. https://doi.org/10.1016/j.ccl.2022.08.003

8. Emery, M. S., & Kovacs, R. J. (2018). Sudden Cardiac Death in Athletes. JACC. Heart failure, 6(1), 30–40. https://doi.org/10.1016/j.jchf.2017.07.014

9. Corrado, D., Drezner, J. A., D'Ascenzi, F., & Zorzi, A. (2020). How to evaluate premature ventricular beats in the athlete: critical review and proposal of a diagnostic algorithm. British journal of sports medicine, 54(19), 1142–1148. https://doi.org/10.1136/bjsports-2018-100529

10. Khan, A. A., Safi, L., & Wood, M. (2016). Cardiac Imaging In Athletes. Methodist DeBakey cardiovascular journal, 12(2), 86–92. https://doi.org/10.14797/mdcj-12-2-86

11. Ben Halima, A., Ben Halima, M., BelHadj, Z., Boukhris, M., Ayachi, S., Ben Salah, H., Addala, H., Chaouachi, A., & Kobaa, D. (2018). Assessment of premature ventricular beats in athletes. La Tunisie medicale, 96(4), 155–159.

12. Moore R. L. (1998). Cellular adaptations of the heart muscle to exercise training. Annals of medicine, 30 Suppl 1(Suppl 1), 46–53. https://doi.org/10.1080/13652060.1998.12527715

13. Finocchiaro, G., Zorzi, A., Abela, M., Baggish, A., Castelletti, S., Cavarretta, E., Claessen, G., Corrado, D., Sanz de la Garza, M., Gati, S., Maestrini, V., Malhotra, A., Niebauer, J., Niederseer, D., Papadakis, M., Pelliccia, A., Sharma, S., & D'Ascenzi, F. (2026). Abnormal electrocardiogram findings in athletes. European heart journal, 47(2), 152–169. https://doi.org/10.1093/eurheartj/ehaf646

14. Malhotra, V. K., Singh, N., Bishnoi, R. S., Chadha, D. S., Bhardwaj, P., Madan, H., Dutta, R., Ghosh, A. K., Sengupta, S., & Perumal, P. (2015). The prevalence of abnormal ECG in trained sportsmen. Medical journal, Armed Forces India, 71(4), 324–329. https://doi.org/10.1016/j.mjafi.2015.06.010

15. La Gerche, A., Wasfy, M. M., Brosnan, M. J., Claessen, G., Fatkin, D., Heidbuchel, H., Baggish, A. L., & Kovacic, J. C. (2022). The Athlete's Heart-Challenges and Controversies: JACC Focus Seminar 4/4. Journal of the American College of Cardiology, 80(14), 1346–1362. https://doi.org/10.1016/j.jacc.2022.07.014

16. Abulí, M., de la Garza, M. S., & Sitges, M. (2020). Differentiating Athlete's Heart from Left Ventricle Cardiomyopathies. Journal of cardiovascular translational research, 13(3), 265–273.

https://doi.org/10.1007/s12265-020-10021-8
17. Zholshybek, N., Khamitova, Z., Toktarbay, B., Jumadilova, D., Khissamutdinov, N., Dautov, T., Rakhmanov, Y., Bekbossynova, M., Gaipov, A., & Salustri, A. (2023). Cardiac imaging in athlete's heart: current status and future prospects. Cardiovascular ultrasound, 21(1), 21. https://doi.org/10.1186/s12947-023-00319-3

18. Al-Othman, S., Boyett, M. R., Morris, G. M., Malhotra, A., Mesirca, P., Mangoni, M. E., & D'Souza, A. (2024). Symptomatic bradyarrhythmias in the athlete-Underlying mechanisms and treatments. Heart rhythm, 21(8), 1415–1427. https://doi.org/10.1016/j.hrthm.2024.02.050

19. Grandys, M., Woźniak, M., & Żołądź, J. A. (2025). The slowing pulse: a history of research on training-induced resting bradycardia. Folia medica Cracoviensia, 65(4), 43–52. https://doi.org/10.24425/fmc.2025.156697

20. Lazzerini, P. E., Bertolozzi, I., Cartocci, A., Ginjupalli, V. K. M., Teneggi, P. A., Pica, D., Merico, G., Bogazzi, I., Salvini, V., Accioli, R., Salvadori, F., Marzotti, T., Cevenini, G., Capecchi, M., Cantara, S., Cantore, A., Infantino, M., Bisogno, S., Finizola, F., D'ascenzi, F., … Boutjdir, M. (2024). Advanced Atrioventricular Block in Athletes: Prevalence and Role of Anti-Ro/Sjögren Syndrome-Related Antigen A Antibodies. Journal of the American Heart Association, 13(12), e034893. https://doi.org/10.1161/JAHA.124.034893

21. Zorzi, A., D'Ascenzi, F., Andreini, D., Castelletti, S., Casella, M., Cavarretta, E., Cipriani, A., Compagnucci, P., Delise, P., Dello Russo, A., Graziano, F., Palamà, Z., Pelliccia, A., Sarto, P., Corrado, D., & Sciarra, L. (2023). Interpretation and management of premature ventricular beats in athletes: An expert opinion document of the Italian Society of Sports Cardiology (SICSPORT). International journal of cardiology, 391, 131220. https://doi.org/10.1016/j.ijcard.2023.131220
22. Lobo, H. M., Naves, Í. G., Marçal, S. B., Canzi, C. C., Rodrigues, A. B. S., & Menezes, A. S., Jr (2023). Atrial Fibrillation in Endurance Training Athletes: Scoping Review. Reviews in cardiovascular medicine, 24(6), 155. https://doi.org/10.31083/j.rcm2406155

23. Newman, W., Parry-Williams, G., Wiles, J., Edwards, J., Hulbert, S., Kipourou, K., Papadakis, M., Sharma, R., & O'Driscoll, J. (2021). Risk of atrial fibrillation in athletes: a systematic review and meta-analysis. British journal of sports medicine, 55(21), 1233–1238. https://doi.org/10.1136/bjsports-2021-103994

24. Stergiou, D., & Duncan, E. (2018). Atrial Fibrillation (AF) in Endurance Athletes: a Complicated Affair. Current treatment options in cardiovascular medicine, 20(12), 98. https://doi.org/10.1007/s11936-018-0697-9

25. Estes, N. A. M., 3rd, & Madias, C. (2017). Atrial Fibrillation in Athletes: A Lesson in the Virtue of Moderation. JACC. Clinical electrophysiology, 3(9), 921–928. https://doi.org/10.1016/j.jacep.2017.03.019
26. Tlili, R., Ben Ameur, Y., Sdiri, W., Ben Romdhane, R., Bachraoui, K., & Boujnah, M. R. (2012). La mort subite d'origine rythmique chez le sportif: revue de la littérature [Sudden cardiac death of rhythmic origin in athletes: literature review]. La Tunisie medicale, 90(5), 345–350.

27. Thiene, G., Corrado, D., & Basso, C. (2007). Arrhythmogenic right ventricular cardiomyopathy/dysplasia. Orphanet journal of rare diseases, 2, 45. https://doi.org/10.1186/1750-1172-2-45

28. D'Silva, A., & Papadakis, M. (2015). Sudden Cardiac Death in Athletes. European cardiology, 10(1), 48–53. https://doi.org/10.15420/ecr.2015.10.01.48

29. Walker, J., Calkins, H., & Nazarian, S. (2010). Evaluation of cardiac arrhythmia among athletes. The American journal of medicine, 123(12), 1075–1081. https://doi.org/10.1016/j.amjmed.2010.05.008

30. Volpato, G., Falanga, U., Cipolletta, L., Conti, M. A., Grifoni, G., Ciliberti, G., Urbinati, A., Barbarossa, A., Stronati, G., Fogante, M., Bergonti, M., Catto, V., Guerra, F., Giovagnoni, A., Dello Russo, A., Casella, M., & Compagnucci, P. (2021). Sports Activity and Arrhythmic Risk in Cardiomyopathies and Channelopathies: A Critical Review of European Guidelines on Sports Cardiology in Patients with Cardiovascular Diseases. Medicina (Kaunas, Lithuania), 57(4), 308. https://doi.org/10.3390/medicina57040308

31. Gomez, A. T., Prutkin, J. M., & Rao, A. L. (2016). Evaluation and Management of Athletes With Long QT Syndrome. Sports health, 8(6), 527–535. https://doi.org/10.1177/1941738116660294

32. Fernandez Gil, M. J., Carrillo Mora, L. M., Fernandez Vazquez, D., Melgarejo, F., Santos Mateo, J. J., Muñoz Esparza, C., Rodriguez Serrano, A. I., Navarro-Penalver, M., Sanchez Muñoz, J. J., Gimeno-Blanes, F. J., Sabater-Molina, M., & Gimeno, J. R. (2025). Impact of physical activity on presentation and prognosis of Brugada syndrome. Open heart, 12(2), e003119. https://doi.org/10.1136/openhrt-2024-003119

33. Rao, A. L., Salerno, J. C., Asif, I. M., & Drezner, J. A. (2014). Evaluation and management of wolff-Parkinson-white in athletes. Sports health, 6(4), 326–332. https://doi.org/10.1177/1941738113509059

34. Walker, J., Calkins, H., & Nazarian, S. (2010). Evaluation of cardiac arrhythmia among athletes. The American journal of medicine, 123(12), 1075–1081. https://doi.org/10.1016/j.amjmed.2010.05.008

35. Flannery, D., Sully, F., Janssens, K., Kalman, J., La Gerche , A. (2021) Mechanisms of Bradycardia in Athletes: A Case–Control, Blinded, Randomised, Crossover Study. Heart, Lung and Circulation , 30(3), 1443-9506. https://doi.org/10.1016/j.hlc.2021.06.156

36. Ben Halima, A., Kobaa, D., Ben Halima, M., Ayachi, S., Belkhiria, M., & Addala, H. (2019). Assessment of premature ventricular beats in athletes. Annales de cardiologie et d'angeiologie, 68(3), 175–180. https://doi.org/10.1016/j.ancard.2018.10.013

37. D'Ambrosio, P., Claessen, G., Kistler, P. M., Heidbuchel, H., Kalman, J. M., & La Gerche, A. (2024). Ventricular arrhythmias in association with athletic cardiac remodelling. Europace : European pacing, arrhythmias, and cardiac electrophysiology : journal of the working groups on cardiac pacing, arrhythmias, and cardiac cellular electrophysiology of the European Society of Cardiology, 26(12), euae279. https://doi.org/10.1093/europace/euae279