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Sinus Bradycardia in Endurance Athletes: From Physiological Adaptation to Potential Pathology

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ABSTRACT

Sinus bradycardia is a common electrocardiographic finding in trained athletes and a hallmark feature of the athlete's heart. Reduced resting heart rate, particularly in endurance athletes, reflects physiological adaptation to long-term intensive training and is usually asymptomatic. In highly trained individuals, heart rate values may fall well below standard clinical thresholds while maintaining normal cardiovascular function.

Exercise-induced bradycardia has traditionally been attributed to increased parasympathetic activity and reduced sympathetic tone. However, recent evidence indicates that intrinsic remodeling of the sinoatrial node also contributes to this phenomenon. Molecular and electrophysiological adaptations, including alterations in ion channel expression and pacemaker cell function, play an important role in the reduction of intrinsic heart rate.

Although generally considered benign, sinus bradycardia in athletes has raised concerns regarding its potential long-term clinical implications. Emerging data suggest that prolonged endurance training may be associated with an increased risk of clinically significant bradyarrhythmias later in life, including sinus node dysfunction and atrioventricular conduction abnormalities.

Differentiating physiological adaptation from early pathological remodeling remains a key challenge in sports cardiology. Accurate evaluation requires integration of clinical assessment, electrocardiography, ambulatory monitoring, and cardiac imaging.

This review summarizes current knowledge on the mechanisms, clinical significance, and diagnostic evaluation of sinus bradycardia in athletes, with particular emphasis on the balance between beneficial adaptation and potential adverse remodeling associated with long-term endurance training.

Keywords: sinus bradycardia; athletes; sinoatrial node; cardiac remodeling; autonomic nervous system; bradyarrhythmias; sports cardiology

Introduction

Regular physical activity induces numerous beneficial adaptations within the cardiovascular system. Among the most characteristic manifestations of long-term endurance training is a reduction in resting heart rate, commonly referred to as sinus bradycardia.

Sinus bradycardia is defined as a resting heart rate below 60 beats per minute originating from the sinoatrial node and represents one of the most frequently observed electrocardiographic findings in trained athletes [1,2,3]. While such findings may raise concern in the general population due to potential conduction system disease or metabolic disturbances, sinus

bradycardia is commonly observed in trained athletes and is usually considered a physiological adaptation to chronic exercise training [4,5].

This phenomenon represents one of the key components of the "athlete's heart," a term describing the structural, functional, and electrophysiological adaptations of the cardiovascular system to long-term intensive physical activity [6,7].These adaptations include increased ventricular chamber size, enhanced stroke volume, improved diastolic filling, and characteristic electrocardiographic changes such as sinus bradycardia and mild atrioventricular conduction delays [4,8].

Traditionally, exercise-induced bradycardia has been attributed primarily to increased parasympathetic activity and reduced sympathetic tone, resulting in enhanced vagal influence on the sinoatrial node and slower pacemaker activity [6]. However, recent studies suggest that intrinsic remodeling of the sinoatrial node also contributes to this phenomenon. Molecular and electrophysiological investigations have demonstrated training-induced alterations in ion channel expression, particularly involving the hyperpolarization-activated cyclic nucleotide–gated channel 4 (HCN4), which plays a critical role in pacemaker current generation [9].

Although sinus bradycardia in athletes is generally benign, emerging evidence indicates that long-term endurance training may be associated with structural and electrophysiological changes within the cardiac conduction system. In some cases, these changes may predispose individuals to clinically significant bradyarrhythmias later in life, including sinus node dysfunction and atrioventricular conduction disturbances [10,11].

The aim of this review is to summarize current knowledge regarding sinus bradycardia in athletes, focusing on its physiological mechanisms, clinical significance, and diagnostic evaluation. Particular emphasis is placed on the distinction between physiological adaptation and potential pathological remodeling of the cardiac conduction system.

2. Epidemiology of Sinus Bradycardia in Athletes

Sinus bradycardia represents one of the most frequently observed electrocardiographic findings among trained athletes. Numerous studies examining cardiovascular adaptations to exercise have demonstrated that resting heart rates below 60 beats per minute occur in a substantial proportion of individuals engaged in competitive sports, particularly endurance disciplines such as long-distance running, cycling, rowing, and cross-country skiing [12,13,14].

The prevalence of sinus bradycardia varies depending on several factors, including the type of sport, intensity of training, duration of athletic career, and individual physiological characteristics [15,16]. Endurance athletes typically demonstrate more pronounced reductions

in resting heart rate compared with athletes participating in strength or power disciplines. This difference is largely attributed to the higher volume of aerobic training and sustained cardiovascular demand characteristic of endurance sports [17].

Elite endurance athletes often exhibit resting heart rates between 30 and 50 beats per minute. Such values may initially appear concerning in clinical settings but are generally considered normal in asymptomatic athletes with no evidence of structural heart disease [4].

Longitudinal studies suggest that the degree of bradycardia correlates strongly with training volume and duration of athletic exposure [18]. Athletes who have undergone many years of high-intensity endurance training typically demonstrate more pronounced reductions in resting heart rate than individuals with shorter training histories. This observation supports the concept that exercise-induced bradycardia represents a progressive physiological adaptation to chronic cardiovascular conditioning.

Age also plays an important role in the prevalence of bradycardia among athletes. Younger athletes who begin intensive training during adolescence may develop substantial cardiovascular adaptations early in life. However, the persistence of these adaptations into later adulthood remains a subject of ongoing investigation.

Sex differences in resting heart rate have also been reported. Male athletes generally exhibit slightly lower resting heart rates compared with female athletes, although this difference may be influenced by variations in body size, training intensity, and hormonal factors.

Despite the high prevalence of sinus bradycardia in athletes, the vast majority of cases remain asymptomatic and clinically insignificant. However, rare cases of pathological bradyarrhythmias have been described in athletes, emphasizing the importance of careful evaluation when bradycardia is accompanied by symptoms or abnormal electrocardiographic findings [19].

In addition to sinus bradycardia, athletes frequently exhibit other conduction system adaptations, including sinus arrhythmia, first-degree atrioventricular block, and occasional second-degree atrioventricular block during rest or sleep. These findings are typically reversible with exercise or sympathetic stimulation and are therefore considered benign manifestations of increased parasympathetic tone [20].

Understanding the epidemiology of sinus bradycardia in athletes is essential for distinguishing physiological adaptation from pathological conditions that may require further evaluation.

3. Physiological Mechanisms of Exercise-Induced Bradycardia

Exercise-induced bradycardia is a complex phenomenon resulting from multiple physiological mechanisms involving autonomic nervous system adaptation and intrinsic remodeling of pacemaker tissue [21,22]. These mechanisms include adaptations of the autonomic nervous system as well as intrinsic structural and electrophysiological remodeling of the sinoatrial node.

3.1 Autonomic Nervous System Adaptation

The autonomic nervous system plays a central role in regulating heart rate through the balance between sympathetic and parasympathetic influences on the sinoatrial node.

Endurance training leads to increased parasympathetic activity and reduced sympathetic tone during rest. Enhanced vagal modulation slows spontaneous depolarization of pacemaker cells and reduces the rate of impulse generation in the sinoatrial node [23].

Several studies have demonstrated increased heart rate variability in trained athletes compared with sedentary individuals, reflecting greater parasympathetic dominance and improved autonomic regulation [24]. This autonomic shift contributes significantly to the reduction in resting heart rate observed in athletes.

Pharmacological studies have further supported the role of autonomic modulation in training-induced bradycardia. Administration of muscarinic receptor antagonists, which block parasympathetic influence on the heart, partially reverses the reduction in resting heart rate observed in trained individuals.

However, even after complete autonomic blockade, athletes often maintain lower intrinsic heart rates than sedentary controls. This observation suggests that autonomic modulation alone cannot fully explain exercise-induced bradycardia and that intrinsic changes within the sinoatrial node must also contribute to this adaptation [25].

3.2 Intrinsic Sinoatrial Node Remodeling

Recent advances in molecular cardiology have provided new insights into the mechanisms underlying exercise-induced bradycardia. Experimental studies have demonstrated that endurance training leads to intrinsic remodeling of the sinoatrial node, resulting in alterations in pacemaker cell electrophysiology.

One of the key mechanisms involves changes in the expression of ion channels responsible for pacemaker activity. The hyperpolarization-activated cyclic nucleotide–gated channel 4 (HCN4) plays a crucial role in generating the pacemaker current (If), which drives spontaneous depolarization in sinoatrial node cells.

Studies have shown that endurance training reduces the expression of HCN4 channels in pacemaker cells, leading to decreased pacemaker current and slower intrinsic heart rate [26]. This molecular remodeling appears to be a major contributor to exercise-induced bradycardia.

In addition to ion channel alterations, microRNA-mediated regulation has been implicated in the remodeling process. Certain microRNAs regulate gene expression related to pacemaker cell function and may contribute to the electrophysiological changes observed in trained athletes.

Structural remodeling of the sinoatrial node may also occur as a result of chronic endurance training, potentially involving alterations in extracellular matrix composition and pacemaker cell architecture [30]. Animal studies have demonstrated changes in pacemaker cell morphology and extracellular matrix composition within the sinoatrial node following prolonged exercise training.

These findings support the concept that exercise-induced bradycardia is not solely a functional adaptation mediated by the autonomic nervous system but also involves intrinsic structural and molecular changes within the cardiac conduction system.

4. Structural and Molecular Remodeling of the Sinoatrial Node

Although sinus bradycardia in athletes was historically attributed primarily to autonomic nervous system adaptation, increasing evidence indicates that intrinsic remodeling of the sinoatrial node plays a major role in the development of exercise-induced reductions in heart rate. The sinoatrial node, located in the right atrium near the junction with the superior vena cava, functions as the primary pacemaker of the heart and is responsible for initiating electrical impulses that determine heart rhythm.

Endurance training induces a series of electrophysiological, molecular, and structural changes within pacemaker tissue that collectively contribute to reduced intrinsic heart rate. These adaptations involve modifications in ion channel expression, alterations in cellular electrophysiology, and changes in gene regulation mechanisms [9,27].

One of the most extensively studied molecular mechanisms underlying exercise-induced bradycardia involves changes in the hyperpolarization-activated cyclic nucleotide–gated channel family, particularly HCN4. The HCN4 channel mediates the so-called “funny current” (If), which plays a central role in spontaneous diastolic depolarization in pacemaker cells. This current contributes to the gradual depolarization that ultimately triggers action potential generation within the sinoatrial node.

Experimental studies have demonstrated that endurance training leads to downregulation of HCN4 channel expression within pacemaker cells, resulting in a reduction of the pacemaker

current and a slower rate of spontaneous depolarization [9]. As a consequence, the intrinsic firing rate of the sinoatrial node decreases, producing the characteristic resting bradycardia observed in trained athletes.

Importantly, this reduction in intrinsic pacemaker activity persists even when autonomic influences are pharmacologically blocked, suggesting that structural remodeling of the sinoatrial node contributes independently to training-induced bradycardia.

Another important mechanism involves the regulation of gene expression through microRNAs. MicroRNAs are small noncoding RNA molecules that regulate gene expression by inhibiting translation or promoting degradation of messenger RNA. Several studies have identified specific microRNAs that influence pacemaker cell function and ion channel expression.

For example, microRNA-423-5p has been shown to regulate the expression of HCN4 channels, thereby influencing pacemaker activity within the sinoatrial node [9]. Alterations in microRNA expression induced by endurance training may therefore contribute to the molecular remodeling of pacemaker cells.

In addition to molecular changes, structural remodeling of the sinoatrial node may also occur. Chronic endurance training may lead to modifications in extracellular matrix composition and cellular architecture within the pacemaker region. Although these structural changes are typically mild, they may influence electrical conduction and pacemaker function.

Some investigators have proposed that repeated exposure to prolonged exercise may lead to gradual fibrotic changes within the conduction system, particularly in older athletes or individuals with long athletic careers. Such changes may potentially increase susceptibility to clinically significant bradyarrhythmias later in life.

However, it is important to note that the majority of athletes with sinus bradycardia do not develop clinically relevant conduction abnormalities. In most cases, the remodeling observed in the sinoatrial node remains within the physiological range and contributes to improved cardiovascular efficiency.

Nevertheless, the recognition that intrinsic remodeling plays a role in exercise-induced bradycardia has significantly advanced our understanding of the electrophysiological adaptations associated with long-term endurance training.

5. Sinus Bradycardia and the Athlete's Heart

Sinus bradycardia is a key component of the broader physiological phenomenon known as the athlete's heart. The term athlete's heart refers to a constellation of structural and functional cardiovascular adaptations resulting from prolonged and intensive physical training.

These adaptations enable the cardiovascular system to deliver increased oxygen and nutrient supply to skeletal muscles during exercise while maintaining efficient cardiac function during rest. Structural changes associated with the athlete's heart include increased ventricular chamber dimensions, mild ventricular wall thickening, and enhanced myocardial compliance [4,8,31].

One of the most important functional adaptations observed in athletes is an increase in stroke volume. Stroke volume represents the amount of blood ejected by the left ventricle during each cardiac cycle. In highly trained athletes, stroke volume may increase substantially due to enhanced ventricular filling and improved myocardial contractility.

As stroke volume increases, the heart can maintain adequate cardiac output despite a reduction in resting heart rate. This relationship between stroke volume and heart rate explains why sinus bradycardia is generally well tolerated in athletes.

From a physiological perspective, the reduction in heart rate allows for prolonged diastolic filling time, which improves ventricular preload and enhances myocardial perfusion. These changes contribute to the remarkable efficiency of the cardiovascular system observed in endurance athletes.

Electrocardiographic manifestations of the athlete's heart are well documented and include several characteristic findings. Sinus bradycardia is the most common ECG abnormality observed in trained athletes. In addition, sinus arrhythmia and first-degree atrioventricular block are frequently observed due to increased parasympathetic tone [20].

Other electrocardiographic findings associated with athletic training may include:

- incomplete right bundle branch block
- early repolarization patterns
- increased QRS voltage reflecting physiological ventricular hypertrophy
- junctional escape rhythms during rest or sleep

These findings are generally considered benign and represent physiological adaptations rather than pathological abnormalities.

However, interpretation of electrocardiograms in athletes requires careful evaluation because certain physiological patterns may resemble those seen in cardiomyopathies or inherited channelopathies. For example, increased QRS voltage associated with ventricular hypertrophy may mimic hypertrophic cardiomyopathy, while early repolarization patterns may resemble ST-segment abnormalities seen in certain cardiac disorders.

To address these challenges, international consensus criteria have been developed to guide the interpretation of ECG findings in athletes [27]. These criteria help differentiate normal training-related changes from abnormalities suggestive of underlying cardiac pathology.

According to these guidelines, isolated sinus bradycardia in asymptomatic athletes is considered a normal physiological finding and does not require further evaluation. However, additional investigation may be warranted if bradycardia is accompanied by symptoms or other abnormal ECG findings.

6. Clinical Spectrum of Bradyarrhythmias in Athletes

Although sinus bradycardia in athletes is typically benign, the spectrum of bradyarrhythmias observed in trained individuals ranges from physiological conduction changes to clinically significant disorders of the cardiac conduction system.

Physiological bradyarrhythmias are common in endurance athletes and are usually associated with increased parasympathetic tone. These rhythm disturbances are generally transient and occur predominantly during rest or sleep. Typical examples include sinus bradycardia, sinus arrhythmia, and first-degree atrioventricular block, all of which are considered normal variants in asymptomatic athletes [20].

In contrast, pathological bradyarrhythmias involve structural or functional abnormalities of the sinoatrial node or atrioventricular conduction system. Sinus node dysfunction represents one of the most important pathological conditions and may manifest as persistent bradycardia, sinus pauses, or chronotropic incompetence. Chronotropic incompetence refers to the inability of the heart to appropriately increase its rate during exercise and may result in reduced exercise capacity and fatigue.

Higher-degree atrioventricular block may also occur, although it is considerably less common in athletes. While first-degree atrioventricular block is frequently observed and usually benign, advanced atrioventricular conduction abnormalities may indicate underlying pathology.

Recent epidemiological studies suggest that former endurance athletes may exhibit an increased incidence of clinically significant bradyarrhythmias later in life, including sinus node dysfunction and atrioventricular block requiring pacemaker implantation [10,11,32]. The mechanisms underlying this association remain incompletely understood but may involve chronic structural remodeling of the cardiac conduction system, including fibrotic changes within the sinoatrial node and surrounding tissue.

Despite these observations, the majority of athletes with sinus bradycardia remain asymptomatic and do not develop clinically relevant conduction disorders. Nevertheless,

careful clinical evaluation is warranted in athletes presenting with symptoms such as syncope, dizziness, or exercise intolerance.

7. Diagnostic Evaluation of Sinus Bradycardia in Athletes

Accurate differentiation between physiological sinus bradycardia and pathological conduction abnormalities represents a key aspect of cardiovascular evaluation in athletes. Although reduced resting heart rate is a common and generally benign finding in trained individuals, certain clinical situations require careful diagnostic assessment to exclude underlying cardiac pathology.

The evaluation of sinus bradycardia in athletes should begin with a detailed clinical history and symptom assessment. Physiological bradycardia associated with athletic training is typically asymptomatic and discovered incidentally during routine cardiovascular screening. However, symptoms such as syncope, presyncope, dizziness, fatigue, or decreased exercise tolerance may indicate impaired cardiac output or conduction system disease and warrant further investigation [26,33].

Resting electrocardiography (ECG) represents the primary diagnostic tool in the assessment of athletes. Common ECG findings related to athletic training include sinus bradycardia, sinus arrhythmia, and first-degree atrioventricular block. These patterns are usually considered benign when observed in asymptomatic athletes without evidence of structural heart disease [26]. Nevertheless, certain electrocardiographic abnormalities—including high-grade atrioventricular block, pathological Q waves, or significant T-wave inversions—may suggest underlying cardiac pathology and require additional evaluation.

Ambulatory rhythm monitoring, typically performed using 24-hour Holter monitoring, may provide valuable information regarding intermittent bradyarrhythmias or nocturnal conduction disturbances. In trained athletes, sinus pauses of short duration may occur during sleep due to enhanced parasympathetic tone and are generally considered benign. However, prolonged pauses or pauses associated with symptoms may indicate sinus node dysfunction.

Exercise testing is another important diagnostic tool, as it allows evaluation of the heart rate response to physical exertion. In physiological bradycardia, heart rate typically increases rapidly and proportionally to exercise intensity, reflecting preserved chronotropic competence. In contrast, impaired heart rate acceleration during exercise may indicate chronotropic incompetence or dysfunction of the sinoatrial node.

Cardiac imaging also plays an essential role in the diagnostic process. Echocardiography is the first-line imaging modality and provides information regarding ventricular size, wall thickness,

and overall cardiac function. These findings help differentiate physiological adaptations associated with the athlete's heart from structural heart disease. In selected cases, cardiac magnetic resonance imaging may be performed to further evaluate myocardial structure or detect fibrosis that could affect the cardiac conduction system [29].

Through integration of clinical assessment, electrocardiographic findings, rhythm monitoring, exercise testing, and cardiac imaging, clinicians can effectively distinguish physiological sinus bradycardia related to athletic training from pathological conduction abnormalities requiring further management.

8. Pre-Participation Cardiovascular Screening and Clinical Guidelines

Pre-participation cardiovascular screening plays a central role in identifying potentially dangerous cardiac conditions in athletes. The primary objective of such screening programs is to prevent sudden cardiac death and other adverse cardiovascular events during sports participation.

Most screening protocols include a detailed medical history, physical examination, and resting electrocardiography. These components allow detection of many cardiovascular abnormalities that may predispose athletes to arrhythmias or structural heart disease.

International organizations, including the European Society of Cardiology and the International Olympic Committee, have developed guidelines for cardiovascular evaluation of athletes. These guidelines provide recommendations for the interpretation of electrocardiographic findings and the management of athletes with identified abnormalities [28].

According to current recommendations, isolated sinus bradycardia in asymptomatic athletes is considered a normal physiological finding and does not require additional evaluation. However, further diagnostic testing is recommended when bradycardia is accompanied by symptoms or other abnormal findings on ECG or imaging.

For example, athletes presenting with unexplained syncope, significant conduction abnormalities, or persistent arrhythmias should undergo comprehensive cardiovascular evaluation before being cleared for competitive sports.

In some cases, temporary restriction from competitive sports may be necessary until diagnostic evaluation is completed. Once pathological conditions have been excluded, athletes with physiological bradycardia can safely return to training and competition.

Implementation of standardized ECG interpretation criteria has significantly improved the effectiveness of athlete screening programs. By distinguishing normal training-related changes

from pathological findings, these criteria reduce unnecessary investigations while maintaining high sensitivity for detecting potentially dangerous cardiac conditions [20].

9. Discussion

Sinus bradycardia represents one of the most prominent physiological adaptations observed in trained athletes. Historically attributed primarily to increased parasympathetic tone, the mechanisms underlying exercise-induced bradycardia are now understood to involve both autonomic nervous system modulation and intrinsic remodeling of the sinoatrial node.

The autonomic hypothesis remains an important component of the physiological explanation for bradycardia in athletes. Increased parasympathetic activity and reduced sympathetic tone during rest contribute significantly to the reduction in resting heart rate observed in trained individuals [1].

However, recent molecular studies have demonstrated that intrinsic remodeling of pacemaker cells also plays a major role. Alterations in ion channel expression, particularly involving HCN4 channels responsible for the pacemaker current, have been shown to reduce intrinsic firing rates within the sinoatrial node [9,34,35].

These findings indicate that exercise-induced bradycardia represents a complex adaptation involving both functional and structural changes within the cardiac conduction system.

At the same time, growing evidence suggests that prolonged exposure to high volumes of endurance training may influence the long-term health of the cardiac conduction system. Epidemiological studies indicate that former endurance athletes may exhibit a higher incidence of sinus node dysfunction and atrioventricular conduction disturbances later in life [10,11].

Several hypotheses have been proposed to explain this association. Chronic mechanical stress, repetitive cardiac remodeling, and gradual fibrotic changes within the conduction system may contribute to the development of clinically significant bradyarrhythmias in some individuals.

Nevertheless, it is important to emphasize that the overall cardiovascular benefits of regular physical activity far outweigh the potential risks associated with conduction system remodeling. The vast majority of athletes with sinus bradycardia remain asymptomatic and never develop clinically significant arrhythmias.

Future research should focus on identifying factors that distinguish benign physiological adaptation from early pathological remodeling. Such factors may include genetic predisposition, duration and intensity of training, and individual variability in cardiac remodeling responses.

Improved understanding of these mechanisms will enhance our ability to evaluate athletes with bradycardia and provide individualized recommendations regarding sports participation.

10. Conclusions

Sinus bradycardia is a common and generally benign finding in trained athletes, reflecting physiological adaptation of the cardiovascular system to long-term endurance training. Enhanced parasympathetic activity, increased stroke volume, and intrinsic remodeling of the sinoatrial node all contribute to the reduction in resting heart rate observed in athletes.

Although most cases represent normal physiological adaptation, clinicians must remain vigilant for signs of pathological conduction system disease. Persistent bradycardia accompanied by symptoms, abnormal ECG findings, or structural cardiac abnormalities should prompt comprehensive diagnostic evaluation.

Modern screening strategies and standardized electrocardiographic interpretation criteria have significantly improved the ability to differentiate physiological bradycardia from pathological conditions. Through appropriate evaluation and monitoring, athletes with sinus bradycardia can usually continue to participate safely in competitive sports.

Continued research into the molecular and structural mechanisms of exercise-induced bradycardia will further clarify the long-term implications of endurance training on the cardiac conduction system and help refine clinical guidelines for athlete evaluation.

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