PATHOLOGICAL CARDIAC REMODELING IN ENDURANCE ATHLETES: A NARRATIVE REVIEW

REMODELAMENTO CARDÍACO PATOLÓGICO EM ATLETAS DE ENDURANCE: UMA REVISÃO NARRATIVA

REMODELADO CARDÍACO PATOLÓGICO EN ATLETAS DE RESISTENCIA: UNA REVISIÓN NARRATIVA

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ABSTRACT

Exercise-induced atrial remodeling in endurance athletes has emerged as a topic of growing interest in sports cardiology, particularly given the possibility that initially physiological adaptations may evolve into structural and functional changes with potential clinical relevance. Prolonged and high-intensity exercise imposes chronic volume overload on the atrial chambers, promoting progressive dilation, increased wall stress, and alterations in atrial mechanics. In this context, this narrative review aimed to critically analyze the available evidence on atrial remodeling in endurance athletes, with emphasis on the underlying mechanisms, determinants of adaptation, and the limits between physiology and potential pathology. The analyzed studies consistently demonstrate that endurance athletes present increased atrial volumes, especially of the left atrium, with a dose-response relationship

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between cumulative training load and the magnitude of dilation. From a functional perspective, the literature shows heterogeneous findings, with reduced atrial strain indices in some athlete groups, whereas other studies describe preservation of mechanical function within physiological limits. In addition, sex-related differences, training level, and duration of exposure appear to modulate the intensity of these adaptations. An association has also been observed between atrial remodeling, inflammatory biomarkers, and a higher propensity for the development of atrial fibrillation in veteran athletes, suggesting that atrial adaptation should not be interpreted in a dichotomous manner. It is concluded that atrial remodeling in endurance athletes represents an adaptive continuum whose final expression depends on the interaction among training load, individual susceptibility, and duration of exposure, requiring careful interpretation within the context of sports cardiology.

Keywords: Atrial Remodeling. Endurance Athletes. Atrial Fibrillation. Athlete's Heart. Sports Cardiology.

RESUMO

O remodelamento atrial induzido pelo exercício em atletas de endurance tem emergido como um tema de crescente interesse na cardiologia do esporte, especialmente diante da possibilidade de que adaptações inicialmente fisiológicas possam evoluir para alterações estruturais e funcionais com potencial impacto clínico. O exercício prolongado e de alta intensidade impõe sobrecarga volumétrica crônica às câmaras atriais, favorecendo dilatação progressiva, aumento do estresse parietal e alterações na mecânica atrial. Nesse contexto, esta revisão narrativa teve como objetivo analisar criticamente as evidências disponíveis sobre o remodelamento atrial em atletas de endurance, com ênfase nos mecanismos envolvidos, nos determinantes da adaptação e nos limites entre fisiologia e potencial patologia. Os estudos analisados demonstram que atletas de endurance apresentam, de forma consistente, aumento dos volumes atriais, especialmente do átrio esquerdo, com relação dose-resposta entre carga acumulada de treinamento e magnitude da dilatação. Do ponto de vista funcional, a literatura mostra achados heterogêneos, com redução dos índices de strain atrial em alguns grupos de atletas, enquanto outros estudos descrevem preservação da função mecânica dentro de limites fisiológicos. Além disso, diferenças relacionadas ao sexo, ao nível de treinamento e ao tempo de exposição parecem modular a intensidade dessas adaptações. Observou-se ainda associação entre remodelamento atrial, biomarcadores inflamatórios e maior propensão ao desenvolvimento de fibrilação atrial em atletas veteranos, sugerindo que a adaptação atrial não deve ser interpretada de forma dicotômica. Conclui-se que o remodelamento atrial em atletas de endurance integra um contínuo adaptativo, cuja expressão final depende da interação entre carga de treinamento, susceptibilidade individual e tempo de exposição, exigindo interpretação criteriosa no contexto da cardiologia esportiva.

Palavras-chave: Remodelamento Atrial. Atletas de Endurance. Fibrilação Atrial. Coração do Atleta. Cardiologia do Esporte.

RESUMEN

El remodelado auricular inducido por el ejercicio en atletas de resistencia ha emergido como un tema de creciente interés en la cardiología del deporte, especialmente ante la posibilidad de que adaptaciones inicialmente fisiológicas puedan evolucionar hacia alteraciones estructurales y funcionales con potencial impacto clínico. El ejercicio prolongado y de alta intensidad impone una sobrecarga crónica de volumen sobre las cavidades auriculares, favoreciendo la dilatación progresiva, el aumento del estrés parietal y alteraciones en la mecánica auricular. En este contexto, esta revisión narrativa tuvo como objetivo analizar críticamente la evidencia disponible sobre el remodelado auricular en atletas de resistencia, con énfasis en los mecanismos implicados, los determinantes de la adaptación y los límites

entre la fisiología y la potencial patología. Los estudios analizados demuestran de manera consistente que los atletas de resistencia presentan aumento de los volúmenes auriculares, especialmente de la aurícula izquierda, con una relación dosis-respuesta entre la carga acumulada de entrenamiento y la magnitud de la dilatación. Desde el punto de vista funcional, la literatura muestra hallazgos heterogéneos, con reducción de los índices de strain auricular en algunos grupos de atletas, mientras que otros estudios describen preservación de la función mecánica dentro de límites fisiológicos. Además, las diferencias relacionadas con el sexo, el nivel de entrenamiento y el tiempo de exposición parecen modular la intensidad de estas adaptaciones. También se ha observado una asociación entre el remodelado auricular, biomarcadores inflamatorios y una mayor propensión al desarrollo de fibrilación auricular en atletas veteranos, lo que sugiere que la adaptación auricular no debe interpretarse de forma dicotómica. Se concluye que el remodelado auricular en atletas de resistencia integra un continuo adaptativo, cuya expresión final depende de la interacción entre la carga de entrenamiento, la susceptibilidad individual y el tiempo de exposición, exigiendo una interpretación cuidadosa en el contexto de la cardiología deportiva.

Palabras clave: Remodelado Auricular. Atletas de Resistencia. Fibrilación Auricular. Corazón del Atleta. Cardiología del Deporte.

1 INTRODUCTION

Exercise-induced cardiac remodeling is a phenomenon well described in the literature, characterized by structural and functional adaptations of the heart in response to chronic physical training, especially in endurance athletes (Pelliccia et al., 2005; D'andrea et al., 2016). These adaptations include enlargement of cardiac cavities, changes in myocardial mass, and changes in diastolic and systolic function, and are largely considered physiological.

Although regular physical activity is widely associated with cardiovascular benefits, chronic exposure to extreme loads has been related to the development of adaptations that may exceed the limits of benign physiology, a phenomenon often discussed under the concept of "Phidippides cardiomyopathy" (Contreras-Briceño et al., 2021; Spencer et al., 2024).

However, recent evidence suggests that, under certain conditions, these adaptations can take on potentially pathological characteristics, particularly when associated with high training volumes and long periods of exposure (D'andrea et al., 2010; Spencer et al., 2024). This phenomenon is characterized by exaggerated structural remodeling, involving myocardial fibrosis deposition and tissue disorganization mediated by inflammatory processes (Elliott et al., 2016; Contreras-Briceño et al., 2021; Spencer et al., 2024). Such modifications, driven by high parietal stress in thin-walled chambers, can mimic the phenotype of arrhythmogenic cardiomyopathies and consolidate the substrate for atrial fibrillation, showing that the cumulative load of effort is the critical determinant for this transition (Wilhelm et al., 2012; Pagourelias et al., 2013; Sanz-de la Garza et al., 2016; Diaz Babio et al., 2021).

In this context, atrial remodeling has stood out as an area of growing interest, especially due to its relationship with structural and functional changes that go beyond physiological adaptation (D'andrea et al., 2016; Simard et al., 2022).

The atrium, due to its thinner structure and greater sensitivity to variations in hemodynamic load, may be particularly susceptible to chronic stress imposed by high-intensity, long-duration exercise (Contreras-briceño et al., 2021). Alterations such as atrial dilation, increased parietal stress, and changes in myocardial tissue have been described in endurance athletes, suggesting a possible spectrum between physiological adaptation and pathological remodeling (Wilhelm et al., 2012; Sanz-de la garza et al., 2016).

Despite the recognition of exercise-induced physiological cardiac adaptations, recent evidence has challenged the classical paradigm by suggesting that chronic exposure to high volumes of training can promote structural and functional changes with a potential

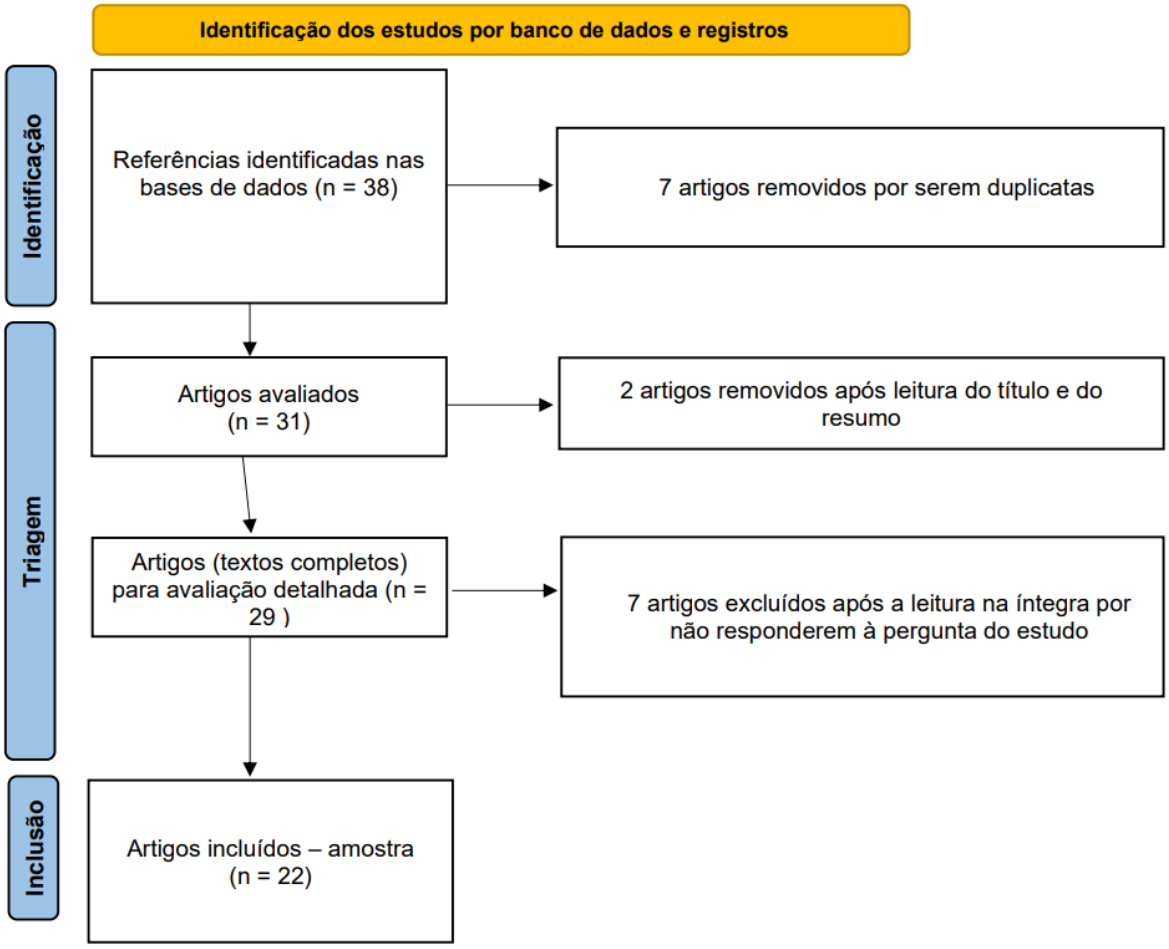
pathological character. In this context, a central controversy remains in the literature: whether the atrial remodeling observed in endurance athletes represents a benign adaptation or a progressive process associated with increased arrhythmic risk. Thus, this review aims to critically analyze the available evidence, seeking to clarify the mechanisms, determinants, and limits that distinguish physiological adaptation from potentially pathological atrial remodeling

2 METHODOLOGY

The present study was conducted as a narrative review of the literature dedicated to exercise-induced atrial remodeling in endurance athletes, focusing on potentially pathological structural and functional changes. The literature search was carried out in the PubMed, Scopus and Google Scholar databases, using the descriptors in English "atrial remodeling", "exercise-induced cardiac remodeling", "endurance athletes", "atrial dilation", "atrial function", "left atrium", "right atrium" and "exercise", combined using the Boolean operators AND and OR. Priority was given to studies published between 2016 and 2026, written in English, available in full, and conducted in humans, which directly addressed atrial remodeling associated with endurance exercise. In addition, relevant studies published from 2005 onwards were incorporated with the objective of providing theoretical and physiological basis, especially with regard to cardiac anatomy, healthy heart physiology, and cardiovascular adaptations to exercise. Duplicate studies, experimental research in animal models, publications not directly related to the theme, and studies focused predominantly on structural heart diseases unrelated to exercise were excluded. The initial search identified 38 records; After reading the titles and abstracts, 29 studies were considered potentially eligible and, after removing duplicates, 22 articles were included for final analysis.

Figure 1

Flowchart of the process of identification, screening and eligibility of the selected studies



Legend: Flowchart of the selection process of the studies included in the narrative review, including the stages of identification in the databases, removal of duplicates, screening by title and abstract, evaluation of full texts and final inclusion of the sample.
Source: Authors.

3 RESULTS

Based on the 22 selected articles, the results indicate a scientific convergence regarding the existence of significant structural atrial remodeling induced by endurance exercise, characterized primarily by progressive biatrial dilation (Pelliccia et al., 2005; D'Andrea et al., 2010; Wilhelm et al., 2012; Pagourelias et al., 2013). Elite athletes have substantially higher atrial volumes than sedentary individuals, with cumulative training volume acting as the main predictor of this adaptation (D'Andrea et al., 2010; Zimmermann et al., 2022). A dose-response relationship was identified in which the probability of clinically relevant atrial dilation (LAVi 34 mL/m²) rises sharply after the threshold of 3,600 hours of exercise accumulated over a lifetime (Wilhelm et al., 2012; Diaz Babio et al., 2021).

In the functional sphere, a complex pattern of adaptation of atrial mechanics through strain analysis is observed. Several studies have shown that high-performance athletes

have lower reservoir strain and contractility values than those of sedentary controls at rest (D'Ascenzi et al., 2013; Lakatos et al., 2020; Di Gioia et al., 2025). Although this reduction is often interpreted as a recruitable "functional reserve" during exertion, there is divergent evidence: while some authors suggest that function remains stable and within physiological limits (D'Ascenzi et al., 2014, 2015), findings in amateur athletes indicate that short-term training may actually increase left atrial contractile strain, especially in young women (Szałek-Goralewska et al., 2025).

Differences between sexes are also determinants of the results, with males having larger cavity volumes and a more pronounced remodeling (Simard et al., 2022; Di Gioia et al., 2025). However, women tend to exhibit superior mechanical performance profiles, possibly due to the protective influence of ovarian hormones in attenuating inflammatory processes (Simard et al., 2022). Regarding the acute effects of extreme exercise, the findings are heterogeneous. Some cohorts demonstrate a transient, dose-dependent impairment in right atrial reservoir function immediately after ultra-endurance competitions (Sanz-de la Garza et al., 2016), whereas studies in master athletes have not identified acute biatrial dysfunctions after ultramarathons (Cavigli et al., 2022).

Clinically, atrial remodeling is recognized as the main mechanical promoter for the development of arrhythmias, with atrial fibrillation being the most prevalent manifestation in veteran athletes (Elliott et al., 2016; Spencer et al., 2024). This pathological process is supported by the positive correlation (rho = 0.51) between increased atrial volume and biomarkers of inflammation and fibrosis, such as sVCAM-1 (Contreras-Briceño et al., 2021).

However, a relevant diagnostic mismatch is observed: marked structural dilation is often not accompanied by changes in P-wave morphology on the electrocardiogram, suggesting that in a healthy "athlete's heart", electrical stability and tissue compliance can be preserved despite the volumetric increase (D'Ascenzi et al., 2016).

4 DISCUSSION

A healthy heart has a high capacity to adapt to the hemodynamic demands imposed by physical exercise. However, when subjected to high chronic loads, especially in endurance athletes, structural and functional changes may occur that go beyond the spectrum of physiological adaptation (D'Andrea et al., 2016; Spencer et al., 2024). The atrium, due to its smaller thickness and greater compliance, is particularly susceptible to increased parietal stress due to repetitive volumetric overload (Wilhelm et al., 2012; Sanz-de la Garza et al., 2016). According to Laplace's law, the increase in the radius of the atrial cavity leads to an increase in the tension in the wall, favoring structural remodeling

processes (Contreras-Briceño et al., 2021).

In this context, the mechanisms underlying these changes involve a cascade of molecular and morphological events triggered by persistent mechanical stretching. Biophysically, volume overload elevates intra-atrial pressures, acting as a trigger for the activation of pro-inflammatory pathways and the release of cytokines, such as tumor necrosis factor-alpha (TNF-), in addition to promoting tissue oxidative stress (Elliott et al., 2016; Spencer et al., 2024). Such molecular stimuli result in the activation of fibroblasts and the subsequent excessive deposition of collagen in the extracellular matrix, consolidating interstitial fibrosis (Elliott et al., 2016; Contreras-Briceño et al., 2021). From a morphological point of view, this process culminates in progressive dilation and increased atrial volume, modifying the myocardial architecture to accommodate the hemodynamic burden of chronic exercise (D'Andrea et al., 2016; Spencer et al., 2024).

Furthermore, contemporary evidence demonstrates that sex is a relevant determinant in the magnitude of this remodeling. Male athletes tend to have higher indexed atrial volumes and cavity diameters than women, reflecting, in part, larger body size and exposure to higher training ceilings (Simard et al., 2022; Di Gioia et al., 2025). On the other hand, hormonal factors seem to exert a protective influence; estrogen can modulate the adaptive response by attenuating inflammation and myocardial hypertrophy, resulting in distinct functional profiles (Simard et al., 2022; Spencer et al., 2024). Thus, although both sexes have significant adaptations to endurance, women often preserve higher rates of atrial mechanics compared to men (Simard et al., 2022; Di Gioia et al., 2025).

Regarding the clinical repercussions, atrial remodeling directly impacts the mechanical function of the chamber, and can reduce the rates of myocardial deformation (*strain*) of the reservoir and contractility (D'Ascenzi et al., 2015; Di Gioia et al., 2025). While in physiological "athlete's heart" compliance is preserved and atrial stiffness remains low, the transition to pathology is marked by increased stiffness and incipient systolic failure (D'Ascenzi et al., 2015; Spencer et al., 2024). These functional changes are associated with adverse outcomes, since tissue disorganization compromises the functional reserve necessary to sustain high cardiac output during exertion (D'Andrea et al., 2016; Spencer et al., 2024). Therefore, the clinical distinction between benign adaptation and sports-induced atrial cardiomyopathy is imperative for cardiovascular risk stratification (Pagourelias et al., 2013; Spencer et al., 2024).

The interpretation of atrial functional alterations in athletes remains controversial. While studies have shown a reduction in reservoir strain and contractility indices in high-performance athletes (D'Ascenzi et al., 2013; Lakatos et al., 2020; Di Gioia et al., 2025),

suggesting possible functional impairment, other evidence indicates preservation of atrial function within physiological limits (D'Ascenzi et al., 2014; 2015). Additionally, investigations in amateur athletes undergoing short-term training demonstrate an increase in contractile strain, particularly in young women (Szałek-Goralewska et al., 2025). These divergences suggest that the atrial functional response is not uniform, being modulated by factors such as training intensity, exposure time, and characteristics of the population studied.

From a clinical point of view, the evaluation of atrial enlargement can be performed using standardized echocardiographic parameters, such as the volume of the left atrium indexed to the body surface. Values higher than 34 mL/m² are generally considered increased, and can be classified into mild, moderate, and severe degrees according to the magnitude of dilation (Lang et al., 2015). However, in athletes, these boundaries may overlap with physiological adaptations, which makes it difficult to distinguish between benign remodeling and potentially pathological changes. This environment can favor the development of alterations such as atrial dilation, inflammation, and extracellular matrix deposition, contributing to the formation of fibrosis (Elliott et al., 2016; Spencer et al., 2024). These structural changes can impact electrical conduction and atrial mechanical function (D'Ascenzi et al., 2015; Spencer et al., 2024).

In addition, repeated episodes of acute hemodynamic stress may contribute to cumulative myocardial microlesions (Spencer et al., 2024). Studies show that these changes can occur even in the absence of previous cardiovascular disease (D'Ascenzi et al., 2016). Exercise-induced atrial remodeling has been described as potentially associated with the development of arrhythmias, especially atrial fibrillation, although this relationship is not uniform across studies (Elliott et al., 2016; Wilhelm et al., 2012).

The acute effects of endurance exercise on atrial function also present divergent results in the literature. While some studies demonstrate transient and dose-dependent impairment of atrial function after ultra-endurance competitions (Sanz-de la Garza et al., 2016), other investigations do not identify significant changes in master athletes subjected to extreme exertion (Cavigli et al., 2022). These discrepancies may reflect differences in athlete profile, fitness level, and magnitude of load imposed, suggesting that the acute impact of exercise is not uniform across different populations.

It is worth noting that atrial remodeling has been associated with the development of arrhythmias, particularly atrial fibrillation in veteran athletes (Elliott et al., 2016; Spencer et al., 2024; Wilhelm et al., 2012). However, this association is not uniform, since significant structural changes are often not accompanied by electrocardiographic modifications, as evidenced by the preservation of P-wave morphology even in the presence of atrial dilation

(D'Ascenzi et al., 2016). This mismatch suggests that structural adaptation can coexist with electrical stability in certain contexts, reinforcing the complexity of the transition between physiology and pathology.

Thus, the findings suggest that atrial remodeling in endurance athletes should not be interpreted in a dichotomous way, but as a progressive process, in which initially beneficial adaptations can evolve into structural changes with potential clinical impact, depending on the intensity, duration and individual susceptibility.

5 CONCLUSION

Endurance exercise-induced atrial remodeling represents a complex phenomenon, which involves a spectrum between physiological adaptation and possible structural changes with clinical relevance. Evidence suggests that factors such as intensity, duration and accumulated training load play a fundamental role in this process.

In this sense, atrial remodeling should not be understood as an isolated phenomenon, but as part of an adaptive continuum whose final expression depends on the interaction between training load, individual susceptibility and exposure time.

Understanding these mechanisms is essential for the proper clinical evaluation of athletes, allowing the distinction of benign adaptations from possible alterations with potential impact on cardiovascular health.

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