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Exercise Volume and the Risk of Atrial Fibrillation in Athletes: A Literature Review

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Abstract

Background. Despite the proven positive effects of physical activity on the cardiovascular system, an increasing number of veteran athletes are being diagnosed with atrial fibrillation (AF). Over the past few decades, a growing body of evidence suggests that prolonged endurance training may contribute to the development of AF in certain groups of athletes. The recognition of this link has sparked a debate among researchers regarding the determination of the amount of training at which the risk of AF significantly increases. However, current research findings are inconsistent regarding this relationship; therefore, the relationship between training volume, the type of sport practiced, and the risk of arrhythmia remains not fully understood.

Aim. This literature review aims to review the mechanisms involved in the development of atrial fibrillation in athletes and to evaluate the association between exercise volume and the risk of atrial fibrillation.

Material and methods. A literature review was conducted using PubMed and Google Scholar. Meta-analyses, systematic reviews, randomized control trials, systematic and literature reviews from past 20 years were analyzed.

Results. The strongest available evidence suggests that athletes have approximately 2.5 times the risk of AF compared to the general population. The available data suggest a U-shaped or J-shaped relationship between exercise and the risk of AF. Moderate physical activity still remains cardioprotective. Studies involving the population of female athletes remain limited.

Conclusions. The relationship between the risk of AF and physical activity is complex and seems paradoxical. Moderate physical activity has a protective effect, while excessive training increases the risk of AF.

Keywords: atrial fibrillation; athletes; endurance exercise; physical activity; sports cardiology; exercise volume; cardiac remodeling

Introduction

Atrial fibrillation (AF) is the most common arrhythmia worldwide and poses a serious health problem due to its association with higher risk of stroke, heart failure, and overall mortality in the general population [1]. The prevalence of this condition increases significantly with age, but it is increasingly being diagnosed in groups previously considered to be at low cardiovascular risk, including athletes. [2].

Regular physical activity is a recognized factor in the prevention of cardiovascular disease and has been shown to improve metabolic health and reduce mortality [2]. However, despite its well-established benefits, the relationship between physical activity and the incidence of AF appears to be complex and potentially paradoxical. Moderate physical activity has a protective effect; however, a growing body of evidence suggests that long-term, high-intensity training increases the risk of AF, which highlights a dose-dependent relationship [1,3].

Recent studies indicate that athletes especially those engaged in endurance disciplines have an increased risk of AF compared to the general population, with some reports suggesting a 2-to-10-fold greater risk. This observation has led to the hypothesis of a nonlinear U-shaped relationship between physical activity and the risk of AF, in which both low and high levels of physical activity increase the risk of AF. [2] Given these uncertainties, there is a need for a comprehensive synthesis of available evidence to better understand both the mechanisms underlying atrial fibrillation in athletes and the potential association between exercise volume and AF risk.

Patomechanism

It is believed that the development of AF in athletes results from several pathological mechanisms, such as structural and electrical remodeling, oxidative stress, and the inflammatory response. Long-term exposure to high-intensity and high-volume endurance exercise is thought to induce adaptive changes that, while initially physiological, may become arrhythmogenic over time [4,5].

Structural remodeling

One of the main mechanisms underlying the development of AF is structural remodeling of the atria. Chronic volume and pressure overload during many years of training can lead to enlargement of the left atrium and stretching of its walls. Long-term training may activate profibrotic pathways (e.g., TGF-β, galectin-3) and promote focal fibrosis, although in many master athletes its severity is mild and uneven [2,4,6]. In endurance athletes, a sustained

increase in minute volume leads to stretching of the left atrial wall and its enlargement; the degree of enlargement increases with the volume and intensity of training [7]. Increased muscle mass often does not return to its original state, suggesting that some changes are irreversible (e.g., fibrosis) [8,9].

Electrical remodeling

Another significant factor in the development of atrial fibrillation is electrical remodeling of the myocardium. Repeated exposure to high training volumes leads to a shortening of the refractory period and increased heterogeneity in impulse conduction, which facilitates the formation of re-entry circuits responsible for AF initiation and maintenance [5,3]. Ectopic activities originating in the pulmonary veins can enter such re-entry circuits and cause atrial fibrillation [3].

Autonomic adaptations

Adaptations of the autonomic nervous system also play a major role in the development of atrial fibrillation. Endurance athletes typically exhibit increased vagal tone and resting bradycardia. It has been shown that increased vagal activity shortens atrial repolarization time and promotes repolarization dispersion, which increases the chance of an episode of atrial fibrillation. This effect is strongest during sleep and rest, when parasympathetic tone increases [5,3]. This so-called “vagal AF” phenotype is frequently observed in otherwise healthy, young athletes.

Inflammatory response

Another important mechanism is exercise-induced myocardial inflammatory response. Intense and prolonged endurance exercise may result in repetitive microinjury to the myocardium, leading to inflammatory activation and subsequent fibrotic remodeling [4,8]. Repeated exposure to such stress may trigger chronic low-grade inflammation and activation of fibrotic pathways, ultimately resulting in atrial fibrosis [9]. Fibrotic remodeling disrupts normal electrical conduction and creates a substrate for the perpetuation of atrial fibrillation [9].

Hemodynamic factors

Hemodynamic factors, defined as a sustained increase in atrial pressure and cardiac output during exercise, cause stretching of the atrial myocardium. This as a result affects the function of ion channels in the membranes of cardiomyocytes and promotes arrhythmia [6].

Additionally, long-term endurance training has been associated with changes in ventricular compliance and diastolic function, indirectly affecting atrial load and function [10].

Others

Some studies suggest that individual predispositions play a significant role. Factors such as gender, age, and the lifetime cumulative amount of training influence the risk [11,12]. For instance, men-particularly those with a long history of training-have a higher risk compared to women [11].

Finally, the concept of an "exercise dose" is gaining increasing attention. Moderate physical activity helps prevent atrial fibrillation; however, excessive training can exceed the heart's physiological limits of adaptation, leading to pathological remodeling and increasing the risk of arrhythmia [5,13,14]. However, the precise threshold at which exercise transitions from beneficial to potentially harmful remains unclear and is a subject of ongoing research.

Epidemiology and risk factors

Over the past few decades, AF has become an increasingly common condition among athletes particularly among long-term endurance athletes [1]. Although moderate physical activity is strongly associated with a reduction in cardiovascular morbidity and mortality, epidemiological data suggest that excessive exercise may increase the risk of AF in certain groups of athletes [1,15].

Current evidence suggests that the relationship between training volume and the risk of AF follows a nonlinear J-shaped or U-shaped curve [1,16]. This phenomenon is most consistently observed in endurance disciplines characterized by prolonged dynamic exercise, including marathon running and cycling [17].

Several epidemiological studies and meta-analyses have shown a significant increase in the prevalence of AF among endurance athletes compared to a non-athletic control group [1,15]. One of the earliest meta-analyses showed that athletes had approximately a fivefold higher risk compared to a sedentary group [15]. More recent analyses confirmed that endurance athletes may exhibit a 2,5 times higher AF incidence, particularly in middle-aged male athletes with extensive training histories [1].

Lifetime cumulative training load appears to be one of the most important predictors of the risk of developing AF. Studies indicate that the risk of AF increases among athletes who engage in high-volume training, especially when the amount of training exceeds 10 hours per week or several thousand cumulative lifetime training hours [16,17]. Long-term competitive

participation lasting more than 10 years has also been associated with increased AF prevalence [17]. It is worth noting that not all forms of physical activity carry the same risk. Endurance sports that require prolonged aerobic exercise appear to carry the highest risk of developing arrhythmias, while resistance training and moderate recreational activity are less strongly associated with the risk of AF [17].

Sex-related differences represent another important aspect of AF in athletes. Most of the available evidence shows that male athletes have a significantly higher risk of developing AF than female athletes. [1,18]. Male sex has been identified as an independent predictor of AF in athletes, which may partially explain why most AF cohorts consist primarily of men [18]. Several mechanisms have been proposed to explain this disparity, including larger atrial dimensions, greater cumulative exercise exposure, higher prevalence of atrial fibrosis, and differences in autonomic nervous system adaptation among male athletes [19].

Female athletes, on the other hand, tend to exhibit a lower incidence of AF despite participation in similar endurance disciplines [18]. Hormonal factors, particularly the protective effects of estrogen on myocardial remodeling and inflammatory response may contribute to this phenomenon [20]. However, women remain underrepresented in sports cardiology research, and the current evidence regarding the prevalence of sports-related AF in women remains limited [18].

In the athlete population, just as in the general population, age is strongly associated with the prevalence of AF. Although younger athletes may sometimes experience atrial ectopy and exercise-induced palpitations, clinically significant AF most commonly occurs in athletes over the age of 40 with a long history of training. [1,21]. The delayed onset of AF likely reflects the cumulative effects of chronic hemodynamic stress, atrial enlargement, fibrosis, and electrical remodeling that develop progressively over years of intensive exercise exposure [21].

Additional cardiovascular and lifestyle-related risk factors may further potentiate AF development in athletes. Hypertension, obstructive sleep apnea, alcohol consumption, obesity, genetic predisposition, and the use of performance-enhancing substances have all been demonstrated as a contributing factors [22].

Despite increasing awareness of AF in athletes, important uncertainties remain regarding the precise threshold at which exercise transitions from beneficial to potentially harmful [16]. Current evidence continues to strongly support moderate physical activity as a cornerstone of cardiovascular prevention. However, prolonged high-volume endurance exercise, particularly in middle-aged male athletes with extensive cumulative training exposure, may require individualized cardiovascular assessment and monitoring [1,16]

Discussions

The available literature suggests that long-term, high-volume endurance training increases the risk of AF, especially in middle-aged male athletes with extensive training histories [1,15]. Despite the growing number of studies on this phenomenon, some important questions remain unanswered, and many aspects of exercise-related AF are based on limited evidence.

One of the most widely discussed aspects involves determining the threshold above which physical activity changes from being protective to potentially arrhythmogenic. Although many studies confirm a J-shaped or U-shaped relationship between physical exertion and the incidence of AF [1,16], there is still no universally accepted definition of "excessive physical exercise.". Different studies use highly variable measures of training intensity and cumulative lifetime exercise volume, making direct comparisons difficult [17,21]. Some authors suggest a threshold of about 10 hours per week or several thousand lifetime training hours [16,17]. Others, however, place greater emphasis on the type of training rather than its total volume [3]. Consequently, current evidence remains insufficient to establish clear evidence-based recommendations regarding safe upper limits of endurance exercise.

Another important limitation of the current literature is the predominance of observational and retrospective study designs. Most available data originate from cohort studies, athlete registries, or case-control analyses [1,15,18]. While these studies demonstrate associations between endurance exercise and AF, they cannot definitively establish causality. Athletes frequently differ from the general population in numerous additional factors, including body composition, autonomic tone, dietary habits, alcohol consumption, and use of supplements or performance-enhancing substances [22]. These confounding variables may partly influence the observed increase in AF prevalence.

The mechanisms underlying the development of AF in athletes are also not fully understood. Although atrial enlargement, fibrosis, autonomic remodeling, and inflammation are commonly proposed mechanisms [4,5,9,12], direct human evidence remains limited. Much of the knowledge about these mechanisms comes from animal studies and indirect findings from imaging studies. For example, experimental models have demonstrated that prolonged endurance exercise may promote atrial fibrosis and autonomic imbalance [19]; however, it is unclear to what extent these findings apply to athletes participating in endurance sports. Similarly, although imaging studies have revealed structural remodeling of the atria in endurance athletes [7, 10], it is uncertain whether these changes always represent pathological remodeling or whether they may partly reflect a physiological adaptation to exercise.

Sex-related differences in exercise-induced AF also require further investigation. Most currently available studies include predominantly male cohorts, resulting in a significant underrepresentation of female athletes [18]. For this reason, the lower prevalence of AF among female athletes may be due in part to insufficient statistical power rather than to an actual protective effect. Hormonal factors have been proposed as potential mechanisms, particularly the anti-inflammatory and antifibrotic effects of estrogens; however, there is still a lack of reliable prospective studies in a female population [20]. Future studies should focus more on sex-related differences in determining the risk of AF in athletes.

The least understood risk factor for AF is individual genetic predisposition. Available evidence suggests that some athletes have an innate susceptibility to arrhythmias that becomes clinically significant only after prolonged exposure to excessive physical exertion [22]. However, the interaction between genetic factors and exercise-induced cardiac remodeling remains poorly understood, it is possible that in individuals with a genetic predisposition, endurance exercise primarily acts as a triggering factor rather than an independent cause of the condition in all athletes.

The heterogeneity of athletic populations also complicates interpretation of the available evidence. Various studies compare athletes who participate in different sports, at different intensities, and at different skill levels [1,15]. Turns out that ports such as marathon running, cycling, rowing, and triathlon place greater stress on the cardiovascular system and autonomic nervous systems than resistance training or intermittent sports [17]. For that reason, future studies should aim to analyze sport-specific AF risk instead of treating athletes as a homogeneous population.

Nevertheless, based on current knowledge, the benefits of participating in sports still outweigh the risk of developing AF [16]. Moderate aerobic exercise reduces morbidity and mortality of cardiovascular disease and decreases all-cause mortality [16]. For this reason, the potential increase in AF risk observed in population of endurance athletes should not discourage participation in regular physical activity. Instead, current evidence supports a more individualized approach, particularly for aging endurance athletes with long-standing high-volume training exposure and additional cardiovascular risk factors.

Finally, future research should prioritize large prospective longitudinal studies with standardized definitions of training exposure and balanced representation of both sexes. A better understanding of the relationship between training load, structural remodeling of the heart, and the development of arrhythmias will help identify athletes at increased risk for AF.

Despite the observed increase in the prevalence of atrial fibrillation among endurance athletes, it is not currently possible to recommend regularly limiting physical activity, as the available evidence does not support a cause-and-effect relationship.

Conclusions

This literature review shows that the relationship between physical exertion and AF is not linear. Although regular physical activity is a recognized protective factor against cardiovascular disease, prolonged high-volume training increases the risk of developing AF in population of athletes, mainly in middle-aged men with many years of competitive endurance sports participation.

Current evidence suggests that exercise-induced AF results from the interaction of several complementary pathophysiological mechanisms rather than a single isolated process. Current scientific evidence suggests that exercise-induced AF results from the interaction of several patomechanisms. Chronic exercise promotes structural remodeling, manifested by atrial enlargement, due to prolonged volume overload. Repeated high mechanical stress on the atria leads to fibrosis, which creates conditions for abnormal impulse conduction. In addition, athletes develop autonomic nervous system adaptations, in this case increased vagal tone, which shortens the refractory period in the atria and promotes the formation of reentry loops. Additional factors such as an inflammatory response, oxidative stress, electrical remodeling, and genetic predisposition may lead to the induction of arrhythmias in predisposed individuals in the future. The available evidence suggests that AF in athletes is therefore the result of cumulative structural and electrophysiological adaptations rather than acute exercise exposure alone.

The largest increase in the prevalence of AF among athletes was observed among those who participate in marathon running, cycling, rowing, cross-country skiing, and triathlon. Although there is currently no generally accepted definition of an “excessive” amount of training, the available literature indicates that an increased risk emerges after many years of high-volume training. A number of studies suggest that weekly training exceeding approximately 8–10 hours or a total lifetime training time of several thousand hours may be associated with a higher incidence of atrial fibrillation compared to recreationally active individuals.

However, these threshold values should be interpreted with caution, as the extent of physical activity was measured differently across studies, and there remains significant heterogeneity in the results.

Importantly, the data currently available do not justify advising against regular physical activity. Instead, they point to the need for an individual assessment of cardiovascular health in athletes undergoing long-term, high-volume endurance training, especially in those over 40 or those with additional cardiovascular risk factors. Clinicians should keep in mind that exercise-induced AF is the result of many coexisting factors, and exercise is not the sole cause. Future research should focus on well-designed prospective studies that use standardized measures of training volume and include an adequate number of female participants. A thorough understanding of the relationship between training volume and the incidence of AF in athletes will be crucial for establishing evidence-based recommendations regarding the upper limits of training volume that do not increase the risk of arrhythmia.

Disclosure

AUTHOR'S CONTRIBUTION

Conceptualization: Kamil Marchlik

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Software: not applicable;

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Artificial Intelligence use declaration

AI tools were used only to refine the language and improve text readability. The authors retain full accountability for the academic integrity of the work. The conceptual design, literature review, data analysis, and final drafting were executed solely by the authors.

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