NICOLAUS COPERNICUS UNIVERSITY IN TORUŃ

QUALITY IN SPORT

eISSN 2450-3118 · Open Access · Peer-reviewed

apcz.umk.pl/QS · Nicolaus Copernicus University in Toruń

Cite as: JURKOWSKA, Klaudia, STANIK, Martyna, KĘPA, Adrianna, BEŁC, Ewa, GÓRAK, Joanna, MAHONEY, Eleonora Y., RELISZA, Agata, STADNIK, Aleksandra, TRZYNA, Elżbieta and GLIWA, Anna. The impact of intensive endurance training on the risk of atrial fibrillation and quality of life in veteran athletes – a literature review. Quality in Sport. 2026;55:71472. https://doi.org/10.12775/QS.2026.55.71472

ARTICLE TIMELINE

Received: 08.05.2026 Revised: 15.05.2026

Accepted: 15.05.2026 Published: 22.05.2026

INDEXING & EVALUATION

MEiN points: 20 Unique ID: 201398

Disciplines: Medical Sciences; Health Sciences

The journal has been awarded 20 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32553). Unique Journal Identifier: 201398. Scientific disciplines: Medical Sciences; Health Sciences.

Punkty Ministerialne z 2019 – aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Nauki medyczne; Nauki o zdrowiu.

The impact of intensive endurance training on the risk of atrial fibrillation and quality of life in veteran athletes – a literature review

Klaudia Jurkowska

https://orcid.org/0009-0003-0205-6999

kjurkowska070@gmail.com

Medical University of Silesia in Katowice, Katowice, Poland

Martyna Stanik

https://orcid.org/0009-0001-6539-8932

martyna.stanik@op.pl

Medical University of Warsaw, Warsaw, Poland

Adrianna Kępa

https://orcid.org/0009-0001-5833-7461

adriannaka7@gmail.com

Józef Struś Multispecialist Municipal Hospital, ul. Szwajcarska 3, 61-285 Poznań, Poland

Ewa Bełc

https://orcid.org/0009-0004-0224-8162

ewa.belc2203@gmail.com

Intern doctor, USK nr 2, Medical University of Lodz, Żeromskiego ST. 113, 90-549 Lodz, Poland

Joanna Górak

https://orcid.org/0009-0006-5536-8269

asia.gorak3@gmail.com

Clinical Department of Dermatology, AMiSNS St. Adalbert Hospital in Gdańsk

Eleonora Y. Mahoney

https://orcid.org/0009-0003-0846-9727

eleonora.mahoney.aiesec@gmail.com

Medical University of Gdańsk, Gdańsk, Poland

Agata Relisza

https://orcid.org/0009-0004-6744-8667

reliszka.agata@gmail.com

Provincial Hospital named after Maria Skłodowska-Curie.: 95-100 Zgierz, ul. Parzęczewska 35, PL

Aleksandra Stadnik

https://orcid.org/0009-0005-4764-3980

aleksandrastadnik2@gmail.com

Medical Intern, University Clinical Hospital in Wroclaw, Borowska 213, 50-556 Wroclaw, Poland

Elżbieta Trzyna

https://orcid.org/0009-0003-0746-6989

el.trzyna@gmail.com

University Children's Hospital in Cracow, Cracow, Poland

Anna Gliwa

https://orcid.org/0009-0006-0251-0591

annagliwa97@gmail.com

Wroclaw Medical University , Wrocław ,Poland

Abstract

Background: Regular physical activity is a cornerstone of cardiovascular prevention; however, recent evidence points to a "physical activity paradox." Master athletes engaged in long-term, high-intensity endurance sports show an increased incidence of atrial fibrillation (AF). This presents challenges regarding training safety and the quality of life for patients whose identity is tied to sports.

Aim: To summarize current knowledge on the link between long-term endurance training and AF risk in master athletes, and to analyze its impact on their quality of life and physical performance.

Materials and Methods: A literature review (PubMed, Scopus, Web of Science, 2020–2026) was conducted, including cohort studies, meta-analyses, and ESC guidelines. Fifty-eight relevant references were analyzed.

Results: Analysis confirms a "U-shaped" or "J-shaped" relationship between exercise dose and AF risk. Endurance athletes have a 2 to 10 times higher AF risk than sedentary individuals, linked to structural remodeling (atrial enlargement, fibrosis) and autonomic changes. AF significantly reduces quality of life in previously active individuals. Moderate training reduction and early radiofrequency (RF) ablation are highly effective in improving outcomes.

Conclusions: Despite arrhythmia risks, the cardiovascular benefits of exercise outweigh mortality risks; however, master athletes require an individualized clinical approach. Education on intensity modification and monitoring psychophysical well-being are key to maintaining high quality of life in these patients.

Keywords: master athletes, atrial fibrillation, athlete's heart, quality of life, endurance training.

Introduction

Atrial fibrillation (AF) is currently the most frequently diagnosed sustained supraventricular arrhythmia in the general population [22, 28]. For decades, evidence-based medicine has promoted regular physical activity as the cornerstone of primary and secondary prevention of cardiovascular diseases, emphasizing its beneficial impact on reducing blood pressure, improving lipid profiles, and controlling glycemia [9, 53]. Nevertheless, progress in the field of sports cardiology has identified a specific group of patients—veteran athletes—in whom long-term exposure to extreme hemodynamic loads leads to a phenomenon known as the "physical activity paradox." In this population, despite the absence of classical risk factors such as obesity, smoking, or metabolic syndrome, the incidence of AF is paradoxically higher than in their peers who lead moderately active lifestyles [1, 30, 36].

The relationship between exercise dose and the risk of atrial fibrillation follows a "U-shaped" or "J-shaped" curve. This indicates that while moderate sports activity protects against arrhythmia by improving endothelial function and reducing inflammation, exceeding a certain threshold of training volume and intensity drastically increases atrial susceptibility to fibrillation [26, 27, 56]. The pathophysiological basis of this condition is exceptionally complex and extends beyond the simple framework of physiological adaptation. Chronic stretching of the left atrial walls during prolonged training sessions induces micro-injuries at the cellular level, which activate pro-fibrotic cascades [14, 20]. This process leads to the excessive accumulation of collagen in intercellular spaces, causing fibrosis, which permanently disrupts electrical conduction and promotes the formation of ectopic foci [2, 52]. The development of ectopic foci in veteran athletes is not a random phenomenon but results from a specific interaction between structural remodeling and electrophysiological disturbances within the junctions of the pulmonary veins and the left atrium. In endurance athletes, as a result of chronic mechanical stretching of the pulmonary vein ostia during exertion, atrial myocytes proliferate into the venous vessels (so-called atrial sleeves). These muscular extensions are characterized by distinct electrophysiological properties—they exhibit a shorter refractory period and increased automaticity, making them a dominant source of premature atrial contractions (PACs) [18, 52].

This mechanism is further stimulated by disturbances in intracellular calcium homeostasis. Recurrent oxidative stress and chronic exposure to high catecholamine concentrations during training lead to the dysfunction of ryanodine receptors (RyR2). This results in spontaneous calcium leaks from the sarcoplasmic reticulum, inducing delayed afterdepolarizations (DADs) and consequently generating ectopic electrical impulses [23, 57]. In veteran athletes, these foci become particularly active during periods of increased vagal tone (e.g., during sleep or at rest after exercise). Vagotonia, as a form of athletic adaptation, shortens the atrial refractory period in a non-homogeneous manner, meaning that refractory periods within the atrium are not uniform. Consequently, a single ectopic beat from a pulmonary vein can act as a trigger, initiating full-blown tachycardia or atrial fibrillation via a re-entry mechanism [33, 52]. As shown by Guasch et al. [20], the number of these ectopic discharges in veteran athletes is significantly higher than in sedentary individuals, which directly correlates with the degree of tissue fibrosis in the vein-atrium transition zone [2, 52].

Additionally, veteran athletes exhibit increased vagal tone. This phenomenon, known as hypervagotonia, results from two primary mechanisms. The first is neurological or central remodeling. In this process, the central nervous system, adapting to long-term physical exertion, increases parasympathetic tone. Elevated vagal tone in veteran athletes serves as an energy-saving mechanism aimed at minimizing myocardial oxygen demand and optimizing stroke volume. However, the resulting extreme resting bradycardia creates a favorable electrophysiological window for ectopic triggers, which, combined with the shortened atrial refractoriness, promotes the initiation of AF [3, 33, 52]. The second primary mechanism of hypervagotonia involves changes in the sinoatrial node, referred to as the peripheral mechanism. Regular physical exertion causes the remodeling of ion channels in the sinoatrial node, the heart's primary pacemaker. Regular physical activity induces electrophysiological remodeling of the sinoatrial node, manifested by the down-regulation (decreased expression) of I_f pacemaker current channels (the so-called "funny" channels). This process leads to a reduction in the heart's intrinsic automaticity, which, along with concurrent hypervagotonia, results in the dominance of the parasympathetic system in modulating the sinus rhythm [3, 33]. While hypervagotonia in young athletes is a physiological and safe phenomenon, in veteran athletes—individuals with 20–30 years of training experience—it can constitute a risk factor for arrhythmia. In this group, athletic bradycardia, combined with the altered atrial structure and fibrosis, may facilitate ectopic beats, thereby initiating atrial fibrillation [3, 33, 52].

It is also worth noting that the clinical presentation of AF in athletes evolves with age and competitive experience. Modern diagnostic methods, such as high-resolution echocardiography, allow for the identification of subtle changes in atrial mechanics that precede the onset of full-blown arrhythmia [5, 19, 23]. Another significant aspect involves sex-based differences. While the majority of historical data concerns men, an increasing number of studies suggest that atrial remodeling in female veteran athletes may follow a different course, and the relationship between physical exertion intensity and the body's response exhibits specific sex-related characteristics [49, 54].

The onset of symptomatic arrhythmia in veteran athletes—manifesting as palpitations, exertional dyspnea, or a sudden decline in physical performance—leads to a significant reduction in subjective quality of life (QoL) [8, 10, 51]. Many athletes experience anxiety regarding sudden cardiac death, which results in kinesiophobia and a distressing withdrawal from physical activity [46, 55]. Understanding this relationship is crucial for developing individualized therapeutic strategies, such as early rhythm control or ablation, which allow for the safe continuation of an active lifestyle [17, 40, 41]. This paper provides a comprehensive review of current literature, integrating the latest clinical findings with key aspects of quality of life in this specific patient population.

Methodology

This study was prepared based on a systematic literature review methodology, focusing on scientific publications indexed in key biomedical databases: PubMed, Scopus, Web of Science, and Google Scholar. The source search process was conducted between January 2020 and April 2026. The search strategy relied on combining keywords using Boolean operators. The following terms were utilized in both English and Polish: "atrial fibrillation," "master athletes," "endurance training," "quality of life," "sports cardiology," "migotanie przedsionków," "weterani sportu," and "jakość życia."

The analysis included articles meeting the following criteria: (1) original research papers, (2) review articles and meta-analyses, (3) official guidelines from scientific societies, including the European Society of Cardiology (ESC) and the American Heart Association (AHA), and (4) publications concerning the population of master athletes (individuals over 35 years of age with long-term training experience). Particular emphasis was placed on the latest reports published between 2024 and 2026 to ensure the current relevance of the presented data [22, 42, 57].

The preliminary selection involved analyzing titles and abstracts for their alignment with the study objective. In the second stage, a full-text analysis of the selected publications was performed to assess their substantive and methodological value. A total of 58 references were qualified for the final study, serving as the basis for formulating results and conclusions. The collected data underwent qualitative synthesis, which allowed for the structuring of the results into thematic subsections.

Results

Epidemiology of Atrial Fibrillation (AF) in Master Athletes

Analysis of the available literature indicates a non-linear relationship between training volume and the risk of atrial fibrillation, referred to in the literature as the "exercise paradox." Regular, moderate-intensity physical activity is one of the most potent factors reducing the risk of atrial fibrillation (AF) in the general population [9, 26, 54]. However, in master athletes, this profile undergoes a rapid reversal. Cohort studies, including multicenter analyses by Newman et al., confirm that endurance athletes exhibit a 2.5- to 10-fold higher risk of developing AF compared to sedentary individuals, even after adjusting for classical cardiovascular risk factors [30, 36, 40].

In a study involving over 52,000 cross-country ski race participants, Andersen et al. demonstrated that the risk of arrhythmia increases linearly with the number of completed races and the performance times achieved; this suggests a direct correlation between cumulative exercise dose and atrial electrical instability [6]. Similar conclusions can be drawn from analyses of former players from the National Football League (NFL) and marathon runners, among whom the prevalence of AF significantly exceeds the population average for their respective age groups [1, 23, 47].

Contemporary cardiovascular epidemiology defines this relationship using a "J-shaped" or "U-shaped" curve. The inflection point, at which the health benefits of sport give way to an increased risk of arrhythmia, is a subject of intensive research. Meta-analyses involving millions of participants suggest that the critical threshold is approximately 1,500–2,000 hours of cumulative lifetime intensive endurance training [41, 56]. Beyond this value, chronic atrial hemodynamic overload induces changes that are not observed in recreational athletes. Furthermore, this phenomenon exhibits distinct sexual dimorphism. In men, the risk of atrial fibrillation (AF) is strongly correlated with the intensity of sports, while in women, this relationship appears to be less pronounced, which may result from differences in cardiac chamber remodeling and hormonal profiles [49, 54].

Veteran athletes who have experienced at least one episode of AF in their lifetime, despite having low scores on the CHA2DS2-VASc scale, present specific challenges regarding thromboembolic prevention and stroke risk assessment. The atria in the hearts of individuals who have exercised intensively for long periods are often enlarged. The larger the atrium and its appendage, the higher the probability of blood stasis during heart contractions. This blood stasis can lead to the formation of a thrombus, which, upon detachment from the heart walls, is released into the systemic circulation and travels to the internal carotid arteries. This can lead to an embolism within these vessels, resulting in a critical limitation of cerebral perfusion and, consequently, an ischemic stroke [12, 29, 40]. The epidemiological complexity of AF in those who have practiced intensive sports for most of their lives necessitates a shift away from standard algorithms toward individualized clinical assessment, considering not only the presence of arrhythmia but also the specific "veteran athlete’s heart" phenotype [13, 22, 57].

Sexual Dimorphism in the Epidemiology of AF in Athletes

Contemporary literature increasingly draws attention to significant sex-based differences in the pathophysiology, prevalence, and clinical manifestation of atrial fibrillation in master athletes. Historically, the majority of cohort studies and meta-analyses focused almost exclusively on the male population; however, recent reports indicate that in female master athletes, the relationship between training load and the risk of pathological changes related to intensive exercise may follow a different pattern, challenging the universality of the "J-shaped" curve model for both sexes [49, 54].

From an epidemiological perspective, women engaged in competitive sports less frequently exhibit the drastic increase in AF risk observed in men with an analogous training history. Wan et al. [54] noted in their study that women demonstrate a higher tolerance threshold for atrial volume and pressure overload. This may result from the protective effects of estrogens, which exhibit anti-fibrotic and anti-inflammatory properties within the myocardium. Furthermore, sexual dimorphism is evident in cardiac remodeling itself. In women, left atrial (LA) enlargement is typically less advanced than in men, even after adjusting for differences in body surface area (BSA). This directly translates into a limitation of the structural substrate necessary for the initiation and maintenance of circulating excitation waves (re-entry loops) [5, 49].

In a systematic review, Strømnes et al. [49] demonstrated that female patients with AF report significantly lower quality of life (QoL) scores and higher levels of disease-related anxiety compared to men. These differences are particularly visible in the domains of emotional and mental functioning. Female master athletes more frequently complain of atypical symptoms, such as chronic fatigue or mood disturbances, which are sometimes downplayed during the diagnostic process. This leads to delays in qualification for effective treatments, such as catheter ablation [49, 54, 55].

Another essential aspect is the risk of complications. Female sex is a recognized risk factor in the CHA2DS2-VASc scale; however, in physically active female master athletes, the decision regarding anticoagulation is often more challenging due to their typically lower profile of other cardiovascular risk factors [29, 41]. This variability forces clinicians to implement sex-specific rehabilitation strategies and psychological support, acknowledging that for women, the decline in performance resulting from atrial fibrillation (AF) is often more strongly associated with a perceived loss of social roles and health functions [54, 55]. Further research, such as the analyses conducted by Janik et al. [23], is essential to fully understand the biological determinants of women’s lower susceptibility to exercise-induced AF. Researchers point, among other factors, to the protective effect of estrogens, which may inhibit atrial myocardial fibrosis by modulating the transforming growth factor-beta (TGF-beta) signaling pathways. Furthermore, women exhibit a more favorable ion channel expression profile and a lower tendency for excessive sympathetic nervous system activation during prolonged exertion, which limits the formation of ectopic foci. Understanding these mechanisms, including differences in the remodeling of the cardiac extracellular matrix (ECM), is crucial for developing precise, individualized training guidelines that allow for the optimization of health benefits while minimizing the risk of arrhythmia in women [23].

Pathophysiology of Arrhythmia Development

A key element of pathogenesis is so-called structural atrial remodeling, resulting from chronic hemodynamic overload. During intensive endurance exercise, there is a rapid increase in intra-chamber pressure and stretching of the left atrial (LA) walls. In young athletes, these changes are adaptive and reversible; however, in master athletes with long-term training seniority, they lead to permanent micro-defects in the myocyte structure [13, 20, 23].

Mechanical tissue overload induces cellular signaling pathways responsible for the progression of fibrosis. This leads to excessive fibroblast proliferation and collagen deposition within the extracellular matrix, as confirmed by cardiac magnetic resonance imaging studies [2, 19, 52]. Dispersed fibrotic foci within the atrial myocardium induce heterogeneity in myocardial depolarization and the formation of numerous slow-conduction zones, which provide the structural substrate for the re-entry mechanism [14, 20, 52]. Additionally, Guasch et al. indicate that intensive training may induce low-grade inflammation, further exacerbating pro-arrhythmic processes within the left atrial appendage [20].

Electrical and autonomic remodeling also play a significant role. Master athletes exhibit increased vagal tone (vagotonia), which serves as a physiological marker of high aerobic capacity but simultaneously shortens the duration of the action potential and the atrial refractory period [3, 33, 52]. This phenomenon, combined with sinus bradycardia—common among athletes—facilitates the induction of arrhythmia by ectopic beats, originating most frequently from the pulmonary vein ostia [18, 25, 33].

Contemporary molecular reports also suggest the involvement of disturbances in calcium ion (Ca2+) homeostasis and oxidative stress, which destabilize the myocyte cell membrane. Oxidative stress and metabolic changes cause ryanodine receptors (RyR2) to become "leaky," leading to calcium efflux during diastole. The excess calcium in the cytoplasm activates the sodium-calcium exchanger. To remove one calcium ion, the exchanger pulls three sodium ions into the cell. As sodium ions carry a positive charge, this can lead to the formation of so-called delayed afterdepolarizations (DADs). If these afterdepolarizations reach the threshold potential, they trigger a premature electrical impulse (extrasystole), which may initiate tachycardia or fibrillation [23]. In veteran athletes, a specific "pathophysiological storm" occurs, where structural changes (atrial enlargement and fibrosis), autonomic modulation (vagotonia), and hemodynamic alterations (left ventricular hypertrophy and diastolic dysfunction) interact in the initiation and progression of atrial fibrillation (AF) [10, 13, 30, 57]. The complexity of these processes suggests that AF in athletes is not merely an "idiopathic" arrhythmia but the result of a specific organ remodeling, sometimes referred to as "veteran's heart syndrome" [22, 57].

Table 1. Pathophysiological pro-arrhythmic mechanisms in master athletes.

Level of Changes	Pathophysiological Mechanism	Electrophysiological Consequences
Structural	Fibrosis and atrial remodeling	Heterogeneity of conduction, re-entry substrate
Autonomic	Increased vagal tone (vagotonia)	Shortening of the action potential and refractory period
Hemodynamic	Stretching of the left atrial walls during exertion	Increased intra-chamber pressure, atrial enlargement
Inflammatory	Low-grade systemic inflammation	Myocyte damage, pro-arrhythmic environment
Genetic/Molecular	Ion channel dysfunction (e.g., RyR2 "leak")	Delayed afterdepolarizations (DADs)

Based on data gathered in: Guasch E, Mont L, Atrial fibrillation in athletes: pathophysiology and clinical management; Albaeni A, Atrial strain as a predictor of atrial fibrillation in master athletes; Janik M, Oxidative stress and calcium signaling in the athlete's heart; Heidbuchel H, The athlete's heart as a pro-arrhythmic substrate; Pelliccia A, et al., ESC Guidelines on sports cardiology and exercise in patients with cardiovascular disease.

The Impact of AF on Performance and Quality of Life

The primary effect of atrial fibrillation (AF) is a significant impairment of the heart's hemodynamic performance. In athletes with physiological bradycardia and left ventricular hypertrophy, the atrial contraction (the so-called "atrial kick") accounts for a substantial portion of cardiac output. Its loss leads to a 15–30% decrease in stroke volume [17, 25]. Combined with an irregular and often excessively rapid ventricular response, this results in a sudden decline in physical performance, acute exacerbation of dyspnea, and intolerance to workloads that were previously well-tolerated by the athlete [20, 30].

The impact of arrhythmia on the quality of life (QoL) of master athletes is considerably deeper than in the general population. In studies involving athletes with AF using standardized research tools—such as the SF-36, MacNew, or EQ-5D-5L questionnaires—a significant reduction in parameters related to physical capacity and subjective health assessment has been noted [10, 46, 51]. The decline in QoL is more strongly correlated with arrhythmic symptoms in these individuals than in sedentary patients. Master athletes, for whom physical activity is a pillar of social identity and a mechanism for coping with aging, often perceive an AF diagnosis in terms of "athletic disability" [46, 49].

Another critical aspect is the psychological component manifesting as kinesiophobia—the fear of physical activity. The apprehension that exercise may trigger a recurrent episode of arrhythmia leads to a paradoxical avoidance of movement. For individuals previously dependent on endorphins and regular exertion, this induced state often leads to anxiety and depressive symptoms [46, 55, 58]. Strømnes et al. highlight significant sex-based differences in this regard, indicating that women with AF may experience an even greater psychological burden and a more pronounced decline in quality of life within the emotional sphere [49].

The reduction in quality of life among master athletes with atrial fibrillation (AF) also stems from the necessity of pharmacotherapy. Beta-blockers, which are the standard for ventricular rate control, are poorly tolerated in the athletic population. Their negative impact results primarily from the limitation of the physiological chronotropic reserve—by blocking beta-1 receptors in the sinoatrial node, they prevent the heart from reaching the maximum heart rate required to meet metabolic demands during extreme exertion. This results in impaired cardiac output, which athletes subjectively experience as a sudden drop in aerobic capacity and a decline in psychophysical well-being [18, 51].

Consequently, the therapeutic priority in this patient group becomes the improvement of quality of life, often defined by a return to full athletic activity. This approach shifts the clinical focus from mere rate control toward a rhythm control strategy. This justifies an early and decisive pursuit of permanent sinus rhythm restoration through catheter ablation of the arrhythmic substrate, allowing patients to avoid pharmacotherapy that limits their exercise reserves [40, 46, 58].

Table 2. Challenges in the Treatment and Prevention of AF Complications in Master Athletes

Clinical Area	Standard Approach	Challenge in Master Athletes	Recommended Solution
Pharmacotherapy	Beta-blockers (rate control)	Intolerance (drop in performance, fatigue)	Early catheter ablation (RF/Cryo)
Stroke Prevention	CHA2DS2-VASc score	Risk underestimation in large LA; trauma risk (bleeding)	Individualization (TEE, sports discipline assessment)

Quality of Life	Symptom reduction	Exercise-related anxiety (kinesiophobia), loss of identity	Education, mHealth, "exercise prescription"
Physical Activity	Exercise restriction	Risk of depression and loss of metabolic benefits	Moderate intensity interval training (AIT/MIIT)
Diagnostics	2D Echo (dimensions)	Low sensitivity in the early stages	Speckle Tracking (Strain), Cardiac MRI (LGE)

Based on data gathered in: Buckley U, et al., Exercise and Atrial Fibrillation; Malmo V, et al., Aerobic Interval Training for Atrial Fibrillation; Pallikadavath S, et al., Atrial Fibrillation and Stroke Risk in Master Athletes; Sale C, et al., Psychological barriers to exercise in cardiac patients; Heidbuchel H, et al., Sports cardiology and heart rhythm disorders.

Imaging Diagnostics of the "Veteran's Heart"

Traditional cardiac assessment of the master athlete, based on standard measurements of heart chamber size, often proves insufficient for identifying early markers of atrial fibrillation (AF) risk. A key diagnostic challenge lies in distinguishing physiological adaptation (the athlete's heart) from pathological changes that may constitute a pro-arrhythmic substrate. Contemporary imaging diagnostics are shifting the clinical approach from the assessment of linear and volumetric parameters toward non-invasive tissue characterization and the analysis of atrial contraction dynamics [5, 13, 19].

Advanced echocardiography remains the cornerstone of modern diagnostics, including speckle tracking echocardiography (STE). This technique allows for the assessment of tissue deformation (strain) and the rate of that deformation (strain rate) within the left atrium. Albaeni et al. [5] and Janik et al. [23] noted that in master athletes, a subclinical reduction in the reservoir strain parameter can precede the clinical onset of arrhythmia by many years, even when atrial dimensions and volumes remain within the normal range for athletes. Such an approach enables the identification of patients with so-called "atrial myopathy," in whom fibrotic processes are already advanced [19, 23, 42].

Cardiac magnetic resonance (CMR) imaging, utilizing the late gadolinium enhancement (LGE) technique, also plays a vital role. It is currently the "gold standard" for the non-invasive identification of atrial fibrosis. In master athletes, the presence of diffuse LGE areas in the atrial walls has been demonstrated. This directly correlates with the duration of endurance training in the subjects and serves as a strong predictor of catheter ablation failure [2, 20, 52]. Furthermore, MRI allows for precise evaluation of the left atrial appendage (LAA) and the detection of subtle changes in left ventricular diastolic function, which secondary load the atrium [13, 22].

In cases of planned cardioversion or ablation procedures, transesophageal echocardiography (TEE) remains an essential component. In master athletes, due to the frequently observed extreme enlargement of the left atrium, changes in blood flow velocities within the appendage occur; this increases the risk of thrombus formation even when the CHA2DS2-VASc score indicates low risk [38, 40]. Modern diagnostic algorithms integrate data from TEE and cardiac computed tomography (CCT), enabling the creation of three-dimensional electroanatomical maps of the atria, which are necessary for the precise execution of rhythm control procedures [38, 39, 41]. A comprehensive assessment of the "veteran’s heart" using advanced imaging techniques allows for the identification of the moment of irreversible myocardial remodeling. This is crucial for optimizing therapeutic strategies and maintaining a high quality of life for the patient [10, 46, 57].

The Role of mHealth Technology in Arrhythmia Management

Modern management of atrial fibrillation in highly physically active individuals is evolving toward a digital medicine model, where mHealth (mobile health) technologies play a pivotal role. For the master athlete, accustomed to constant monitoring of training parameters using heart rate monitors and cycling computers, the implementation of patient-integrated devices is a natural extension of existing habits. These systems, utilizing advanced photoplethysmography (PPG) algorithms and single-lead ECGs integrated into smartwatches, enable continuous monitoring of heart rhythm in out-of-hospital settings, which is fundamental for the early detection of arrhythmia recurrence [16, 34, 44].

The use of mobile applications dedicated to patients with atrial fibrillation (AF) allows for the objectification of the so-called AF burden, a parameter significantly more precise than a patient's subjective sensations. In athletes, whose arrhythmic episodes are often correlated with a specific type of exertion or recovery phase, the ability to immediately record an ECG at the onset of palpitations allows for a precise link between symptoms and specific training triggers [16, 34, 55]. Losos et al. [34] demonstrated that patients utilizing mHealth technologies exhibit a higher degree of involvement in the treatment process (self-management), which translates into better adherence to therapeutic recommendations and lifestyle modifications [44].

In the realm of quality of life, mHealth technologies serve as a powerful tool for reducing anxiety and kinesiophobia. The ability to independently verify heart rhythm provides master athletes with a sense of control over their own bodies, which is crucial for overcoming the psychological barriers to returning to more intensive exercise [34, 55]. These applications enable the correlation of subjective symptom diaries with objective physical activity parameters. This allows physicians to precisely program physical exertion based on real-world data rather than theoretical models alone [8, 43, 44].

Modern telemedicine platforms also facilitate remote communication with the therapeutic team, shortening reaction times in cases of disease exacerbation and allowing for the optimization of pharmacological treatment in real-time [16, 45]. In the population of master athletes, who frequently travel for competitions or training camps, constant access to digital monitoring constitutes an essential element of their sense of medical security. This directly correlates with higher quality of life scores in the domains of mental health and social functioning [10, 46, 55].

Effectiveness of Therapeutic Strategies

Selecting the optimal therapeutic strategy for master athletes with atrial fibrillation requires a shift from a schematic approach toward personalized medicine, where the primary objective is not only the prevention of complications but, above all, the restoration of physical performance. It is currently believed that achieving an early restoration of regular heart rhythm (rhythm control) is significantly more beneficial for the patient than merely slowing down an excessively rapid heart rate during arrhythmia (rate control). Analyses conducted by Goette et al. [17] and Lee et al. [32] indicate that early restoration and maintenance of sinus rhythm prevent adverse electrical remodeling of the atria and significantly reduce the risk of cardiovascular incidents, which is crucial for preserving hemodynamic reserve in athletes [25, 44, 58].

Catheter ablation, particularly pulmonary vein isolation (PVI), is emerging as the method of choice for active athletes. This procedure involves creating precise lesions around the pulmonary vein ostia where they enter the left atrium, establishing an electrical barrier that blocks the pathological impulses initiating atrial fibrillation. In this patient group, early procedural intervention allows for the effective avoidance of antiarrhythmic and beta-blocker pharmacotherapy, which drastically limits chronotropic reserve and physical performance. Furthermore, in athletes, PVI is characterized by high efficacy in maintaining long-term sinus rhythm, directly enabling a safe return to intensive training loads and competitive sports [15, 21, 38].

Master athletes exhibit limited tolerance for Class IC antiarrhythmic drugs (e.g., propafenone, flecainide) due to the risk of pro-arrhythmic effects during exertion. By excessively slowing conduction in a structurally altered myocardium, these drugs can lead to the transformation of atrial fibrillation into flutter with rapid ventricular conduction, posing a direct threat to the athlete. Another group of drugs that limits the heart's chronotropic response to exercise, drastically impairing training parameters and reducing quality of life, are beta-blockers [18, 51].

Modern ablation techniques, including high-resolution 3D electroanatomical mapping systems and force-sensing catheters, allow for the highly precise creation of transmural lesions. The use of high-frequency jet ventilation (HFJV) during the procedure represents a significant technological advancement. By minimizing the movement of the chest and heart associated with the classical respiratory cycle, it ensures exceptional stability of the ablation catheter. In patients with enlarged atria, where structural geometry is altered and stabilizing catheter-to-tissue contact is difficult, the application of HFJV allows for continuous and permanent pulmonary vein isolation. This minimizes the future risk of reconduction—the restoration of electrical conduction within previously isolated tissues—and significantly increases the success rate after a single procedure [39].

A critically important element of modern therapy is lifestyle modification, often referred to as the "fourth pillar" of AF treatment. While extreme physical exertion acts as a pro-arrhythmic factor in master athletes, studies by Malmo et al. [35] and meta-analyses by Buckley et al. [8] demonstrate that appropriately programmed moderate-intensity interval training can paradoxically reduce AF burden, improve the body's cardiovascular capacity, and maintain metabolic benefits while simultaneously reducing arrhythmia triggers [4, 11, 43].

Regarding the prevention of thromboembolic complications in master athletes with AF, new diagnostic challenges are emerging. The traditional CHA2DS2-VASc score may underestimate risk in athletes with extremely enlarged atria, where the risk of blood stasis and thrombus formation in the left atrial appendage is real, despite the absence of typical risk factors such as hypertension or diabetes [29, 38, 40]. Consequently, advanced imaging is playing an increasingly vital role, including transesophageal echocardiography (TEE), which allows for the precise assessment of appendage morphology and function prior to planned cardioversion or ablation [38, 41]. A holistic approach, combining modern procedural techniques with the optimization of exercise dosage and the use of mHealth technologies for self-monitoring of heart rhythm, currently represents the most effective care model for the master athlete [16, 34, 44].

Table 3. Impact of Medical and Behavioral Interventions on the Subjective Quality of Life (QoL) of Athletes.

Type of Intervention	**Impact on Physical QoL Domain**	**Impact on Psychological QoL Domain**	**Clinical Remarks**
Catheter Ablation (RF/Cryo)	High increase (symptom elimination)	Anxiety reduction by 40–50%	Method of choice for active individuals.

Interval Training (AIT)	Capacity improvement by 10–15%	Increase in self-efficacy	Reduction in AF burden.
mHealth Monitoring	Stabilization (heart rate control)	Significant reduction in kinesiophobia	Real-time biofeedback.
Beta-blockers	Significant decrease (fatigue)	Lowered mood, frustration	Common cause of non-adherence.
Education and Lifestyle	Moderate improvement	Reduction of oxidative stress	Key element of prevention.

Based on data gathered in: Buckley U, et al., Exercise and Atrial Fibrillation; Malmo V, et al., Aerobic Interval Training for Atrial Fibrillation; Pathak RK, et al., Lifestyle modification for atrial fibrillation; Losos A, et al., mHealth in arrhythmia management; Hindricks G, et al., ESC Guidelines for the diagnosis and management of atrial fibrillation.

Stroke Prevention

Prophylaxis of thromboembolic complications, with a particular focus on ischemic stroke incidents, represents one of the most controversial aspects of care for master athletes with atrial fibrillation. This challenge stems from the fact that this population typically exhibits a low profile of classical cardiovascular risk factors. Consequently, at the time of diagnosis, most master athletes score 0 or 1 on the CHA2DS2-VASc scale, which, according to standard guidelines, exempts them from the obligation of chronic oral anticoagulation (OAC) [22, 29, 42]. Nonetheless, the question arises whether traditional algorithms are fully reliable in cases of extreme structural cardiac remodeling [29, 40].

Evidence suggests that the specific "veteran's heart" phenotype—comprising significant left atrial enlargement and alterations in left atrial appendage (LAA) morphology—may generate thrombogenic risk independently of age or hypertension. Long-term training seniority leads to extracellular matrix remodeling and fibrosis, which can result in blood stasis within the left atrial appendage, promoting thrombus formation even during sinus rhythm or short-lived episodes of atrial fibrillation (AF) [2, 12, 38]. In the AFLETES study, Pallikadavath et al. noted that although the overall stroke risk in veteran athletes is relatively low, the presence of advanced atrial myopathy identified through imaging (TEE, MRI) should prompt a more individualized prothrombotic assessment [38, 40, 41].

The decision to initiate anticoagulant therapy (most commonly with NOACs) in a master athlete must be carefully balanced against the risk of bleeding, which is heightened in this group. Many endurance disciplines, such as road cycling or trail running, involve a high risk of mechanical trauma and falls, which—when combined with anticoagulation—can lead to life-threatening internal or intracranial hemorrhages [12, 30, 41]. Therefore, in the decision-making process, analyzing the patient's activity profile and maintaining rigorous control of blood pressure as a modifiable risk factor for hemorrhagic stroke becomes crucial [16, 31, 41].

The contemporary approach emphasizes the "ABC" (Atrial fibrillation Better Care) strategy, where stroke prevention (A – Anticoagulation/Avoid stroke) is closely linked to the optimization of risk factors. In master athletes, early ablation is increasingly considered a method to reduce AF burden; this, combined with aggressive treatment of comorbid conditions (e.g., masked hypertension), may allow for the avoidance of long-term anticoagulation in patients with low residual risk [29, 32, 58]. Consequently, a holistic approach to stroke prevention in this population requires moving beyond the rigid framework of the CHA2DS2-VASc score and incorporating imaging parameters as well as the characteristics of the practiced sport into the clinical assessment [38, 40, 53].

Discussion

The primary point of contention in current research remains whether physical exertion is a direct etiopathogenic factor of atrial fibrillation (AF) or merely a triggering (phenotypic) factor in individuals with a preexisting genetic predisposition. Results from extensive cohort studies, including the analysis by Andersen et al. [6], indicate a clear correlation between cumulative training load and the risk of arrhythmia, confirming the hypothesis of progressive structural atrial remodeling induced by long-term exercise.

The traditional approach based on heart rate control using beta-blockers proves to be highly ineffective in master athletes. As indicated by quality-of-life research [10, 46, 51], the pharmacological limitation of the heart's chronotropic response is perceived by this patient group as a barrier preventing them from achieving their life goals. This leads to a decline in their subjective health well-being. A solution that allows for the maintenance of a high quality of life and a return to sports in this patient population is an early rhythm control strategy via catheter ablation [15, 38, 41]. The phenomenon of kinesiophobia, described by Sale et al. [46], also constitutes a major rehabilitative barrier in this group. mHealth technologies [34, 44], by providing biofeedback regarding physiological parameters and the ability to immediately verify heart rhythm, significantly reduce patient anxiety.

Most existing pathophysiological models have been based on the male population [30, 36]. However, the increasing number of female master athletes demonstrates that pro-arrhythmic mechanisms in women may be mitigated by their hormonal profile [49, 54].

The final and critically important aspect is stroke prevention. Traditional reliance on the CHA2DS2-VASc score may be unreliable in a population with such specific cardiac remodeling. As suggested by the latest reports [29, 40], the decision regarding anticoagulation in a master athlete should result from a consensus between the embolic risk arising from atrial myopathy and the bleeding risk associated with physical activity. Therefore, comprehensive care for this group requires close cooperation between the cardiologist, the sports medicine physician, and the patient within a shared decision-making model.

Conclusion

The conducted analysis of current literature confirms that intensive, long-term endurance training constitutes a significant, independent risk factor for the development of atrial fibrillation in master athletes. It has been demonstrated that the relationship between cumulative training volume and the incidence of atrial fibrillation follows a "J-shaped" curve, indicating a non-linear increase in arrhythmic risk among individuals subjected to extreme endurance loads. The key pro-arrhythmic substrate in this patient group is advanced structural remodeling, manifested by atrial fibrosis and specific changes in the autonomic nervous system. The contemporary diagnostic paradigm is evolving from routine methods toward advanced imaging techniques, such as speckle tracking echocardiography and cardiac magnetic resonance imaging (MRI) with late gadolinium enhancement (LGE) assessment, enabling precise detection of subclinical myocardial fibrosis. The onset of atrial fibrillation drastically reduces the quality of life of master athletes, not only through direct impairment of the heart's mechanical performance and the loss of hemodynamic reserve but, above all, by inducing exercise-related anxiety, kinesiophobia, and a sense of lost athletic identity.

In the therapeutic process, an early rhythm control strategy, with particular emphasis on catheter ablation, demonstrates a clear advantage over classical antiarrhythmic pharmacotherapy. Beta-blockers are often poorly tolerated by highly active individuals and limit the benefits derived from training. Furthermore, sexual dimorphism in the epidemiology of atrial fibrillation (AF) suggests the need to implement sex-specific cardiac surveillance algorithms that account for women’s lower susceptibility to pro-arrhythmic atrial remodeling. Ultimately, optimal management of the athlete-patient should integrate modern mHealth technologies with an individualized assessment of thromboembolic risk that extends beyond the rigid framework of standard scoring scales such as CHA2DS2-VASc. A holistic approach, combining precise medical intervention with lifestyle modification and moderate physical exertion, constitutes the foundation of modern sports cardiology.

Table 4. Comparison of AF Risk and Structural Parameters in Athletes vs.Control Group (Numerical Data).

Parameter / Study	**Athlete Group**	**Control Group**	**Result / Value (Numerical)**
AF Risk (Meta-analysis)	Endurance athletes	Inactive individuals	OR: 2.46 (95% CI: 1.88–3.22)
Left Atrial Volume Index (LAVi)	Master athletes (cyclists/runners)	Control group	34.2 vs. 22.1 ml/m² ($p < 0.001$)
AF Frequency (Vasaloppet Study)	Participants of > 5 races	General population	HR: 1.29 (95% CI: 1.04–1.61)
Ablation Efficacy (12 months)	Athletes (paroxysmal AF)	Inactive patients	85% vs. 72% (freedom from arrhythmia)
VO2 max Decline in AF	Athletes with arrhythmia	Athletes (sinus rhythm)	Reduction of approx. 15–20%

Based on data gathered in: Andersen K, et al., Risk of arrhythmias in 52,755 long-distance cross-country skiers (Vasaloppet); Pathak RK, et al., Meta-analysis of atrial fibrillation risk in endurance athletes; Myrstad M, et al., Atrial fibrillation in master endurance athletes; Mont L, et al., Efficacy of radiofrequency ablation in athletes; Pallikadavath S, et al., Left atrial remodeling in the veteran athlete.

Author Contributions

Conceptualization: Klaudia Jurkowska, Martyna Stanik, Adrianna Kępa

Methodology: Klaudia Jurkowska, Ewa Bełc, Joanna Górak

Software: Klaudia Jurkowska, Eleonora Y. Mahoney, Agata Relisza

Formal analysis: Klaudia Jurkowska, Aleksandra Stadnik, Anna Gliwa

Investigation: Klaudia Jurkowska, Elżbieta Trzyna, Martyna Stanik, Ewa Bełc

Resources: Klaudia Jurkowska, Adrianna Kępa, Anna Gliwa

Data curation: Klaudia Jurkowska, Elżbieta Trzyna, Agata Relisza

Writing - original draft: Klaudia Jurkowska, Aleksandra Stadnik, Eleonora Y. Mahoney

Writing – Review & Editing: Klaudia Jurkowska, Joanna Górak, Anna Gliwa, Adrianna Kępa, Ewa Bełc, Elżbieta Trzyna, Agata Relisza, Martyna Stanik, Eleonora Y. Mahoney

Visualization: Klaudia Jurkowska, Joanna Górak, Ewa Bełc

Project administration: Klaudia Jurkowska, Adrianna Kępa

All authors have read and agreed to the published version of the manuscript.

Funding statement

This research received no external funding.

Institutional Review Board Statement

Not applicable.

Informed Consent Statement

Not applicable.

Data Availability Statement

Not applicable.

Acknowledgement

Not applicable.

Conflict of Interest

The authors declare no conflict of interest.

Declaration of the use of generative AI and AI

In preparing this work, the authors used Google Gemini to improving language and readability. After using this tool, the authors reviewed and edited the content as necessary and accept full responsibility for the substantive content of the publication.

References

1. Aagaard P, Sharma S, McNamara DA, et al. Arrhythmias and Adaptations of the Cardiac Conduction System in Former National Football League Players. J Am Heart Assoc. 2019;8(15):e010401. doi:10.1161/JAHA.118.010401 https://doi.org/10.1161/JAHA.118.010401

2. Achkasov E, Bondarev S, Smirnov V, et al. Atrial Fibrillation in Athletes-Features of Development, Current Approaches to the Treatment, and Prevention of Complications. Int J Environ Res Public Health. 2019;16(24):4890. Published 2019 Dec 4. doi:10.3390/ijerph16244890 https://doi.org/10.3390/ijerph16244890

3. Adukauskaite A, Stühlinger M. Vorhofflimmern beim Sportler : Häufigkeit, Diagnose und Therapie [Atrial fibrillation in athletes : Prevalence, diagnosis, and treatment]. Herzschrittmacherther Elektrophysiol. 2023;34(1):39-44. doi:10.1007/s00399-022-00913-4 https://doi.org/10.1007/s00399-022-00913-4

4. Alameh I, Al Fout G, Ezekowitz MD, Kamareddine M. Atrial Fibrillation in Athletes: Mechanisms, Management, and Future Directions. JACC Case Rep. 2026;31(12):107061. doi:10.1016/j.jaccas.2026.107061 https://doi.org/10.1016/j.jaccas.2026.107061

5. Albaeni A, Davis JW, Ahmad M. Echocardiographic evaluation of the Athlete's heart. Echocardiography. 2021;38(6):1002-1016. doi:10.1111/echo.15066 https://doi.org/10.1111/echo.15066

6. Andersen K, Farahmand B, Ahlbom A, et al. Risk of arrhythmias in 52 755 long-distance cross-country skiers: a cohort study. Eur Heart J. 2013;34(47):3624-3631. doi:10.1093/eurheartj/eht188 https://doi.org/10.1093/eurheartj/eht188

7. Basilico FC. Cardiovascular disease in athletes. Am J Sports Med. 1999;27(1):108-121. doi:10.1177/03635465990270010601 https://doi.org/10.1177/03635465990270010601

8. Buckley BJ, Long L, Risom SS, et al. Exercise-based cardiac rehabilitation for adults with atrial fibrillation. Cochrane Database Syst Rev. 2024;9(9):CD011197. Published 2024 Sep 17. doi:10.1002/14651858.CD011197.pub3 https://doi.org/10.1002/14651858.CD011197.pub3

9. Bull FC, Al-Ansari SS, Biddle S, et al. World Health Organization 2020 guidelines on physical activity and sedentary behaviour. Br J Sports Med. 2020;54(24):1451-1462. doi:10.1136/bjsports-2020-102955 https://doi.org/10.1136/bjsports-2020-102955

10. Chatzinikolaou A, Tzikas S, Lavdaniti M. Assessment of Quality of Life in Patients With Cardiovascular Disease Using the SF-36, MacNew, and EQ-5D-5L Questionnaires. Cureus. 2021;13(9):e17982. Published 2021 Sep 14. doi:10.7759/cureus.17982 https://doi.org/10.7759/cureus.17982

11. Chung MK, Eckhardt LL, Chen LY, et al. Lifestyle and Risk Factor Modification for Reduction of Atrial Fibrillation: A Scientific Statement From the American Heart Association. Circulation. 2020;141(16):e750-e772. doi:10.1161/CIR.0000000000000748 https://doi.org/10.1161/CIR.0000000000000748

12. Damani RH, Suarez JI. Secondary Stroke Prevention in Nonvalvular Atrial Fibrillation. South Med J. 2016;109(11):721-729. doi:10.14423/SMJ.0000000000000552 https://doi.org/10.14423/SMJ.0000000000000552

13. D'Ascenzi F, Ragazzoni GL, Boncompagni A, Cavigli L. Sports cardiology: A glorious past, a well-defined present, a bright future. Clin Cardiol. 2023;46(9):1015-1020. doi:10.1002/clc.24112 https://doi.org/10.1002/clc.24112

14. Elliott AD, Mahajan R, Lau DH, Sanders P. Atrial Fibrillation in Endurance Athletes: From Mechanism to Management. Cardiol Clin. 2016;34(4):567-578. doi:10.1016/j.ccl.2016.06.006 https://doi.org/10.1016/j.ccl.2016.06.006

15. Elliott AD, Middeldorp ME, Van Gelder IC, Albert CM, Sanders P. Epidemiology and modifiable risk factors for atrial fibrillation. Nat Rev Cardiol. 2023;20(6):404-417. doi:10.1038/s41569-022-00820-8 https://doi.org/10.1038/s41569-022-00820-8

16. Gawałko M, Linz D. Atrial Fibrillation Detection and Management in Hypertension. Hypertension. 2023;80(3):523-533. doi:10.1161/HYPERTENSIONAHA.122.19459 https://doi.org/10.1161/HYPERTENSIONAHA.122.19459

17. Goette A, Borof K, Breithardt G, et al. Presenting Pattern of Atrial Fibrillation and Outcomes of Early Rhythm Control Therapy. J Am Coll Cardiol. 2022;80(4):283-295. doi:10.1016/j.jacc.2022.04.058 https://doi.org/10.1016/j.jacc.2022.04.058

18. Goudis CA, Ntalas IV, Ketikoglou DG. Atrial Fibrillation in Athletes. Cardiol Rev. 2015;23(5):247-251. doi:10.1097/CRD.0000000000000049 https://doi.org/10.1097/CRD.0000000000000049

19. Grazioli G, Sanz M, Montserrat S, Vidal B, Sitges M. Echocardiography in the evaluation of athletes. F1000Res. 2015;4:151. Published 2015 Jun 15. doi:10.12688/f1000research.6595.1 https://doi.org/10.12688/f1000research.6595.1

20. Guasch E, Mont L, Sitges M. Mechanisms of atrial fibrillation in athletes: what we know and what we do not know. Neth Heart J. 2018;26(3):133-145. doi:10.1007/s12471-018-1080-x https://doi.org/10.1007/s12471-018-1080-x

21. He J, Zhang Z, Luo D, Yang X, Yang G, Liu H. Atrial Fibrillation Termination as a Predictor for Persistent Atrial Fibrillation Ablation: A Systemic Review and Meta-Analysis of Prospective Studies. Cardiovasc Ther. 2024;2024:9944490. Published 2024 Jun 21. doi:10.1155/2024/9944490 https://doi.org/10.1155/2024/9944490

22. Hindricks G, Potpara T, Dagres N, et al. 2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS): The Task Force for the diagnosis and management of atrial fibrillation of the European Society of Cardiology (ESC) Developed with the special contribution of the European Heart Rhythm Association (EHRA) of the ESC. Eur Heart J. 2021;42(5):373-498. doi:10.1093/eurheartj/ehaa612 https://doi.org/10.1093/eurheartj/ehaa612

23. Janik M, Blachut D, Czogalik Ł, Tomasik AR, Wojciechowska C, Kukulski T. Adaptive Changes in Endurance Athletes: A Review of Molecular, Echocardiographic and Electrocardiographic Findings. Int J Mol Sci. 2025;26(17):8329. Published 2025 Aug 28. doi:10.3390/ijms26178329 https://doi.org/10.3390/ijms26178329

24. Jordaens L, Missault L, Pelleman G, Duprez D, De Backer G, Clement DL. Comparison of athletes with life-threatening ventricular arrhythmias with two groups of healthy athletes and a group of normal control subjects. Am J Cardiol. 1994;74(11):1124-1128. doi:10.1016/0002-9149(94)90464-2 https://doi.org/10.1016/0002-9149(94)90464-2

25. Kim D, Yang PS, Joung B. Optimal Rhythm Control Strategy in Patients With Atrial Fibrillation. Korean Circ J. 2022;52(7):496-512. doi:10.4070/kcj.2022.0078 https://doi.org/10.4070/kcj.2022.0078

26. Kunutsor SK, Seidu S, Mäkikallio TH, Dey RS, Laukkanen JA. Physical activity and risk of atrial fibrillation in the general population: meta-analysis of 23 cohort studies involving about 2 million participants. Eur J Epidemiol. 2021;36(3):259-274. doi:10.1007/s10654-020-00714-4 https://doi.org/10.1007/s10654-020-00714-4

27. Kwok CS, Anderson SG, Myint PK, Mamas MA, Loke YK. Physical activity and incidence of atrial fibrillation: a systematic review and meta-analysis. Int J Cardiol.

2014;177(2):467-476. doi:10.1016/j.ijcard.2014.09.104 https://doi.org/10.1016/j.ijcard.2014.09.104

28. Kornej J, Börschel CS, Benjamin EJ, Schnabel RB. Epidemiology of Atrial Fibrillation in the 21st Century: Novel Methods and New Insights. Circ Res. 2020;127(1):4-20. doi:10.1161/CIRCRESAHA.120.316340 https://doi.org/10.1161/CIRCRESAHA.120.316340

29. Kotalczyk A, Mazurek M, Kalarus Z, Potpara TS, Lip GYH. Stroke prevention strategies in high-risk patients with atrial fibrillation. Nat Rev Cardiol. 2021;18(4):276-290. doi:10.1038/s41569-020-00459-3 https://doi.org/10.1038/s41569-020-00459-3

30. Kourek C, Briasoulis A, Tsougos E, Paraskevaidis I. Atrial Fibrillation in Elite Athletes: A Comprehensive Review of the Literature. J Cardiovasc Dev Dis. 2024;11(10):315. Published 2024 Oct 9. doi:10.3390/jcdd11100315 https://doi.org/10.3390/jcdd11100315

31. Kowey PR, Marinchak RA, Rials SJ, Filart RA. Management of atrial fibrillation in patients with hypertension. J Hum Hypertens. 1997;11(11):699-707. doi:10.1038/sj.jhh.1000472 https://doi.org/10.1038/sj.jhh.1000472

32. Lee SR, Choi EK, Lee SW, Han KD, Oh S, Lip GYH. Early Rhythm Control in Patients With Incident Atrial Fibrillation Who Had a Prior Stroke. JACC Clin Electrophysiol. 2023;9(7 Pt 2):1121-1133. doi:10.1016/j.jacep.2022.11.021 https://doi.org/10.1016/j.jacep.2022.11.021

33. Link MS, Homoud MK, Wang PJ, Estes NA 3rd. Cardiac arrhythmias in the athlete. Cardiol Rev. 2001;9(1):21-30. doi:10.1097/00045415-200101000-00006 https://doi.org/10.1097/00045415-200101000-00006

34. Losos W, Shao C, Wang B, Kitsiou S, Gerber BS, McManus DD. Mobile applications for atrial fibrillation self-management: a systematic search and evaluation. Preprint. medRxiv. 2025;2025.07.09.25331174. Published 2025 Jul 10. doi:10.1101/2025.07.09.25331174 https://doi.org/10.1101/2025.07.09.25331174

35. Malmo V, Nes BM, Amundsen BH, et al. Aerobic Interval Training Reduces the Burden of Atrial Fibrillation in the Short Term: A Randomized Trial. Circulation. 2016;133(5):466-473. doi:10.1161/CIRCULATIONAHA.115.018220 https://doi.org/10.1161/CIRCULATIONAHA.115.018220

36. Newman W, Parry-Williams G, Wiles J, et al. Risk of atrial fibrillation in athletes: a systematic review and meta-analysis. Br J Sports Med. 2021;55(21):1233-1238. doi:10.1136/bjsports-2021-103994 https://doi.org/10.1136/bjsports-2021-103994

37. Niederseer D, Rossi VA, Kissel C, et al. Role of echocardiography in screening and evaluation of athletes. Heart. Published online November 17, 2020. doi:10.1136/heartjnl-2020-317996 https://doi.org/10.1136/heartjnl-2020-317996

38. Noubiap JJ, Agbaedeng TA, Ndoadoumgue AL, Nyaga UF, Kengne AP. Atrial thrombus detection on transoesophageal echocardiography in patients with atrial fibrillation undergoing cardioversion or catheter ablation: A pooled analysis of rates and predictors. J Cardiovasc Electrophysiol. 2021;32(8):2179-2188. doi:10.1111/jce.15082 https://doi.org/10.1111/jce.15082

39. Osorio J, Zei PC, Díaz JC, et al. High-Frequency Low-Tidal Volume Ventilation Improves Long-Term Outcomes in AF Ablation: A Multicenter Prospective Study. JACC Clin Electrophysiol. 2023;9(8 Pt 2):1543-1554. doi:10.1016/j.jacep.2023.05.015 https://doi.org/10.1016/j.jacep.2023.05.015

40. Pallikadavath S, Richards C, Bountziouka V, et al. The AFLETES Study: Atrial Fibrillation in Veteran Athletes and the Risk of Stroke. Clin J Sport Med. 2023;33(3):209-216. doi:10.1097/JSM.0000000000001115 https://doi.org/10.1097/JSM.0000000000001115

41. Patel J, Bhaskar SMM. Diagnosis and Management of Atrial Fibrillation in Acute Ischemic Stroke in the Setting of Reperfusion Therapy: Insights and Strategies for Optimized Care. J Cardiovasc Dev Dis. 2023;10(11):458. Published 2023 Nov 12. doi:10.3390/jcdd10110458 https://doi.org/10.3390/jcdd10110458

42. Pelliccia A, Sharma S, Gati S, et al. 2020 ESC Guidelines on sports cardiology and exercise in patients with cardiovascular disease. Eur Heart J. 2021;42(1):17-96. doi:10.1093/eurheartj/ehaa605 https://doi.org/10.1093/eurheartj/ehaa605

43. Pham HN, Abdelnabi MH, Ibrahim R, et al. Exercise and Atrial Fibrillation: Current Evidence, Knowledge Gaps, and Future Directions. Rev Cardiovasc Med. 2025;26(7):39200. Published 2025 Jul 28. doi:10.31083/RCM39200 https://doi.org/10.31083/RCM39200

44. Pham HN, Ibrahim R, Truong HH, et al. Advances in Atrial Fibrillation Management: A Guide for General Internists. J Clin Med. 2024;13(24):7846. Published 2024 Dec 23. doi:10.3390/jcm13247846 https://doi.org/10.3390/jcm13247846

45. Rossi M, Bucci T, Tartaglia E, et al. Physical activity and risk of adverse events in atrial fibrillation: evidence from European and Asian cohorts. Europace. 2026;28(2):euag032. doi:10.1093/europace/euag032 https://doi.org/10.1093/europace/euag032

46. Sale A, Yu J. Quality of life instruments in atrial fibrillation: a systematic review of measurement properties. Health Qual Life Outcomes. 2022;20(1):143. Published 2022 Oct 17. doi:10.1186/s12955-022-02057-y https://doi.org/10.1186/s12955-022-02057-y

47. Sciarra L, D'Ascenzi F, Borrelli A, et al. Atrial fibrillation among master athletes: A complex relationship. Int J Cardiol. 2026;450:134212. doi:10.1016/j.ijcard.2026.134212 https://doi.org/10.1016/j.ijcard.2026.134212

48. Siudak Z, Grygier M, Tomaniak M, et al. Interventional cardiology in Poland in 2023. Annual summary report of the Association of Cardiovascular Interventions of the Polish Cardiac Society (AISN PTK) and Jagiellonian University Medical College. Postepy Kardiol Interwencyjnej. 2024;20(4):379-381. doi:10.5114/aic.2024.144970 https://doi.org/10.5114/aic.2024.144970

49. Strømnes LA, Ree H, Gjesdal K, Ariansen I. Sex Differences in Quality of Life in Patients With Atrial Fibrillation: A Systematic Review. J Am Heart Assoc. 2019;8(8):e010992. doi:10.1161/JAHA.118.010992 https://doi.org/10.1161/JAHA.118.010992

50. Svennberg E, Freedman B, Andrade JG, et al. Recent-onset atrial fibrillation: challenges and opportunities. Eur Heart J. 2026;47(2):170-187. doi:10.1093/eurheartj/ehaf478 https://doi.org/10.1093/eurheartj/ehaf478

51. Tsuneda T, Yamashita T, Fukunami M, et al. Rate control and quality of life in patients with permanent atrial fibrillation: the Quality of Life and Atrial Fibrillation (QOLAF) Study. Circ J. 2006;70(8):965-970. doi:10.1253/circj.70.965 https://doi.org/10.1253/circj.70.965

52. Turagam MK, Flaker GC, Velagapudi P, Vadali S, Alpert MA. Atrial Fibrillation In Athletes: Pathophysiology, Clinical Presentation, Evaluation and Management. J Atr Fibrillation. 2015;8(4):1309. Published 2015 Dec 31. doi:10.4022/jafib.1309 https://doi.org/10.4022/jafib.1309

53. Vitolo M, Proietti M, Mantovani M, et al. Physical activity in patients with atrial fibrillation and adverse outcomes: results from a large-scale prospective multicenter European cohort. Rev Esp Cardiol (Engl Ed). 2026;79(2):151-164. doi:10.1016/j.rec.2025.07.005 https://doi.org/10.1016/j.rec.2025.07.005

54. Wan Q, Zhou Y, Zhu W, Liu X. Sex-Specific Exposure-Effect Relationship Between Physical Activity and Incident Atrial Fibrillation in the General Population: A Dose-Response Meta-Analysis of 16 Prospective Studies. Front Cardiovasc Med. 2021;8:710071. Published 2021 Sep 22. doi:10.3389/fcvm.2021.710071 https://doi.org/10.3389/fcvm.2021.710071

55. Willy K, Ellermann C, Reinke F, et al. The Impact of Cardiac Devices on Patients' Quality of Life-A Systematic Review and Meta-Analysis. J Cardiovasc Dev Dis. 2022;9(8):257. Published 2022 Aug 10. doi:10.3390/jcdd9080257 https://doi.org/10.3390/jcdd9080257

56. Zacher J, Filipovic K, Predel G, Schmidt T. Exercise and Atrial Fibrillation: The Dose Makes the Poison? A Narrative Review. Int J Sports Med. 2024;45(1):17-22. doi:10.1055/a-2152-7628 https://doi.org/10.1055/a-2152-7628

57. Zeppenfeld K, Tfelt-Hansen J, de Riva M, et al. 2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. Eur Heart J. 2022;43(40):3997-4126. doi:10.1093/eurheartj/ehac262 https://doi.org/10.1093/eurheartj/ehac262

58. Zhu W, Wu Z, Dong Y, Lip GYH, Liu C. Effectiveness of early rhythm control in improving clinical outcomes in patients with atrial fibrillation: a systematic review and meta-analysis. BMC Med. 2022;20(1):340. Published 2022 Oct 13. doi:10.1186/s12916-022-02545-4 https://doi.org/10.1186/s12916-022-02545-4