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Review Article

Atrial Fibrillation in Athletes: A Narrative Review of Training-Induced Remodeling, Thromboembolic Risk, and Return-to-Play Guidelines

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Abstract

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia and is increasingly recognized among endurance athletes despite the well-established cardiovascular benefits of regular physical activity. While moderate exercise reduces AF risk, prolonged high-intensity training may promote structural, electrical, and autonomic cardiac remodeling that predisposes susceptible athletes to atrial arrhythmogenesis. These adaptations, including left atrial enlargement, myocardial fibrosis, heightened vagal tone, and altered electrophysiological properties, create a complex clinical paradox in which exercise serves as both a protective and predisposing factor. The unique characteristics of athlete-associated AF also present important challenges in diagnosis, thromboembolic risk assessment, and clinical management, as conventional risk prediction tools and treatment strategies may not fully reflect the physiological adaptations or performance demands of this population. This narrative review synthesizes current evidence on the mechanisms linking training-induced cardiac remodeling to AF development in athletes, with emphasis on the interplay between structural remodeling, autonomic regulation, inflammation, and genetic susceptibility. It further examines the clinical presentation and diagnostic evaluation of AF in athletic populations, highlighting considerations for thromboembolic risk stratification and the limitations of existing scoring systems. Contemporary management approaches, including lifestyle modification, pharmacological therapy, catheter ablation, and individualized return-to-play recommendations, are critically discussed within the context of shared decision-making and athlete-centered care. Finally, emerging research priorities, including improved athlete-specific risk prediction, advanced imaging, wearable technologies, and long-term outcome evaluation, are outlined. By integrating current mechanistic and clinical evidence, this review provides a concise framework to support evidence-based prevention, diagnosis, management, and safe return-to-play decisions for athletes with AF while identifying key areas requiring further investigation.

Keywords: Atrial fibrillation, Athletes, Cardiac remodeling, Thromboembolic risk and Catheter ablation, and Return-to-play

Introduction

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia and a major contributor to cardiovascular morbidity and healthcare burden worldwide (Van Gelder *et al.,* 2024). Characterized by rapid and disorganized atrial electrical activity, AF increases the risk of thromboembolism, ischemic stroke, heart failure, hospitalization, and premature mortality (Joglar *et al.*, 2024). Although AF predominantly affects older adults with cardiometabolic disease, it is increasingly recognized among endurance athletes who otherwise exhibit excellent cardiovascular health (Abah *et al.,*

2025; Pham *et al.*, 2025). This paradox challenges the traditional view that increasing exercise always confers greater cardiovascular protection. While regular moderate-intensity exercise lowers AF risk, prolonged high-intensity endurance training may increase susceptibility in predisposed individuals (Pham *et al.,* 2025). Consequently, athlete-associated AF has emerged as a distinct clinical entity with unique pathophysiological mechanisms and management considerations (Turagam *et al.,* 2015; Moses *et al.,* 2026).

The increased occurrence of AF in athletes is primarily attributed to exercise-induced cardiac remodeling resulting from prolonged hemodynamic stress and physiological adaptation to intensive training (Pham *et al.,* 2025). Although endurance exercise promotes beneficial adaptations characteristic of the "athlete's heart," excessive training may induce atrial dilation, myocardial fibrosis, autonomic imbalance, and electrophysiological remodeling that facilitate atrial arrhythmogenesis (Turagam *et al.,* 2015; Pelliccia *et al.,* 2021). Enhanced vagal tone, recurrent atrial stretch, inflammation, and oxidative stress further contribute to AF development in susceptible individuals (Pham *et al.*, 2025). Emerging evidence also suggests possible sex-related differences in remodeling patterns and arrhythmic risk, although data in female athletes remain limited (Myrstad *et al.*, 2023). Distinguishing physiological adaptation from pathological remodeling therefore remains a major clinical challenge.

The diagnosis of AF in athletes requires careful evaluation because physiological adaptations to intensive training may mimic pathological cardiac changes (Pelliccia *et al.,* 2021). Athletes may present with palpitations, exercise intolerance, fatigue, or reduced performance, although many remain asymptomatic and are diagnosed through screening or ambulatory rhythm monitoring (Turagam *et al.*, 2015). Assessment should include evaluation for structural heart disease, inherited cardiac disorders, and reversible triggers such as alcohol use, sleep-disordered breathing, electrolyte imbalance, and excessive training (Joglar *et al.,* 2024). Thromboembolic risk assessment is equally important; however, conventional tools such as the CHA_2DS_2-VASc score may inadequately estimate stroke risk in younger athletes because they were developed primarily in older populations with multiple comorbidities (Van Gelder *et al.*, 2024). These limitations support the need for athlete-specific risk stratification strategies (Pham *et al.,* 2025).

Management of AF in athletes aims to relieve symptoms, prevent thromboembolism, preserve exercise capacity, and enable safe sports participation (Pelliccia *et al.*, 2021). Initial treatment focuses on lifestyle optimization, including modification of excessive training, management of cardiovascular risk factors, and treatment of reversible conditions such as sleep apnea (Van Gelder *et al.*, 2024). Pharmacological therapy should be individualized because some rate- and rhythm-control agents may impair athletic performance or increase proarrhythmic risk during strenuous exercise (Joglar *et al.*, 2024). Catheter ablation has become an effective rhythm-control strategy for appropriately selected athletes, providing symptom relief and facilitating return to competitive activity (Pham *et al.*, 2025). Current recommendations emphasize individualized management and shared decision-making when determining treatment strategies and return-to-play eligibility (Pelliccia *et al.*, 2021; Van Gelder *et al.,* 2024).

Despite advances in understanding atrial fibrillation, important uncertainties remain regarding its mechanisms, diagnosis, risk assessment, and management in athletes. Physiological cardiac adaptations often overlap with pathological remodeling, making clinical evaluation and treatment decisions particularly challenging. In addition, conventional thromboembolic risk assessment tools and management strategies may not fully address the unique characteristics and performance demands of athletic populations. This narrative review synthesizes current evidence on the relationship between training-induced cardiac remodeling and AF development in athletes. It examines the structural, electrical, and autonomic mechanisms underlying exercise-associated arrhythmogenesis, discusses the clinical presentation, diagnostic evaluation, and challenges of thromboembolic risk assessment, and evaluates contemporary management strategies, including lifestyle modification, pharmacological therapy, catheter ablation, and individualized return-to-play recommendations. The review also highlights existing knowledge gaps and future research priorities to improve athlete-specific prevention, diagnosis, risk stratification, and long-term outcomes, providing an evidence-informed resource for clinicians and researchers involved in the care of athletes with atrial fibrillation.

Training-Induced Cardiac Remodeling and AF Development

Prolonged endurance training induces a spectrum of cardiac adaptations involving structural, electrical, and autonomic remodeling. These changes are central to the development of the athlete's heart, a physiological state characterized by enhanced cardiac efficiency and exercise capacity. However, when exposure to intensive training is sustained over many years, particularly at high volumes, these adaptations may contribute to an arrhythmogenic substrate in susceptible individuals (Sepehri Shamloo *et al.,* 2018). The relationship between exercise and atrial fibrillation (AF) is therefore complex; while regular physical activity reduces cardiovascular risk, excessive endurance exercise may increase; Mfon *et al.,* 2025) AF susceptibility through cumulative remodeling processes (Mont *et al.*, 2009). Structural remodeling is a major component linking endurance exercise to AF development. Repeated increases in atrial pressure and volume load during prolonged exercise promote atrial enlargement and mechanical stretching, which may alter normal atrial architecture. Although moderate atrial enlargement represents an adaptive response that improves cardiac filling, persistent atrial dilation can facilitate electrical instability and increase vulnerability to AF (Sepehri Shamloo *et al.,* 2018). Structural abnormalities within the atrial myocardium, including fibrosis and altered tissue organization, are strongly associated with AF persistence by creating areas of conduction heterogeneity that support re-

entry mechanisms (Platonov *et al.,* 2011). Thus, the transition from physiological remodeling to pathological remodeling depends on the extent of structural changes and individual susceptibility. Fibrotic remodeling represents a key pathway through which chronic exercise-related stress may promote atrial arrhythmogenesis. Repeated mechanical strain, inflammation, and oxidative stress associated with intensive endurance training may stimulate extracellular matrix remodeling and increase collagen deposition within atrial tissue (Nattel *et al.,* 2020). Fibrosis disrupts the uniform propagation of electrical impulses by creating regions of conduction slowing and block, thereby facilitating initiation and maintenance of AF (Platonov *et al.*, 2011). However, the presence and clinical significance of fibrosis in athletes remain influenced by training intensity, duration, genetic factors, and other individual characteristics.

Electrical and autonomic remodeling further contribute to AF susceptibility in endurance athletes. Long-term training alters atrial electrophysiological properties, including conduction characteristics and refractoriness, creating conditions favourable for arrhythmia initiation (Nattel *et al.,* 2020). In addition, endurance athletes often exhibit enhanced parasympathetic activity, reflected by resting bradycardia and increased vagal tone. Although these autonomic adaptations improve cardiovascular efficiency, excessive vagal influence may shorten atrial refractory periods and promote re-entry pathways associated with AF, particularly during periods of rest and recovery (Mont *et al.,* 2009). The interaction between autonomic changes and structural remodeling is therefore considered an important mechanism underlying exercise-associated AF. Evidence also suggests that exercise-induced remodeling may extend beyond the atria and involve broader cardiac adaptations associated with arrhythmic risk. Intensive endurance training has been associated with transient right ventricular stress and remodeling, particularly following prolonged high-intensity exercise, although the clinical relevance varies among athletes (La Gerche *et al.,* 2015). These findings highlight that the cardiovascular effects of extreme endurance exercise are multifaceted and depend on the balance between adaptive responses and excessive physiological stress. Emerging data further indicate possible sex-related differences in remodeling patterns, with female endurance athletes demonstrating distinct adaptations that require additional investigation (Myrstad *et al.,* 2023). Overall, training-induced remodeling represents an interplay between atrial enlargement, fibrosis, electrical alterations, and autonomic regulation. While these adaptations generally support athletic performance, excessive or prolonged exposure may create an electrophysiological and structural environment conducive to AF development. Understanding the factors that determine this transition is essential for improving athlete-specific risk assessment, prevention strategies, and management approaches.

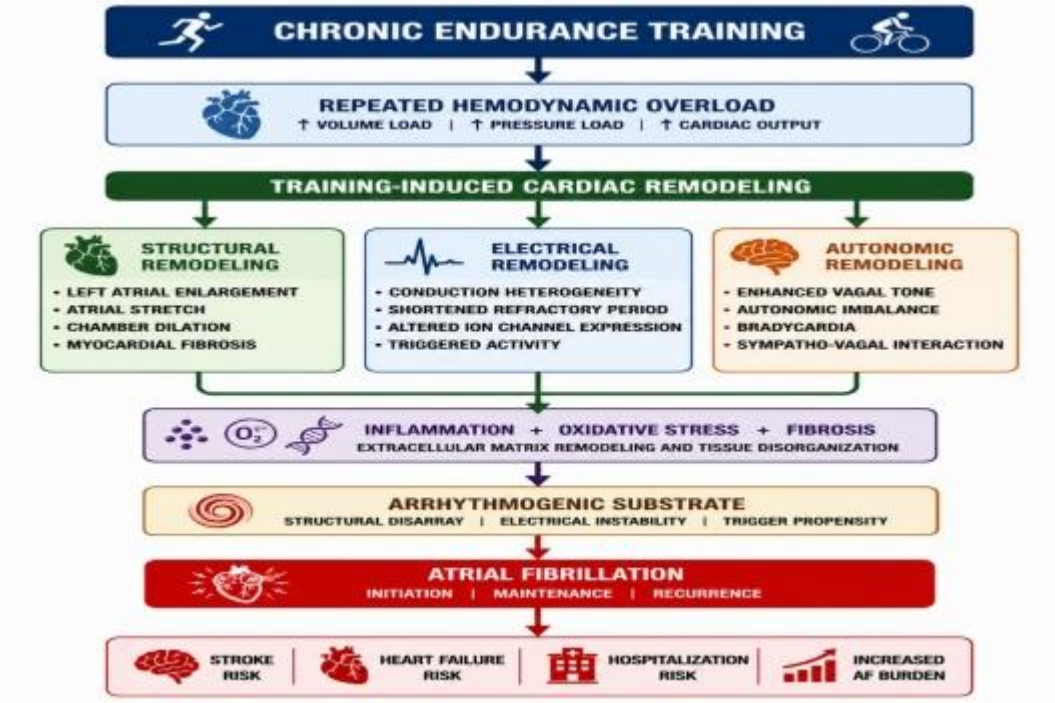

Figure 1. Mechanisms linking training-induced cardiac remodeling to atrial fibrillation in endurance athletes. Chronic endurance training produces repeated hemodynamic loading that induces structural, electrical, and autonomic cardiac remodeling. In susceptible athletes, these adaptations may promote atrial enlargement, fibrosis, inflammation, oxidative stress, and electrophysiological alterations, creating an arrhythmogenic substrate that predisposes to atrial fibrillation and its clinical consequences. **Source:** Adapted from Nattel *et al.* (2020)

Clinical Features, Diagnosis, and Thromboembolic Risk Assessment

Early recognition of atrial fibrillation (AF) in athletes is essential because its clinical manifestations are often subtle and may overlap with physiological adaptations to intensive exercise. Accurate diagnosis requires a systematic evaluation that distinguishes exercise-induced cardiac remodeling from underlying cardiovascular disease while identifying factors that influence disease severity and progression. Equally important is thromboembolic risk assessment, which guides anticoagulation decisions but remains challenging in athletic populations because conventional risk stratification tools may not fully reflect their unique clinical profile. This section reviews the clinical presentation, contemporary diagnostic approaches, and current strategies for assessing AF burden and thromboembolic risk in athletes, with emphasis on individualized, evidence-based evaluation.

Clinical Presentation and Diagnostic Evaluation

Atrial fibrillation (AF) in athletes often presents differently from AF in the general population, making early recognition challenging. Most affected athletes are younger, physically fit, and have few conventional cardiovascular risk factors, allowing the arrhythmia to remain undetected until symptoms interfere with athletic performance (Kourek *et al.*, 2024). Clinical manifestations range from palpitations, irregular heartbeat, reduced exercise tolerance, fatigue, and exertional dyspnea to dizziness or reduced competitive performance, although many athletes remain asymptomatic and are diagnosed incidentally during cardiovascular screening or rhythm monitoring (Lampert *et al.*, 2024). Because these symptoms may resemble normal physiological responses to intensive training or overtraining syndrome, clinicians should maintain a high index of suspicion, particularly in endurance athletes with recurrent or unexplained declines in performance (Abah *et al.,* 2025; Pham *et al.*, 2025). Diagnosis requires a comprehensive evaluation that integrates symptom history, training exposure, family history of cardiovascular disease, and potential reversible triggers such as alcohol intake, stimulant use, sleep-disordered breathing, electrolyte disturbances, and recent illness (Van Gelder *et al.*, 2024). A resting 12-lead electrocardiogram remains the standard initial investigation, but intermittent AF frequently necessitates prolonged ambulatory monitoring to establish symptom–rhythm correlation (Joglar *et al.,* 2024). Advances in wearable technologies, including smartwatch-based electrocardiography and photoplethysmographic monitoring, have improved the detection of paroxysmal and asymptomatic AF, although abnormal findings should always be confirmed using conventional electrocardiographic methods before treatment decisions are made (Vyas *et al.*, 2024; Zarak *et al.*, 2024; Park and Bae, 2025). Beyond confirming AF, diagnostic evaluation should identify underlying structural or inherited cardiac disease and distinguish pathological abnormalities from physiological exercise-induced remodeling. Transthoracic echocardiography is recommended to assess atrial size, ventricular function, and valvular disease, while cardiac magnetic resonance imaging may be considered when cardiomyopathy, myocardial fibrosis, or inflammatory heart disease is suspected (Lampert *et al.,* 2024). Exercise testing can further evaluate exercise-related symptoms and guide clinical decision-making in selected athletes (Pelliccia *et al.,* 2021). A systematic and individualized diagnostic approach therefore provides the foundation for accurate risk stratification, appropriate treatment selection, and subsequent thromboembolic risk assessment.

Thromboembolic Risk Assessment

Thromboembolic risk assessment is a fundamental component of AF management because it determines the need for anticoagulation and influences long-term clinical outcomes. Although athletes with AF are generally younger and have fewer cardiovascular comorbidities than the general AF population, they are not exempt from thromboembolic complications (Van Gelder *et al.*, 2024). Stroke risk in these individuals reflects a complex interaction between AF burden, atrial structural remodeling, and conventional clinical risk factors rather than athletic status alone. Consequently, risk assessment should balance evidence-based guideline recommendations with the unique physiological characteristics and sporting demands of individual athletes (Minardi *et al.*, 2024). Current international guidelines recommend using validated clinical risk scores, particularly the CHA_2DS_2-VASc score, to estimate stroke risk and guide anticoagulation decisions in patients with AF (Joglar *et al.,* 2024; Van Gelder *et al.,* 2024). However, these scoring systems were developed primarily in older populations with hypertension, diabetes mellitus, heart failure, and vascular disease, making their application to younger athletes less straightforward. Many athletes with exercise-associated AF have low CHA_2DS_2-VASc scores despite evidence of atrial enlargement or prolonged AF episodes, raising concerns that conventional risk models may underestimate clinically relevant thromboembolic risk in selected individuals (Kourek *et al.*, 2024). Conversely, indiscriminate anticoagulation based solely on arrhythmia presence may expose athletes to unnecessary bleeding complications without substantial clinical benefit (Minardi *et al.,* 2024). These limitations underscore the importance of individualized clinical judgment rather than exclusive reliance on risk scores. Assessment of AF burden provides additional information that complements conventional stroke risk stratification. The frequency, duration, and pattern of AF episodes may influence symptom severity, atrial remodeling, and disease progression, although their independent relationship with thromboembolic events remains incompletely defined (Van Gelder *et al.,* 2024). episodes, facilitating earlier clinical review and longitudinal follow-up (Vyas *et al.*, 2024).

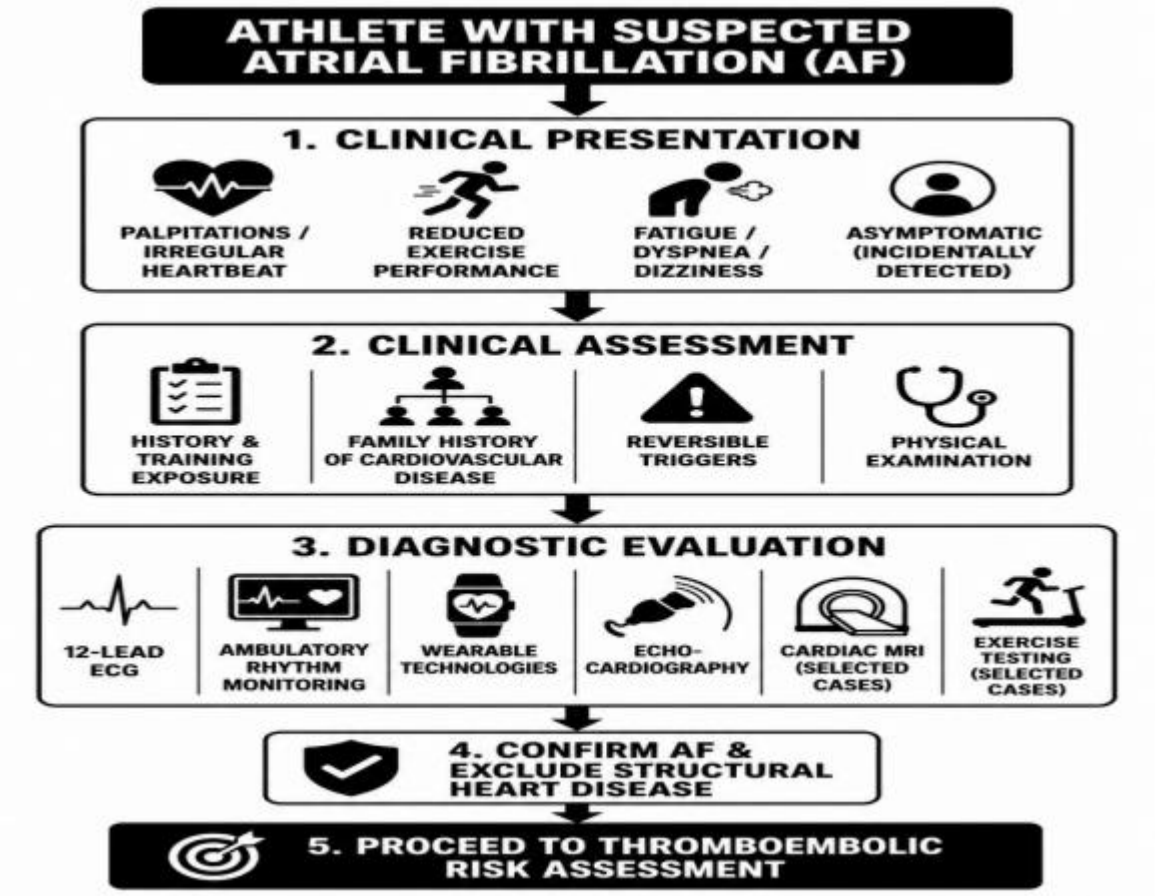

Figure 2. Clinical evaluation pathway for athletes with suspected atrial fibrillation. This flowchart summarizes the stepwise clinical evaluation of athletes with suspected atrial fibrillation, from symptom recognition and clinical assessment to electrocardiographic confirmation, complementary cardiac investigations, and exclusion of structural heart disease. The diagnostic pathway supports individualized thromboembolic risk assessment and informs subsequent management decisions.
Source: Modified from Joglar *et al.* (2024)

Continuous rhythm monitoring using ambulatory ECG devices, implantable loop recorders, and increasingly wearable technologies enables more accurate quantification of AF burden than intermittent clinical evaluation alone (Park and Bae, 2025). Smartwatch-based ECG systems and photoplethysmographic monitoring have demonstrated growing utility for detecting recurrent or asymptomatic AF Nevertheless, wearable devices should be regarded as adjunctive screening tools, with abnormal findings requiring confirmation using standard electrocardiographic methods before therapeutic decisions are made (Zarak *et al.,* 2024). Individualized assessment should also identify factors that may modify thromboembolic or bleeding risk beyond traditional scoring systems. Echocardiographic evaluation of left atrial size and function, assessment for structural heart disease, renal function, and coexisting cardiovascular disorders contribute to a more comprehensive evaluation of overall risk (Lampert *et al.,* 2024). Equally important are sport-specific considerations. Athletes participating in contact or collision sports may face a substantially increased risk of traumatic bleeding if anticoagulation is initiated, whereas those engaged in non-contact endurance sports generally present lower injury-related bleeding risks (Minardi *et al.,* 2024). Decisions regarding anticoagulant therapy should therefore integrate stroke risk, bleeding risk, competitive demands, and athlete preferences within a shared decision-making framework, consistent with contemporary guideline recommendations (Joglar *et al.,* 2024; Van Gelder *et al.*, 2024). Current evidence increasingly supports a personalized approach to thromboembolic prevention in athletes with AF rather than a uniform treatment strategy. Risk assessment should incorporate validated clinical scores together with AF burden, structural cardiac evaluation, sport-specific bleeding risk, and individual clinical characteristics to optimize therapeutic decisions (Kourek *et al.,* 2024). Continued research is needed to determine whether athlete-specific risk models can improve prediction of thromboembolic events beyond conventional scoring systems and better inform anticoagulation strategies in this unique population. Until such evidence becomes available, individualized, guideline-directed assessment remains the cornerstone of thromboembolic risk evaluation in athletes with atrial fibrillation.

Table 1. Components of thromboembolic risk assessment in athletes with atrial fibrillation

Components	Clinical relevance	Athlete-specific consideration
CHA_2DS_2-VASc score	Estimates stroke risk	May underestimate risk in young athletes
AF burden	Frequency and duration of AF	Assessed using ambulatory/wearable monitoring
Cardiac structure	Left atrial size and structural disease	Echocardiography/CMR findings
Bleeding risk	Determines anticoagulation safety	Contact vs non-contact sports
Shared decision-making	Individualizes treatment	Balances stroke prevention with athletic participation

Management and Return-to-Play Guidelines

Effective management of atrial fibrillation (AF) in athletes extends beyond rhythm control to encompass symptom relief, prevention of thromboembolic events, preservation of exercise capacity, and safe continuation of sports participation. Unlike the general AF population, treatment decisions in athletes must account for the physiological demands of intensive exercise, the potential effects of therapy on athletic performance, and the risks associated with competitive sports. Contemporary management therefore emphasizes individualized, evidence-based strategies that combine lifestyle optimization, pharmacological or interventional therapies, and carefully planned return-to-play decisions within a shared decision-making framework (Lampert *et al.*, 2024; Van Gelder *et al.,* 2024).

Therapeutic Management

Management of atrial fibrillation (AF) in athletes aims to relieve symptoms, prevent thromboembolic complications, maintain exercise capacity, and facilitate safe athletic participation. Treatment should be individualized according to symptom burden, AF pattern, underlying cardiac disease, and the athlete's competitive goals, while integrating current guideline recommendations with sport-specific considerations (Lampert *et al.*, 2024; Van Gelder *et al.*, 2024). Lifestyle and risk-factor modification forms the foundation of therapy and should accompany all treatment strategies. Addressing reversible factors such as excessive endurance training, alcohol consumption, stimulant use, obesity, hypertension, sleep-disordered breathing, and electrolyte imbalance can reduce AF recurrence and improve long-term cardiovascular health (Joglar *et al.,* 2024). Athletes with recurrent AF may also benefit from temporary reductions in training intensity while precipitating factors are corrected, although regular moderate exercise should be maintained whenever clinically appropriate (Sepehri Shamloo *et al.,* 2018; Pham *et al.,* 2025). Pharmacological therapy should be selected with consideration of both therapeutic efficacy and athletic performance. Although rate-control agents effectively control ventricular rate, they may impair maximal exercise capacity, making rhythm-control strategies preferable for many competitive athletes (Alsunbuli, 2020). Antiarrhythmic drugs can reduce AF recurrence but require careful selection because of their potential adverse effects and proarrhythmic risk during vigorous physical activity (Van Gelder *et al.,* 2024). Contemporary evidence further supports early rhythm-control therapy to improve symptom control, reduce AF progression, and preserve long-term cardiovascular outcomes in appropriately selected patients (Kirchhof *et al.*, 2020). Catheter ablation has become an important rhythm-control option for symptomatic athletes with recurrent or drug-refractory AF. Advances in ablation techniques have improved procedural safety and long-term maintenance of sinus rhythm, allowing many athletes to avoid prolonged antiarrhythmic drug therapy and return to high-level physical activity (Tzeis *et al.,* 2024). Current expert consensus recommends individualized patient selection based on symptom severity, AF characteristics, structural heart disease, and procedural risk rather than adopting a uniform treatment approach (Calkins *et al.,* 2017; Tzeis *et al.,* 2024).

Figure 3. Therapeutic management pathway for athletes with atrial fibrillation. This flowchart summarizes the stepwise therapeutic management of athletes with atrial fibrillation, beginning with clinical assessment and lifestyle modification, followed by individualized rate or rhythm control, catheter ablation when indicated, anticoagulation based on thromboembolic risk, and clinical reassessment before return-to-play decisions.
Source: Adapted from Hindricks *et al.* (2021)

Return-to-Play and Shared Decision-Making

Return-to-play (RTP) decisions in athletes with atrial fibrillation (AF) should be individualized, balancing cardiovascular safety with the athlete's performance goals and the demands of the sport. Rather than relying on fixed timelines, RTP should be guided by symptom resolution, rhythm stability, treatment response, underlying cardiac health, and the risk associated with the specific sporting discipline (Lampert *et al.,* 2024). Athletes who achieve adequate symptom control and demonstrate no clinically significant structural heart disease or recurrent arrhythmia can generally resume training following comprehensive clinical reassessment, whereas persistent symptoms or recurrent AF warrant further evaluation and treatment before returning to competition (Pelliccia *et al.*, 2021; Van Gelder *et al.*, 2024). Continuous electrocardiographic monitoring may further support individualized RTP decisions by detecting recurrent or asymptomatic AF during recovery and follow-up (Muir *et al.*, 2025). Treatment strategy also influences RTP recommendations. Athletes receiving antiarrhythmic medications should be monitored for adverse effects that may impair exercise performance, while those requiring anticoagulation need careful assessment of bleeding risk, particularly in contact and collision sports (Minardi *et al.,* 2024). For appropriately selected athletes, successful catheter ablation can restore sinus rhythm, improve functional capacity, and facilitate an earlier return to unrestricted athletic participation, provided post-procedural evaluation confirms sustained rhythm control (Tzeis *et al.,* 2024). Shared decision-making is fundamental to contemporary management because treatment choices often affect both long-term cardiovascular health and athletic careers. Decisions should involve the athlete and a multidisciplinary team, integrating clinical evidence with individual preferences, competitive goals, and the potential benefits and risks of available therapies (Ali-Ahmed *et al.*, 2020). Ongoing clinical follow-up and rhythm surveillance remain essential to detect AF recurrence, optimize treatment, and support safe long-term participation in competitive sports (Joglar *et al.,* 2024). Through an individualized, evidence-based approach, many athletes with AF can successfully return to sport while maintaining optimal cardiovascular outcomes.

Table 2. Factors influencing return-to-play decisions in athletes with atrial fibrillation

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Factor	Clinical consideration
Symptom status	Complete symptom control
Rhythm stability	No recurrent or uncontrolled AF
Structural heart disease	Excluded or adequately managed
Treatment response	Successful medical therapy or catheter ablation
Anticoagulation	Bleeding risk based on sport type
Sport characteristics	Contact vs. non-contact sports
Shared decision-making	Athlete preferences and multidisciplinary input
Follow-up	Ongoing rhythm surveillance

Future Perspectives

Despite substantial advances in understanding atrial fibrillation (AF) in athletes, important knowledge gaps remain regarding its prevention, risk prediction, monitoring, and long-term clinical outcomes. Current evidence is derived predominantly from observational studies involving male endurance athletes, limiting the generalizability of findings to women, younger athletes, and participants in diverse sporting disciplines (Kourek *et al.,* 2024). Future research should prioritize large prospective cohorts to better define the threshold at which physiological cardiac remodeling progresses to pathological atrial remodeling and to identify athlete-specific biomarkers that improve early detection and risk stratification. Advances in digital health and precision medicine offer promising opportunities for improving athlete-specific AF management. Artificial intelligence, machine learning, and wearable electrocardiographic technologies have demonstrated increasing potential for continuous rhythm monitoring, early AF detection, individualized risk prediction, and longitudinal assessment of treatment outcomes (Papalamprakopoulou *et al.,* 2024; Vyas *et al.,* 2024). Integration of clinical characteristics with genetic, imaging, and digital health data may facilitate personalized prevention strategies and more accurate prediction of arrhythmia recurrence and long-term prognosis (Petzl *et al.*, 2024; Wang *et al.,* 2025). Future guideline development should therefore emphasize athlete-specific evidence to refine prevention strategies, optimize therapeutic decision-making, and establish standardized return-to-play recommendations that balance competitive participation with long-term cardiovascular health.

Conclusion

Atrial fibrillation in athletes represents a distinct clinical entity arising from the complex interaction between physiological adaptation to intensive exercise and pathological atrial remodeling. Although regular physical activity remains highly beneficial for cardiovascular health, prolonged high-intensity endurance training may increase susceptibility to atrial fibrillation in predisposed individuals. Accurate diagnosis, individualized thromboembolic risk assessment, and tailored therapeutic strategies are essential to optimize clinical outcomes while preserving athletic performance. Contemporary management emphasizes lifestyle modification, appropriate pharmacological therapy, catheter ablation when indicated, and shared decision-making to support safe return to sport. Continued research is needed to improve athlete-specific risk prediction, refine preventive strategies, and strengthen evidence-based return-to-play recommendations, ultimately enhancing both long-term cardiovascular health and athletic participation.

Authors' Contributions

The authors of this research have significantly contributed to the study's conception, data collection, and manuscript development. All authors were involved in writing the manuscript or critically reviewing it for its intellectual value. They have reviewed and approved the final version for submission and publication and accepted full responsibility for the content and integrity of the work.

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Conflict of Interest

The authors declared that there are no conflicts of interest.

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