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Lifestyle and Atrial Fibrillation: The Role of Obesity, Exercise, and Weight Loss in Risk Reduction and Rhythm Management

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Abstract

Introduction and purpose. Atrial fibrillation (AF) is the most common sustained arrhythmia worldwide, closely associated with rising obesity rates and sedentary lifestyles. This review evaluates the evidence linking obesity, physical activity, and weight reduction with AF development, progression, and management.

Material and methods. A comprehensive literature review was conducted using PubMed and Google Scholar, prioritizing randomized trials, meta-analyses, and recent studies published within the past eight years addressing obesity, physical activity, and weight loss in AF.

Brief description of the state of knowledge. Obesity is a major risk factor for atrial fibrillation, with each 5-unit BMI increase linked to a 28–30% rise in risk. Higher BMI is associated with increased AF incidence, faster progression, and recurrence rates up to 50% after ablation in severe obesity. Moderate physical activity contributes to lower risk of AF and improved rhythm control, while high-intensity endurance exercise may elevate AF risk, particularly in middle-aged men. Sustained weight loss of at least 10% significantly

reduces AF burden, recurrence rates, and symptom severity. Bariatric surgery has been shown to decrease AF incidence in severely obese patients, and long-term weight stability enhances these benefits. Anticoagulant efficacy remains stable across BMI ranges, especially rivaroxaban, which shows favorable profiles in obese patients.

Conclusions. Current evidence supports the integration of lifestyle interventions such as targeted weight loss and individualized physical activity into AF management. These strategies offer significant benefits to AF risk, arrhythmia control, and overall outcomes, supporting routine implementation in clinical practice.

Keywords: atrial fibrillation, obesity, exercise, weight loss

Introduction and puropse

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia encountered in clinical practice with an estimated global prevalence of 50 million individuals as of 2020. In the United States alone, AF is projected to increase to 2.6 million by 2030, a three-fold increase over serval decades. Such rising prevalence creates a considerable burden for healthcare systems with AF-related health costs estimated at $28.4 billion. The clinical importance of AF extends beyond its prevalence, since it is linked with higher risks of death, heart failure, myocardial infarction, stroke, and cognitive decline (1).

Parallel to the rising incidence of AF, obesity has reached epidemic proportions globally and has emerged as an independent risk factor for AF development. Studies indicate a 4% increase in AF risk for each unit increase in body mass index (BMI), with a 5-unit BMI increase contributing to approximately 30% of AF cases. This relationship appears bidirectional, as obesity not only increases AF risk but also influences its progression, treatment efficacy, and clinical outcomes (1-3).

Physical activity represents another critical modifiable factor in AF pathophysiology. The relationship between exercise and AF demonstrates a complex, non-linear pattern that challenges simplistic recommendations. While regular moderate exercise is protective against AF development, excessive endurance exercise paradoxically enhances AF risk in certain subjects. Moreover, structured physical regimens have potential to reduce AF burden in patients with established arrhythmia (4, 5).

The interconnected nature of these factors presents both challenges and opportunities for clinicians. Understanding the mechanisms linking obesity and physical activity to AF, recognizing paradoxical relationships, and implementing evidence-based interventions may substantially improve patient outcomes.

This study aims to investigate the relationship between atrial fibrillation, obesity, physical activity, and weight reduction. It focuses on the mechanisms linking these factors to AF

development and progression, including the impact of exercise intensity and the obesity paradox. The study also evaluates evidence on lifestyle interventions such as weight loss, exercise programs, and bariatric surgery, and their effectiveness in reducing AF burden, supporting the integration of lifestyle modification into standard AF management.

Table 1: 2024 European Society of Cardiology Guideline-Based Recommendations on Physical Activity and Weight Management in AF (6).

Recommendation	Class	Level of Evidence	Interpretation
Bariatric surgery may be considered alongside lifestyle changes and medical therapy in patients with AF and BMI ≥40 kg/m² where rhythm control is pursued.	IIb	C	Usefulness is less well established; based on expert opinion or small studies.
Weight loss should be part of AF risk factor management in overweight or obese individuals, targeting at least a 10% reduction in body weight to reduce symptoms and AF burden.	I	B	Strong recommendation; supported by a single RCT or large non-randomized study.
A tailored exercise program is recommended in patients with paroxysmal or persistent AF to improve fitness and reduce AF recurrence.	I	B	Strong recommendation; supported by a single RCT or large non-randomized study.
Maintaining a normal body weight (BMI 20–25 kg/m²) is recommended for AF prevention in the general population.	I	B	Strong recommendation; supported by a single RCT or large non-randomized study.
An active lifestyle is recommended to prevent AF, with 150–300 minutes/week of moderate or 75–150 minutes/week of vigorous aerobic activity.	I	B	Strong recommendation; supported by a single RCT or large non-randomized study.

Weight reduction should be considered in obese individuals to prevent AF development.	IIa	B	Should be considered; evidence is supportive though not conclusive, from a single RCT or large non-randomized study.

Material and methods

A literature review was conducted using PubMed and Google Scholar databases with search terms such as "atrial fibrillation", "obesity", "physical activity", "weight loss", and all variations related to these terms. Clinical trials randomized controlled trials, meta-analyses, systematic reviews, and other review articles were included in the review. The articles published within the last eight years have been given priority to ensure a deep understanding of the topic and maintain up-to-date results of the research.

Obesity and AF Pathogenesis

Epidemiological data consistently show a strong, dose-dependent relationship between obesity and AF risk, with approximately 18% of AF cases attributable to elevated BMI (3, 7). Obesity not only increases the likelihood of developing AF but also influences its progression, accelerating the transition from paroxysmal to persistent forms and contributing to higher recurrence rates after interventions such as cardioversion or catheter ablation (1, 2). Notably, patients with class III obesity (BMI $\geq$40 kg/m^2) can experience AF recurrence rates as high as 48% within three years post-ablation (8). The presence of common obesity-related comorbidities, including hypertension, diabetes, and obstructive sleep apnea further compounds AF risk.

Obesity promotes AF via numerous interconnected processes of structural and electrical remodeling of the atria that are mediated predominantly by hemodynamic changes, actions of adipose tissue, systemic inflammation, oxidative stress, metabolic dysregulation, and autonomic dysfunction. Hemodynamic alterations, such as increased blood volume, elevated cardiac output, and left ventricular hypertrophy, elevate left atrial pressure and cause atrial dilation, thereby facilitating re-entry circuits and arrhythmogenesis (1, 7). By infiltrating the atrial myocardium and interfering with electrical conduction, epicardial adipose tissue (EAT) contributes significantly to AF. In Table 2, EAT's pathophysiologic mechanisms are described. Concurrently, obesity-associated metabolic disturbances, including insulin resistance,

dyslipidemia, and metabolic syndrome, exacerbate endothelial dysfunction, mitochondrial impairment, and lipotoxicity, further intensifying atrial remodeling (3, 9). Moreover, obesity induces autonomic nervous system imbalance characterized by enhanced sympathetic activity and reduced parasympathetic tone, shortening atrial refractory periods, increasing automaticity, and promoting AF susceptibility and persistence (10). Additionally, obesity-related electrical remodeling, marked by ion channel alterations and impaired calcium regulation, further heightens the arrhythmogenic potential (11).

Additionally, inflammation associated with obesity-related atrial fibrillation is thought to be mediated by the NLRP3 inflammasome. Upregulation of the NLRP3 inflammasome in atrial tissues of obese individuals promotes increased secretion of pro-inflammatory cytokines, particularly interleukin-1β, driving atrial inflammation, fibrosis, and electrical remodeling. Animal studies show that inhibition or genetic deletion of NLRP3 significantly reduces AF susceptibility, highlighting its therapeutic potential as a target to mitigate AF risk in obesity (12).

Table 2: Mechanisms by which EAT Influences Atrial Fibrillation (13).

Mechanism	Description
Fatty infiltration	Adipocytes infiltrate atrial myocardium, disrupting conduction and promoting anisotropy.
Fibrosis	Secretion of pro-fibrotic adipokines (e.g., activin A, MMPs) leads to atrial fibrosis.
Inflammation	Epicardial fat secretes inflammatory markers (e.g., CRP, IL-6, TNF-α), promoting local inflammation.
Oxidative stress	Epicardial fat generates reactive oxygen species, enhancing oxidative stress and atrial remodeling.
Autonomic dysfunction	Contains ganglionated plexi influencing autonomic tone; implicated in arrhythmia initiation.
Cardiac muscle activity	Peri-atrial EAT expresses genes linked to calcium signaling and muscle contraction.

Adipocyte-related gene expression	AF and pacing promote expression of adipocyte genes, suggesting AF-induced adipogenesis.
Diastolic dysfunction	Mechanical compression and paracrine effects from EAT contribute to diastolic dysfunction and atrial enlargement.
Trigger facilitation	Epicardial fat correlates with high-frequency electrical sites, potentially affecting AF triggers.

The Obesity Paradox in AF

Interesting clinical observation in AF is the "obesity paradox," wherein patients who are overweight and obese with known AF appear to have better outcomes than their normal-weight counterparts. Even though obesity is a certain risk factor for the occurrence of AF, numerous studies have shown lower mortality among overweight and obese AF patients compared with normal BMI patients.

Two substudies of the ARISTOTLE trial found that overweight and obese patients with atrial fibrillation had significantly lower risks of adverse outcomes compared to those with normal weight. Higher BMI was independently associated with reduced all-cause mortality (Hazard Ratio (HR) 0.70; 95% CI 0.59–0.84), cardiovascular mortality (HR 0.66; 95% CI 0.51–0.85), and stroke or systemic embolism (HR 0.71; 95% CI 0.52–0.97). (14, 15). Furthermore, this meta-analysis showed that anticoagulated AF patients who were overweight, obese, or morbidly obese had significantly lower risks of stroke or systemic embolism (Relative Risks (RRs) 0.80, 0.63, and 0.42, respectively) and all-cause mortality (RRs 0.73, 0.61, and 0.56, respectively) compared to those with normal BMI. They also had reduced risks of major bleeding (RRs 0.86 and 0.88) and intracranial bleeding (RRs 0.75 and 0.57). In contrast, underweight patients had significantly increased risks of thromboembolism (RR 1.92) and mortality (RR 3.57) (16). The ORBIT-AF registry, involving 9,606 patients with atrial fibrillation, found that higher BMI was linked to greater use of anticoagulation and rhythm control strategies, along with improved clinical outcomes. All-cause mortality and thromboembolic event rates decreased across rising BMI categories ($p < 0.001$). After adjustment, higher BMI remained independently associated with lower all-cause mortality, with the most pronounced benefit in class I obesity (HR 0.65; 95% CI 0.54–0.78). Each 5

kg/m² increase in BMI was associated with a 7% reduction in mortality risk. BMI was not significantly linked to adjusted risks of thromboembolic events or AF progression. (17).

The “obesity paradox” of atrial fibrillation (AF) remains debated, and reasons behind the inconsistent findings on it in AF are not fully understood. While some studies report improved outcomes for overweight and obese patients, others show increased risks. One long-term study of over 3,000 AF patients linked obesity to higher rates of major adverse events and mortality (18). However, the ENGAGE AF-TIMI 48 trial found lower risks of stroke, systemic embolism, and death in this population (19), though another meta-analysis showed no significant difference in mortality between obese and normal-weight patients (20). The bleeding risk data are also conflicting. Some reviews reported reduced rates in obese individuals (16), while ENGAGE AF-TIMI 48 showed a BMI-related increase. Despite these uncertainties, weight loss remains beneficial for reducing AF burden and promoting atrial remodeling (21). Randomized trials tend to show better outcomes with a higher BMI, whereas observational data are inconclusive (22). Early treatment access and better management in obese patients, and potential metabolic reserves, may partly explain these findings, though the evidence is limited.

Impact of Obesity on AF

Despite the 'obesity paradox" retaining some controversy, increasing evidence underscores obesity's role in atrial fibrillation development and progression. In the EPIC-Norfolk cohort study, normal body weight was significantly associated with a reduced risk of AF over a mean follow-up of 17.1 years. Participants with a BMI <25 kg/m² had the greatest risk reduction, and those with BMI between 25–27.5 kg/m² also showed a protective effect. Compared to individuals with optimal lifestyle scores, including healthy weight, those with higher BMI had up to a 2.8-fold increased risk of AF (23). Furthermore, a pooled analysis of 51 studies involving over 600,000 individuals and 20,000 AF events found a clear dose–response relationship between BMI and AF risk. Each 5-unit BMI increase was linked to a 28% higher risk (RR 1.28, 95% CI 1.20–1.38), consistent across age, sex, and comorbidity subgroups, supporting a direct, dose-dependent association (24).

The meta-analysis examining the relationship between obesity and incident of AF demonstrated that obesity significantly increases the risk of developing AF (RR=1.51, 95% CI 1.35–1.68; P<0.00001). Among 587,372 participants, AF incidence was notably higher in obese subjects compared to non-obese controls (6.3% vs. 3.1%). Importantly, the increased risk associated with obesity was consistent across genders, with similar relative risks observed

in men (RR=1.41, P<0.00001) and women (RR=1.53, P<0.00001). These findings confirm obesity as a significant independent risk factor for new-onset AF (25).

Folli et al. evaluated the impact of varying degrees of obesity on newly diagnosed AF and recurrent AF after ablation. Overweight, obese, and morbidly obese patients showed a significantly higher risk of newly diagnosed AF compared to individuals with normal weight ($p<0.01$). Similarly, obese and morbidly obese patients demonstrated increased risk of recurrent AF post-ablation. Notably, the association between obesity and new-onset AF was more pronounced in females compared to males (26).

Regarding the impact of obesity on AF management, a randomized clinical trial (RCT) investigated the efficacy and safety of dual direct-current cardioversion (DCCV) versus traditional single DCCV in patients with obesity (BMI ≥35) undergoing cardioversion. Among 200 patients, dual DCCV using two simultaneous 200-J shocks achieved significantly higher success in restoring sinus rhythm compared to single DCCV (98% vs. 86%, P = .002). Dual cardioversion remained an independent predictor of success (OR 6.7; 95% CI 3.3–13.6; P = .01). Importantly, all patients whose initial single DCCV failed were successfully converted with subsequent dual attempts(27). Intriguing results from anticoagulation treatment in obese AF patients were also noticed. A meta-analysis of ten studies with 168,081 obese patients with non-valvular AF found rivaroxaban significantly more effective and safer than warfarin. It was linked to lower risks of ischemic stroke (RR 0.79), hemorrhagic stroke (RR 0.61), systemic embolism (RR 0.73), and major bleeding (RR 0.75). These results suggest rivaroxaban may offer better outcomes, though further large trials are needed. (28). Building on previous findings, a pooled analysis from COMBINE AF including 58,464 patients (median BMI 28.3 kg/m²) confirmed the efficacy and safety of novel oral anticoagulants (NOACs) over warfarin across BMI ranges. NOACs reduced the risk of stroke/systemic embolism (HRadj 0.80), major bleeding (HRadj 0.88), and all-cause mortality (HRadj 0.91; all P < 0.01). However, these benefits were less pronounced at higher BMI, suggesting that while NOACs remain effective, their safety profile may be attenuated in severe obesity (29). Furthermore, the SORT-AF randomized trial assessed the impact of weight loss on ablation outcomes in 133 obese patients (BMI 30–40 kg/m²). While both groups had safe and successful ablations, those in the structured weight-reduction program saw a significant BMI decrease (P < 0.001). Although overall AF burden post-ablation was similar (P = 0.815), weight loss was linked to reduced AF recurrence in patients with persistent AF (P = 0.032), supporting lifestyle modification as a valuable adjunct to procedural treatment (30).

Physical Activity in AF: Mechanisms and Interventions

The relationship between physical activity and atrial fibrillation (AF) is complex, often described as J- or U-shaped, with risk influenced by exercise intensity and individual factors. Moderate physical activity lowers AF risk through beneficial effects such as improved cardiovascular profiles, reduced inflammation, enhanced parasympathetic tone, and better cardiorespiratory fitness. These changes help reduce atrial strain, limit fibrosis, and support normal electrical function, while also contributing to weight and visceral fat reduction, which are key factors in AF prevention. On the contrary, intense endurance exercise has the potential to increase AF risk via mechanisms that include atrial stretch, structural remodeling, inflammation during recovery, and vagal tone increase. The endurance athletes often have left atrial enlargement and vagally mediated shortened refractory periods, which may promote re-entry circuits and ectopic activity (4, 5). Risk is highest in middle-aged (40–60 years) male endurance athletes, although female athletes may have lower or even protective risk. There is also a difference between endurance sports like long-distance running and resistance or team sports when it comes to AF. A prolonged high-intensity exercise promotes structural changes beyond the left atrium, including bi-atrial enlargement, ventricular remodeling, and delayed conduction. Persistent inflammation and elevated cytokine levels in overtrained athletes also increase the fibrotic processes. Although AF in athletes rarely increases mortality, it may impair quality of life, performance, and competition status. These findings underscore the importance of individualized, balanced exercise in prevention and treatment of AF, considering intensity, duration, and individual risk factors (31, 32).

Recent evidence bridges the gap between theory and practice, demonstrating how tailored physical activity influences AF risk and recurrence. The ACTIVE-AF randomized trial showed that a 6-month structured aerobic exercise program significantly reduced AF recurrence and improved symptoms in patients with symptomatic AF. At 12 months, 40% of the exercise group remained AF-free without antiarrhythmic therapy or ablation, compared to 20% in the control group (HR: 0.50; 95% CI: 0.33–0.78). Symptom severity was lower in the exercise group at both 6 and 12 months, though overall symptom burden improvements were not sustained (33). The CARDIO-FIT study provided additional insights. In obese patients with AF, higher baseline cardiorespiratory fitness and improvements of ≥2 METs were associated with significantly better arrhythmia-free survival, reduced AF burden, and lower symptom severity ($p < 0.001$). Patients with the greatest fitness levels or gains showed superior rhythm control outcomes, regardless of additional therapies (34).

Extending this evidence, a meta-analysis of 15 prospective cohort studies involving over 1.46 million individuals found that who met guideline-recommended physical activity levels (≥450 MET-minutes per week) had a significantly lower risk of developing AF compared to inactive individuals (HR: 0.94; 95% CI: 0.90–0.97; P = .001). According to the dose-response analysis, increasing physical activity levels up to 1900 MET-minutes per week reduced the risk of AF progressively. However, the protective effect appeared to plateau beyond 2000 MET-minutes per week. The results support a positive, dose-dependent relationship between moderate to high PA levels and lower incidence of AF, but with diminishing returns at very high activity levels. (4). In contrast, a meta-analysis of 23 observational studies involving over 1.9 million individuals found no overall association between physical activity and AF risk (RR: 0.99; 95% CI: 0.93–1.05) but identified a sex-specific pattern higher risk in men and lower risk in women. However, the evidence was of low to moderate quality and based solely on observational data, limiting the strength of its conclusions (35).

Finally, a comprehensive systematic review and meta-analysis confirmed that moderate physical activity reduces AF risk in the general population, while long-term high-intensity endurance exercise may increase it—particularly in male athletes. Among over 935,000 individuals, adherence to guideline-recommended activity levels (≥7.5 MET-hours/week) was linked to a modest but significant reduction in AF incidence. In contrast, male endurance athletes, such as professional football players, cyclists, and runners, showed a significantly higher prevalence of AF than non-athletic controls. These results emphasize the need for individualized physical activity recommendations that balance intensity and duration, aligning with personal risk profiles and exercise patterns (36).

Weight Loss Interventions

Beyond exercise, sustained weight loss plays a crucial role in atrial fibrillation management, with growing evidence linking it to improved rhythm control and symptom relief. Aldaas et al. created a systematic review of five studies including 548 individuals with AF. Patients who lost at least 10% of their body weight had a significantly lower risk of arrhythmia recurrence (RR = 0.29; 95% CI: 0.19–0.44). They also experienced fewer AF episodes, shorter episode duration, and less severe symptoms overall (37). The LEGACY study further supports the importance of sustained weight loss in managing AF. Among 355 obese patients with AF, those who lost ≥10% of body weight had significantly lower AF burden, fewer symptoms, and the highest rates of arrhythmia-free survival, both with and without rhythm control therapies. This group had a six-fold greater likelihood of maintaining sinus rhythm compared to those with less weight loss. Nevertheless, weight fluctuation >5% significantly increased

the risk of AF recurrence, highlighting the need for long-term weight stability to maintain the benefits of weight reduction in AF management (38). These findings are reinforced by a meta-analysis of 10 studies involving 1,851 patients undergoing catheter ablation. While the overall recurrence rate was lower in those who lost weight (34.5% vs. 58.2%), the difference was not statistically significant (RR = 0.76; 95% CI: 0.49–1.18; p = .22). However, significant benefits emerged with longer follow-up: at >12 months post-ablation, weight loss was associated with a markedly reduced risk of recurrence (RR = 0.47; 95% CI: 0.32–0.68; p < .0001). Notably, both ≥10% and <10% weight loss showed benefit (39).

Bariatric surgery is an effective option for severely obese patients, with a meta-analysis of 10 studies showing a significant reduction in incident AF compared to medical treatment (OR = 0.665; 95% CI: 0.475–0.929; p = 0.017). Sensitivity analysis excluding one outlier study strengthened the effect (OR = 0.608; 95% CI: 0.454–0.814; p < 0.001) and reduced heterogeneity. Outcomes were influenced by weight loss magnitude, diabetes prevalence, and study quality. (40). These findings are further supported by a separate meta-analysis evaluating both the incidence and risk of atrial fibrillation following bariatric surgery. In this analysis of seven cohort studies involving 7,681 patients, the pooled incidence of AF post-surgery was 5.3% (95% CI: 1.9–13.8%) over a median follow-up of 7.9 years. Importantly, bariatric surgery was associated with a significantly reduced risk of AF compared to non-surgical controls (OR = 0.42; 95% CI: 0.22–0.83). These results reinforce the potential of bariatric surgery as an effective intervention not only for sustained weight loss but also for long-term AF prevention in individuals with obesity (41).

Regarding the pharmacological management of obese patients, a meta-analysis of 13 randomized controlled trials including over 30,000 overweight or obese individuals found that while GLP-1 receptor agonists significantly reduced major cardiovascular events, they had no clear effect on AF incidence (RR = 0.49; 95% CI: 0.17–1.43; p = 0.19) (42). Conversely, a meta-analysis of 10 randomized trials involving 12,651 high cardiovascular risk patients showed that semaglutide significantly reduced AF incidence by 42% compared to placebo (RR = 0.58; 95% CI: 0.40–0.85). The effect was consistent across subgroups, regardless of administration route, diabetes status, or BMI, suggesting a potential drug-specific benefit and variability within the GLP-1 RA class. However, further studies are needed to confirm these findings and clarify the mechanisms involved. (43).

However, not all lifestyle interventions show favorable effects on atrial structure. In a PREDIMED-Plus substudy, 534 overweight or obese individuals with metabolic syndrome were followed for five years to evaluate the impact of an intensive lifestyle intervention—

including a Mediterranean diet, physical activity, and behavioral support—on left atrial (LA) remodeling. Despite greater weight loss in the intervention group (-3.9 kg vs. -0.3 kg), no significant differences were observed in LA volume, stiffness, or function. Both groups showed similar progressive deterioration over time, suggesting that modest lifestyle-induced weight loss may be insufficient to reverse atrial remodeling in this high-risk population. Therefore, continued research is needed to better understand which interventions can effectively modify atrial substrate and reduce long-term AF risk (44).

Selected Clinical Trials Investigating Lifestyle and Metabolic Interventions in Atrial Fibrillation

Trial Number	Description	Primary Outcome
NCT03713775 (45)	Randomized controlled trial (RCT) investigating whether a structured weight loss and risk factor management program reduces AF burden in obese patients (BMI ≥27 kg/m²) compared to standard care.	Atrial fibrillation symptoms severity using Toronto AF Severity Scale (AFSS) score after 8 months.
NCT05600829 (46)	RCT assessing whether adding AF-specific weight loss therapy to cardiac rehabillitaion improves rates of ≥10% weight loss versus rehab alone in patients with AF and obesity.	Proportion of patients achieving ≥10% body weight change at 52 weeks
NCT04560387 (47)	RCT assessing impact of a structured weight loss program (including lifestyle counseling and optional bariatric surgery) on AF burden post-ablation in obese patients vs. standard care.	Atrial fibrillation burden expressed as % of total monitoring time at final visit (18 months)
NCT06401148 (48)	Pilot RCT assessing feasibility and acceptability of supervised exercise and psychoeducation in patients awaiting AF ablation.	Feasibility outcomes: screening, eligibility, CR uptake, adherence, and dropout rates; acceptability assessed via patient focus groups and clinician interviews.

NCT03397602 (49)	Randomized controlled trial comparing high-intensity interval training, moderate-intensity continuous exercise, and standard care on exercise capacity and quality of life in patients with persistent or permanent atrial fibrillation.	Changes in quality of life (SF-36 PCS) and exercise capacity (VO_2peak via CPET) from baseline to 12 weeks..
NCT03603912 (50)	Randomized trial assessing the effect of metformin and lifestyle modification on atrial fibrillation burden and progression in patients with implantable cardiac devices and recent AF episodes.	Composite of change from baseline to 1 year of average daily AF burden % after a 3 month blanking period and survival at 1 year.

Conclusions

In conclusion, the accumulating body of evidence indicates the key role of lifestyle modification, specifically weight loss and exercise in the prevention and long-term management of atrial fibrillation. Obesity is not just an associated condition but a significant, modifiable cause of AF initiation, worsening, and recurrence. The consistent observation that intentional weight reduction, especially losses of >10% of initial body weight, leads to substantial control of the arrhythmia, symptom burden, and quality of life is an argument strongly supporting the restoration of weight control to its therapeutic cornerstone role rather than as an adjunctive optional action. This is also the case in the context of the so-called "obesity paradox," in which higher BMI has been linked to better survival among established AF patients. Nevertheless, targeted weight loss certainly improves rhythm and functional status, emphasizing the complex but modifiable interplay between atrial electrophysiology and body composition.

Likewise, exercise has preventive and therapeutic roles in AF, though they are complex. In general, moderate-intensity exercise has been shown to be highly protective against AF initiation and recurrence, but extreme endurance training, particularly in male athletes, is associated with increased risk. The observations confirm that exercise prescription needs to be individualized, fostering sustainable levels of activity that are aligned with the patient's health status, comorbidities, and personal goals.

Combined, these data suggest a paradigm shift in AF management involving risk factor modification, exercise-guided interventions, and behavioral therapies as equal partners to anticoagulation, rate control, and rhythm control. Cardiologists, nutritionists, exercise physiologists, and psychologists collaborating to provide a multidisciplinary health care model may offer the greatest benefits, particularly to obese patients, patients with metabolic syndrome, and patients with AF burden. Study designs for future studies should consider heterogeneity of response, preferences, and physiological profiles of patients to implement and individualize lifestyle interventions.

Clinicians can significantly improve outcomes in an increasingly large and diverse population of patients if they fully utilize the therapeutic potential of lifestyle change.

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Author Contribution: Conceptualization, SP, and MS; methodology, MS; software, SP; check, MS; formal analysis, SP; investigation, SP, MS; resources, SP; data curation, MS; writing - rough preparation, SP; writing - review and editing, MS, SP ; visualization, MS; supervision, SP; project administration, SP;

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