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REVIEW ARTICLE

ATRIAL FIBRILLATION AND EXERCISE

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ABSTRACT

Atrial fibrillation (AF) is common in adults and the cardiologist and other health professionals, such as physical therapists and physical education teachers, are often faced with situations in which exercise prescription for this population becomes challenging. This article proposes, through an extensive literature review, to address AF in its epidemiological aspects, its pathophysiology, aspects of pharmacological treatment, repercussions of exercise as a trigger for the onset of AF and its effect as part of the treatment. of this arrhythmia. The article will also offer suggestions for an approach through exercise in individuals with AF who seek cardiovascular rehabilitation programs.

KEYWORDS: ATRIAL FIBRILLATION; REHABILITATION; CARDIOLOGY; EXERCISE THERAPY

INTRODUCTION

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia in adults worldwide and is associated with significant morbidity and mortality, in addition to representing an important health problem, with high consumption of financial resources. Currently, the estimated worldwide prevalence of AF in adults varies between 2 and 4%. Studies show that the incidence and prevalence of AF has increased in the last 20 years and will continue to increase in the next 30 years, making it one of the biggest epidemics and public health challenges [1,2].

In Brazil, according to the Brazilian AF Guidelines, about 1.5% of the Brazilian population has this arrhythmia. The trend towards an increase in new cases of AF over the years is notorious. A 2 to 3 fold increase is expected in the coming decades, largely due to population aging and the increase in the prevalence of diseases such as systemic arterial hypertension (SAH), coronary artery disease (CAD), obesity and diabetes mellitus (DM) [2].

The evolution of technology in medicine has provided, in recent years, optimization in the diagnosis and treatment of this arrhythmia; however, mortality and morbidity in patients with AF are still high. Of all patients with ischemic stroke, 20 to 30% have AF. Left ventricular dysfunction and heart failure (HF) have also been reported in patients with AF with high prevalence, reaching 56% in patients with permanent AF [2].

Thus, it is important to emphasize that individuals affected by this arrhythmia are associated with AF repercussions, such as increased mortality and increased risk of sudden death; Increased risk of HF; Insanity; Risk of stroke increased by 5 times, with more serious events; Symptoms such as fatigue and exercise intolerance, compromising health-related quality of life [2].

The practice of regular physical exercises, through the improvement of cardiorespiratory fitness, can reduce or delay the onset of atherosclerotic events and cardiovascular diseases, as well as reduce the incidence of coronary heart disease. However, there are still limited data on cardiovascular rehabilitation, especially with regard to intensity, for patients with AF [3].

METHODOLOGY

This article is an integrative literature review, carried out by searching Pubmed, Lilacs, Scielo and Medline databases, using the following keywords as search criteria: Atrial Fibrillation; Rehabilitation; Cardiology; Exercise Therapy; and their Portuguese equivalents. The period selected for searches was from 2013 to 2021. An article from 1993 was used due to its historical relevance on the topic (Ueshima et al) [12]. Two book chapters also served as instruments for gathering information in writing this update [3,4].

PATHOPHYSIOLOGY OF AF

AF is characterized by being a supraventricular tachycardia with uncoordinated and disorganized atrial electrical activation, in a fibrillatory and non-linear manner, leading to ineffective atrial contraction. Due to the alteration in the atrial tissue, one or more foci of autom-

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atism – triggers or micro-reentry – fire at a high frequency, causing fibrillation of the atria. Multiple reentry circuits in the structure of the atria block and conduct the waves that perpetuate fibrillation. Repeated episodes of AF lead to a process of cellular adaptation that ends up facilitating the maintenance of fibrillary conduction [3].

AF DIAGNOSIS

Documentation of the electrocardiogram (ECG) is necessary to establish the diagnosis of AF. A twelve-lead pattern ECG recording or a 30-second single-lead tracing showing altered atrial activation with a heart rhythm with no identifiable P waves and irregular RR intervals (when atrioventricular conduction is not impaired) is a diagnostic criterion for clinical FA. The QRS complex is irregular and small undulations in the baseline with different amplitudes and morphologies replace the P waves [3]. On physical examination, an irregular arterial pulse and absence of an A wave in the jugular venous pulse are indicative of AF. Considering the clinical history, a history of heart disease should also be investigated, especially when there is suspicion of arrhythmia (unproven AF), with recurrent paroxysms, in which the crisis episodes were very short, and it was not possible to record the ECG. . In the follow-up of AF, Holter monitoring and echocardiography are also suggested to evaluate the evolution/control of the disease. [4] Figure 1 shows the ECG of an individual with AF.

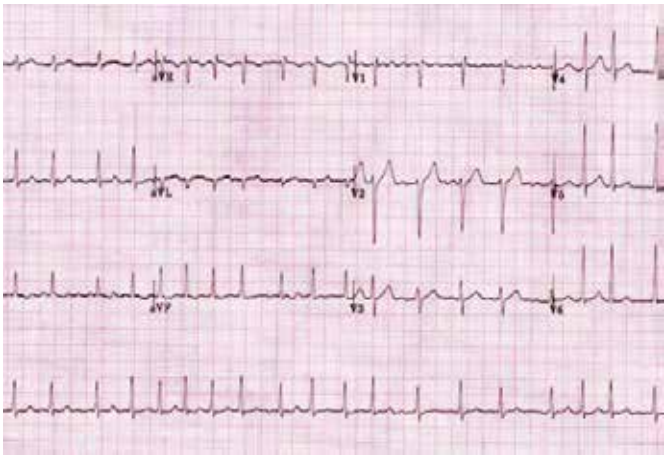

Figure 01. ECG of an individual with AF, with heart rhythm without identifiable P waves and irregular RR intervals.
Source: Authors' personal archive.

Various cardiac and non-cardiac alterations can cause electrophysiological disturbances and affect the atrial myocardium. As a result, the atrial muscle hypertrophies and starts to present contractile dysfunction, arrhythmogenic alterations in the transport and function of ion channels, increased sympathetic and thrombogenic activity and sympathetic discharge [3,4,5].

Systemic arterial hypertension can lead to pressure overload, inducing physiological changes observed in AF, such as myocyte hypertrophy, and atrial stretching and dilation. The reduction in atrial contractility and fibrosis of the atrial muscle, observed in heart failure (HF), promote greater neurohumoral activation, sympathetic hyperactivity and atrial dilatation, which are also precipitating factors for arrhythmias. Controlling blood pressure and treating HF can act in the prevention of atrial remodeling, oxidative stress and increased sympathetic activity, with a consequent reduction in the risk of developing AF [3,4,5].

AF frequently occurs in individuals with predisposing factors, such as those of genetic origin, continuous use of alcohol, and changes in the autonomic nervous system, of vagal or adrenergic origin, triggered by exercise or emotion.[6].

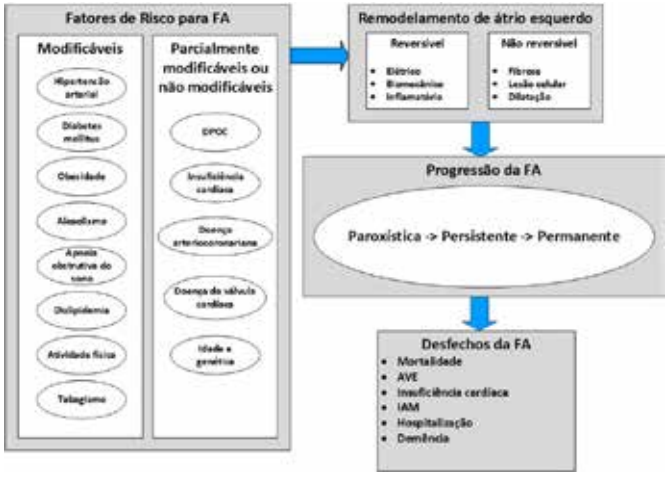

Figure 2: Main risk factors, outcomes and management of AF. COPD= Chronic Obstructive Pulmonary Disease; CVA= Cerebrovascular accidents (stroke); AMI = Acute Myocardial Infarction
Source: Adapted from Hindricks G, Potpara T, Dagres N, Arbelo E, Bax JJ, Blomström-Lundqvist C, et al; ESC Scientific Document Group. 2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS). Eur Heart J. 2021 Feb 1;42(5):373-498 [6]

AF CLASSIFICATION

Regarding its classification, AF can be defined according to the duration of the arrhythmia, symptoms or its clinical presentation and possible pathophysiology. [6].

By its duration it is classified as:

- Single-episode AF: When presented only once
- AF that ends spontaneously within seven days: paroxysmal
- AF lasting more than seven days: persistent (including episodes terminated by cardioversion)
- AF that lasts for more than one year: long-term
- Long-standing AF not responding to cardioversion: permanent.

The classification "permanent" should not be used in the context of a rhythm control strategy with antiarrhythmic drug therapy or ablation. If a rhythm control strategy is adopted, the arrhythmia will be classified as long-term persistent.

The evolution of paroxysmal to non-paroxysmal AF (or from subclinical to clinical AF) is observed by the advancement of atrial structural remodeling or worsening of atrial cardiomyopathy, which includes atrial architectural, contractile and electrophysiological changes with relevant clinical manifestations. The pathophysiological classification and clinical presentation of AF can assist in the stratification, treatment and prognosis of the disease [6], as described in Chart 1.

Chart 1. PATHOPHYSIOLOGICAL CLASSIFICATION OF ATRIAL FIBRILLATION

TYPE OF ATRIAL FIBRILLATION	CLINICAL PRESENTATION	PATHOPHYSIOLOGY
AF secondary to heart disease	Patients with left ventricular systolic or diastolic dysfunction or other structural heart disease. Common cause of hospitalization and predictor of poor outcome.	Elevated left atrial pressure, with remodeling and fibrosis, along with activation of the sympathetic system and renin angiotensin
Focal AF	Secondary to repetitive high-frequency atrial tachyarrhythmia and short episodes of paroxysmal AF, typically occurs in young people without heart disease	Arrhythmogenic triggers from the pulmonary veins can lead to progression of supraventricular tachycardias to AF
Postoperative AF	Occurs after major surgery (especially cardiac) in patients with no previous history of AF	Inflammation, oxidative stress, hydro-electrolyte disturbance, high sympathetic tone, and volume overload as precipitating factors
AF in athletes	Paroxysmal AF in high performance athletes, related to training intensity	Increased vagal tone and cardiac remodeling
Polygenic AF	AF in carriers of genetic variants	The presence of genetic variants may be associated with AF and influence treatment
Monogenic AF	Patients with hereditary cardiomyopathies including channelopathies	Include arrhythmogenic mechanisms responsible for sudden death

Source: Adapted from Hindricks G, Potpara T, Dagres N, Arbelo E, Bax JJ, Blomström-Lundqvist C, Boriani G, Castella M, Dan GA, Dilaveris PE, Fauchier L, Filippatos G, Kalman JM, La Meir M, Lane DA, Lebeau JP, Lettino M, Lip GYH, Pinto FJ, Thomas GN, Valgimigli M, Van Gelder IC, Van Putte BP, Watkins CL; ESC Scientific Document Group. 2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS). Eur Heart J. 2021 Feb 1;42(5):373-498 [6]

The most recurrent symptoms reported by patients with AF are fatigue, dyspnea, palpitation, syncope and dizziness. The goals of treating this arrhythmia include alleviating such symptoms, preventing stroke and controlling associated factors such as high blood pressure, obesity (very associated with sleep apnea) and diabetes mellitus, which can increase the rate of recurrence of AF. and the mortality rate in these patients [6].

AF can also be classified based on its symptoms, as shown in Chart 2.

Chart 02. Classification of symptoms caused by AF.

Grade	Symptoms	Description
1	None	Asymptomatic patient
2a	Discreet	Activities of daily living (ADLs) are not affected
2b	Moderate	Do not interfere with ADLs, but symptoms bother
3	Important	Activities limited by the discomfort of symptoms
4	Incapacitating symptoms	Normal daily activity interrupted

Source: Adapted from Alves LS, Scanavacca MI, Chizzola PR, Guimarães GV. Physical exercise and atrial fibrillation in Exercise cardiology from the athlete to the cardiac patient. 4. ed. São Paulo: Manole; 2019 [4]

AF can have important hemodynamic consequences. During such an arrhythmic event, the atria cannot properly eject blood and do not contribute to the stroke volume, causing a reduction in cardiac output by about 20 to 30%. Irregular ventricular rate, usually high, also promotes a greater reduction in ventricular filling and stroke volume, which may contribute to the development or worsening of HF. [7].

The prevention of left ventricular dysfunction and heart failure are fundamental in the patient with AF and can be achieved through a rhythm and heart rate control strategy. Patients who present with AF can be entered into such a strategy according to their electrocardiographic clinical characteristics and response to previous treatments. [7].

Therapeutic approach to AF

The ideal heart rate (HR) target is much discussed when following HR control. The RACE II study [8] proposed two beats per minute control strategies (bpm):

- more rigid, with HR at rest less than or equal to 80, and below 110 bpm in moderate physical activity;
- more tolerant: with resting HR less than 110 bpm.

The frequency of hospitalization and adverse events was similar in the two groups; however, for patients with HF or who remain symptomatic in the more tolerant strategy, stricter HR control is recommended in order to avoid further left ventricular dysfunction due to its persistent elevation.

Classically, the drugs used to control HR are beta-blockers, calcium channel blockers and digoxin. Beta-blockers are used especially in the presence of increased adrenergic tone and in myocardial ischemia in the presence of AF. Patients with HF and ventricular dysfunction benefit from the use of beta-blockers for rate control.

The rhythm control strategy is associated with the use of pharmacological antiarrhythmic measures, usually associated with electrical cardioversion (EC). Beta-blockers are the drugs of choice when there is no contraindication. Attempting to restore this patient's sinus rhythm should only be performed based on the severity of symptoms, presence of comorbidities, probability of successful cardioversion, and time of AF diagnosis. Its main action is based on blocking adrenergic tone through competitive inhibition of catecholamine-beta

receptor binding. They are able to reduce the spontaneous depolarization ramp in the sinus node cells and conduction through the atrioventricular node, and also promote an increase in the refractoriness of the His-Purkinje system. [7,8].

EC involves the delivery of an electrical shock synchronized with the intrinsic activity of the heart, to avoid the induction of ventricular fibrillation that usually occurs when the shock impinges on the ascending phase of the T wave. The success of reversion to sinus rhythm depends on the baseline heart disease and the density of electrical current received by the atrial myocardium. It should be performed with the patient fasting and under anesthesia or adequate deep sedation. Starting electrical EC with higher doses of energy is more effective, resulting in fewer shocks and less cumulative energy. Initial single-phase shocks of 100 Joules are generally ineffective for the EC of the AF and it is recommended to start with energy equal to or greater than 200 Joules. For biphasic shocks, it is also recommended to start with the application of 100 Joules or more, especially in patients with long-standing AF. [2]

Controlled studies have not yet been able to demonstrate superiority of rhythm control (maintenance of sinus rhythm) when compared to the rate control strategy in terms of mortality and morbidity. Controlling HR in patients with HF seems to be able to reduce symptoms and improve quality of life with a lower rate of adverse events, especially in elderly patients. [7,8].

For adequate control of HR in patients with AF, it should be taken into account that both reduced values for HR and higher values can lead to different clinical consequences. Elevated HR (close to 110 bpm) can lead to increased symptoms of AF, increased risk of HF and stroke, as well as being linked to high costs for the health system. Lower HR maintenance is associated with adverse effects of rate-control drugs such as decreased exercise tolerance, onset of pro-arrhythmic events, and hepatotoxicity Lower HR is also associated with a higher rate of cardiac pacemaker implants and higher costs [8].

In association with risk factors, AF represents the main cardioembolic source, when compared to acute myocardial infarction (AMI) and valvular diseases. The formation of thrombi in the vascular beds or in the heart chambers themselves, in this arrhythmia, is related to multiple factors and make up Virchow's triad: atrial blood stasis, endothelial injury and hypercoagulability state (deficiency of anticoagulant molecules and excess of components responsible for anticoagulation). Complications resulting from such changes, present in AF, such as stroke, can have a great impact on the functional capacity of individuals and are associated with greater morbidity and mortality.[7,8]

According to national guidelines, in patients with AF, regardless of its classification (paroxysmal, persistent or permanent), the risks and benefits of anticoagulation should be evaluated. It is therefore necessary to weigh the risks in preventing bleeding and the benefits in controlling embolic events in patients with arrhythmias. Not all patients with AF develop such complications, evidencing that other factors must be considered to assist in clinical decision making. To stratify the risk of thromboembolic events in patients with AF, the CHADS2 score was created and, later, incorporating factors such as gender, presence of peripheral vascular disease and age, the CHA2DS2-VASc score. With categorization using the CHA2DS2-VASc score, patients classified as very low risk would not require anticoagulation[2]

Table 1 presents the CHA2DS2-VASc score, used to assess the risk of thromboembolic phenomena in patients with AF, even in an outpatient setting. Patients with a score of 0 have a low risk of thromboembolic events and therefore would not need antithrombotic medications. Patients with score 1 are at moderate risk and can be anticoagulated or antiaggregated, while patients with score 2 are at high risk and should be anticoagulated unless there is a contraindication.

CHA2 DS2 -VASc score used for risk assessment for thromboembolic events in patients with atrial fibrillation

CHA2 DS2 -VASc		SCORE
C	Cardiac insufficiency	1
H	Hypertension	1
A_2	Age (≥ 75 years)	2
D	Diabetes Mellitus	1
S^2	Transient ischemic attack or previous stroke	2
V	Vascular disease (previous AMI, peripheral artery disease, or aortic plaque)	1
A A_2	Age (65 to 74 years old)	1
Sc	Gender (Female)	1

Table 1: AMI = Acute Myocardial Infarction
Source: Adapted from II Brazilian Guidelines on Atrial Fibrillation[2]

AF ABLATION THERAPY

Invasive therapy of AF through catheter ablation may be considered in rate or rhythm control scenarios. [2] Refers to the technique of electrical isolation of arrhythmogenic foci located in the pulmonary veins that generate AF. Catheter ablation is indicated to improve symptoms and quality of life related to paroxysmal or persistent AF. Among the various techniques available, conventional, point-to-point radiofrequency ablation, with the aid of electroanatomical mapping, is currently the most used technique. [3].

EFFECT OF PHYSICAL TRAINING ON ATRIAL FIBRILLATION

Regular physical activity and physical training are identified as safe therapy in the prevention and control of cardiovascular diseases and there are several mechanisms that act to decrease cardiovascular risks. [9]

Figure 3 identifies the main cardioprotective effects of regular moderate to high intensity physical activity.

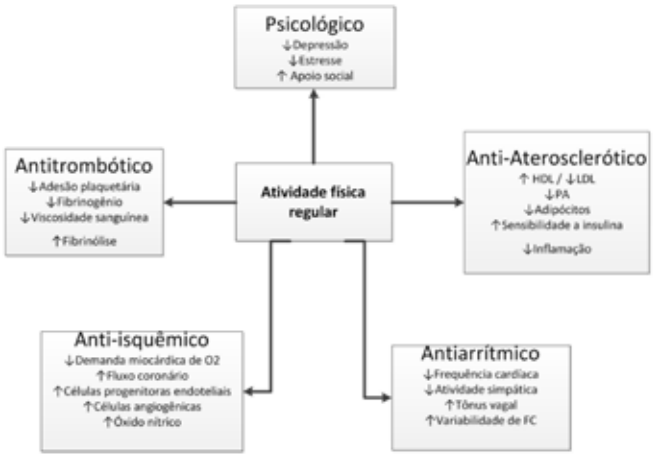

Figure 3: Main cardioprotective effects of regular physical activity
Source: Adapted from Franklin and collaborators (2020) [9]

At this point, it is important to differentiate between physical training and physical activity. Physical training refers to a subcategory of physical activity, being defined as any intervention planned and structured with the objective of maintaining or improving the individual's cardiorespiratory fitness and health. Aerobic capacity can be determined through cardiopulmonary tests with measurement of gas exchange (peak oxygen consumption) and estimation of work done [6, 10].

The risks of coronary and cardiovascular diseases decrease linearly in association with increasing levels of physical activity and physical training. The reduction in the risk of developing and worsening cardiovascular diseases, however, is shown to be about twice as high in individuals who show improvement in cardiorespiratory fitness achieved through physical training compared to the reduction observed with the increase in physical activity. [9,10]

Patients with AF have an altered hemodynamic response due to an irregular rhythm, usually with a rapid ventricular response at rest and during exercise. In individuals with AF, heart rate increases more in the initial phases of exercise, which may limit cardiac performance during exercise by compromising cardiac output (CO), associated with decreased atrial contribution, filling and reduced ventricular diastolic time. The association of these events seems to be responsible for the decrease in exercise tolerance observed in patients with AF when compared to healthy individuals.[11]

A positive impact is observed in reducing the incidence of AF or reducing the burden of AF (based, among other factors, on the reduction of symptoms perceived by the patient), with changes in lifestyle aimed at increasing cardiorespiratory fitness. The recurrence of AF and the severity of symptoms may be reduced in patients who increase their cardiorespiratory fitness through training programs compared to individuals who do not show a gain in aerobic interval training. [8,10,11]

Researchers have suggested that exercise tolerance in patients with isolated AF is similar to normal age-matched individuals. In a group of patients with AF it was reported that despite an increase in maximal and submaximal HR of about 25 beats on average when compared to healthy subjects, there was no difference in exercise capacity. On the other hand, higher lactate values during exercise were described in patients with AF when compared to healthy subjects, indicating early anaerobic metabolism and greater need for chemical and ventilatory buffering. [11]

To assess the response of patients with chronic AF to exercise, Ueshima et al [12] studied 79 men, aged around 64 years, who underwent echocardiography at rest and an ergospirometry. Patients were classified by heart disease into the following groups: isolated AF (n:17), AF + SAH (n:11), AF + CAD (n:13), AF + cardiomyopathy (n:26) AF + valvular heart disease (n: 13). Patients with morphologic disease had approximately 20% lower maximal oxygen consumption than patients with isolated AF or hypertensive AF patients without structural disease. The maximum oxygen consumption, considering the entire sample, was 20% lower than expected for a normal group of the same age. Although exercise capacity in AF is strongly influenced by the underlying disease, this reduction seems to be also intrinsic to the hemodynamic alteration that accompanies this arrhythmia.

In a study carried out to verify the impact of improved cardiorespiratory fitness on AF recurrence (paroxysmal or persistent) in 308 obese individuals, the authors observed that an increase of 1 MET in baseline cardiorespiratory fitness was associated with a 9% decline in the risk of recurrence of AF in the long term, regardless of the benefit conferred by weight loss alone. Such a decline was also due to favorable changes in cardiometabolic risk factors, inflammatory status and cardiac remodeling. The exercise program used was prescribed according to the FITT principle (frequency, intensity, time and type of exercise), taking into account age and cardiorespiratory fitness by estimating metabolic equivalents (MET) [13].

Systolic blood pressure (SBP) responses to the exercise test have been similar to those observed in individuals in sinus rhythm (progressive increase according to the effort performed). SBP in isolated AF is significantly higher than in AF with associated heart disease [11, 12]. In general, studies support blood pressure control as it becomes a strategy to reduce the risk of stroke and other cardiovascular events in patients with AF.

Patients with AF are typically anticoagulated because of the increased risk of ischemic events secondary to thrombus formation and embolism. Thus, it is important to remember that they should not be encouraged to practice physical contact sports, due to the increased risk of unwanted bleeding. It should also be noted that this population benefits from the practice of adequate physical ac-

tivity, and should not be excluded from rehabilitation protocols for fear of complications.

ATRIAL FIBRILLATION IN ATHLETES

Endurance athletes are at increased risk of cardiac arrhythmias, especially AF. Data suggest that the relationship between exercise volume and cardiovascular events presents a curve behavior similar to the letter U, in which moderate to high doses are able to confer cardiovascular protection and health benefits, however, when performed at a very intense volume they are associated with a higher risk for AF [14].

In a study that sought to identify different mechanisms of atrial fibrillation, 144 athletes and non-athletes were evaluated using echocardiography to measure left atrial and ventricular volumes and ventricular function. The authors observed that athletes had low atrial tension and, when associated with AF, had increased left atrial volumes and reduced atrial emptying. In this aspect, AF in athletes can be triggered by atrial myopathy resulting from exercise-induced stretch from increased cardiac output. [15].

The mechanisms responsible for the increased prevalence of AF among athletes are still poorly understood. It is known that physical exercise performed in an exaggerated manner can trigger AF and the presence of risk factors such as atrial ectopia, increased vagal tone, atrial dilatation, and atrial stretch due to high exercise volumes may be associated with the development of AF in athletes. Practitioners of intense resistance physical activity, such as cyclists and runners, are 2 to 10 times more likely to develop AF. [15].

High-intensity exercise (greater than 2000 hours of training or 20 years of training) was strongly associated with increased risk of AF, while moderate-intensity exercise appears to reduce the risk [16]. In a non-linear regression meta-analysis, including 19 studies in patients with AF, it was observed that individuals who performed 5 to 20 MET-hours of training per week had a significantly lower risk of developing AF, while highly active individuals (55 MET - hour per week) tended to increase the risk of developing AF [17].

Figure 4 shows the dose-response association between physical activity volume and relative risk for developing AF. Higher volumes of physical activity (above 55 MET-hours/week) appear to be associated with an increased risk of AF.

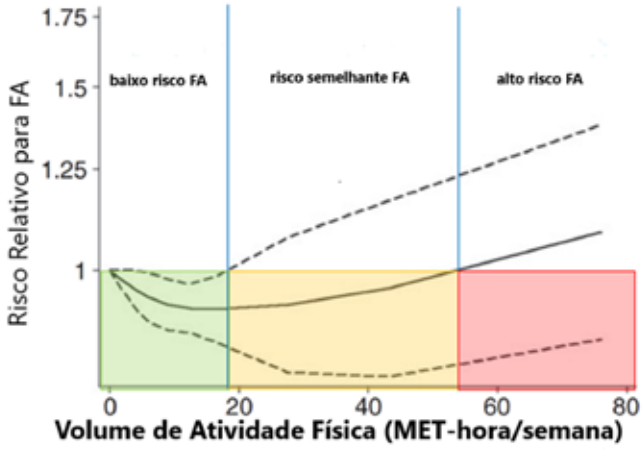

Figure 4: Dose-response association between physical activity volume and relative risk for AF
Source: Adapted from Ricci and collaborators, 2018 [17]

High training volumes and vigorous intensities are both associated with cardiac maladaptives including left atrial remodeling, accelerated coronary artery calcification, exercise-induced release of biomarkers, and myocardial fibrosis enabling the emergence of AF.

EXERCISE PRESCRIPTION FOR PATIENTS WITH AF

It is known that the practice of moderate physical activity can reduce the risk of developing AF and reduce its recurrence, but there are still few studies that address the prescription of exercise for patients with AF.

In order to evaluate the effects of a cardiac rehabilitation program, researchers recruited 51 patients with symptomatic AF (paroxysmal or persistent) determined by electrocardiogram (ECG). Patients were divided into two groups (control and intervention group) to perform three sessions per week of walking or running on the treadmill for 12 months. The protocol of 60 – 70% of maximum cardiac HR was used followed by 4 intervals at 85 - 95% of maximum cardiac HR. The Borg scale was used to assess intensity. The results of the study demonstrated a reduction in arrhythmic load in patients with AF who underwent cardiac rehabilitation. Such results also accompany improvement in maximal exercise capacity, left atrial and ventricular function, lipid levels and improved quality of life. [18].

In another study, carried out with 119 patients who had persistent AF, the authors observed that previously active patients, who also had mild to moderate HF, were able to remain physically active when included in a rehabilitation program, contributing to the improvement and maintenance of their condition. Physical aptitude. The rehabilitation program included supervised physical exercise 2 to 3 times a week, with the final evaluation score of the study being physical exercise for at least 150 minutes per week of moderate intensity (3-6

MET) or 75 minutes. vigorous intensity (greater than 6 MET). However, no benefit was observed in maintaining sinus rhythm, and there was no improvement in the quality of life score of these patients. [19].

In a 12-week randomized clinical trial, 76 patients with paroxysmal and persistent AF were allocated to low- or high-intensity exercise (50% and 80% of maximal perceived exertion). The results were observed according to the change in maximal oxygen consumption (VO2 max) and number of hospitalizations in one year. High-intensity physical exercise was not superior to low-intensity exercise in reducing atrial fibrillation burden. High-intensity exercise was well tolerated by individuals with AF, with no higher risk observed compared to low-intensity exercise. [20]

These results support the theory that interventions aimed at lifestyle and risk factor control should be valued in the management of patients with AF. Patients with atrial fibrillation tend to adopt a sedentary lifestyle due to fear of exercise-induced AF episodes. Moderate-high intensity physical activity is recommended to prevent and control cardiovascular diseases, including AF, but the effect of exercise intensity on the onset and burden of atrial fibrillation still needs to be studied further.

SUGGESTION FOR EXERCISE PRESCRIPTION IN PATIENTS WITH AF

In patients with AF, according to current evidence, a prescription of moderate-intensity aerobic exercise should be performed, aiming to achieve a minimum of 90 and a maximum of 150 minutes of activity per week, preferably divided between 3 and 5 sessions per week. It is important that periodic assessments of the individual's functional capacity are carried out to readjust the training.

HIGHLIGHT

In the authors' experience, a regular exercise program can be smoothly started or resumed after about 30 days when a successful catheter ablation procedure is performed to treat AF.

Chart 3. Moderate-intensity prescription methods for aerobic exercise

Method	*Description*
Subjective feeling of exertion (Borg)	Exercises with self-perceived exertion as moderate, medium or heavy, ranging from 3 to 5 on the Borg 0-10 scale or 10 to 13 on the 6-20 scale
Speech test	Execution of the exercises at an intensity in which breathing is panting, but controlled, so that a sentence can be completed without pauses
Peak HR percentages	Exercises at intensity between 70 and 85% of peak HR* target HR = peak HR x percentage
Reserve HR (Karvonen)	Exercises at intensity between 50 to 80% of HR reserve (HR peak – HR rest)* target HR = resting HR + reserve HR) x percentage
Thresholds in cardiopulmonary exercise testing	Execution of exercises at the intensity between ventilatory thresholds 1 and 2 (anaerobic threshold and respiratory compensation point)
HR: heart rate. * The use of peak HR obtained in a maximal exercise test is preferred, since there are individual variations that cause errors in the prediction of HR by age, especially in patients using medications with a negative chronotropic effect (beta-blockers)	

Source: Adapted from the Brazilian Cardiovascular Rehabilitation Guideline (2020)**(21)**

Chart 3 illustrates suggestions for prescribing aerobic physical training in patients with AF
Source: Adapted from the Brazilian Cardiovascular Rehabilitation Guideline (2020)[21]

It is important to emphasize that in AF, the workloads and the subjective perception of effort can be used in situations where the HR is not a good control parameter. Often, individuals with AF find it difficult to use frequency meters to control the intensity of effort guided by HR, due to the greater appearance of interference in this device. Palpation of the radial pulse in order to monitor the training intensity can also be inefficient, due to the irregular rhythm of the beats.

In addition to aerobic physical training, resistance training should be incorporated into cardiovascular rehabilitation programs for patients with AF. To this end, we suggest the adoption of resistance training between 2 and 3 times a week, in addition to the aerobic training program.

Suggestion of prescription of resistance training

- 90 minutes per week, divided between 2 and 3 sessions.
- 50 to 80% of a maximum repetition or use of the OMNI-RES perception scale between 5 and 8
- 6 to 8 exercises per session, covering the main muscle groups.
- 2 to 3 sets per exercise, with 8 to 10 repetitions.

In addition, all lifestyle changes that are part of a comprehensive cardiovascular rehabilitation program should be emphasized in patients with AF, such as smoking cessation, alcohol consumption, diet, stress management, and others.

Few patients with AF participate in cardiovascular reha-

bilitation programs, despite the benefits of exercise in this population. It is assumed that the term "cardiac arrhythmia" arouses fear on the part of individuals with AF and the exercise professionals who will train them. Importantly, the rate of exercise-related adverse events is low, with one occurrence for every 11,452 minutes of exercise. It is essential, however, that patients with AF in rehabilitation programs maintain adequate pharmacological treatment. ([0

CONCLUSION

AF is a complex arrhythmia and understanding its mechanisms is important for cardiovascular physical therapists who intend to work with this population. Individuals with AF benefit from a regular exercise program, which should be prescribed at moderate intensity. Training should consist of aerobic training combined with resistance training for greater benefits. Large volumes of exercise for long periods are associated with a higher incidence of AF in practitioners. The prescription must be individualized. Control of cardiovascular risk factors such as hypertension, obesity and others is associated with improvement in AF.

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