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Review article

PAFIYAMA, description of a new clinical entity in middle-aged athletes and its long-term repercussions

PAFIYAMA, descripción de una nueva entidad clínica en atletas de edad media y sus repercusiones a largo plazo

PAFIYAMA, description d'une nouvelle entité clinique chez le sportif d'âge moyen et ses répercussions à long terme

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ABSTRACT

Introduction: Atrial fibrillation is the most common cardiac arrhythmia, the risk of which usually increases with age. This condition is commonly associated with major cardiovascular diseases and structural damage to the heart, while it is rarely seen in healthy young people. **Objective:** To analyze the growing evidence related to paroxysmal atrial fibrillation in healthy young or middle-aged endurance athletes. **Methods:** Literature on the topic of atrial fibrillation associated with strenuous resistance exercise was searched. Some 281 references were found with the terms included, but the 50 indexed articles that were considered most relevant were taken into consideration. **Analysis and integration of information**: Growing evidence indicates that paroxysmal atrial fibrillation may also be onset in endurance athletes, particularly young or middle-aged and otherwise healthy athletes. The use of the definition of a new syndrome based on the accumulated data in the literature is suggested: atrial fibrillation related to strenuous physical exercise under the acronym "PAFIYAMA". The objective diagnosis of this condition can be reached by meeting a number of putative diagnostic criteria, once common risk factors for atrial fibrillation and other underlying causes have been ruled out. **Conclusions**: Growing evidence suggests that atrial fibrillation associated with strenuous physical exercise can now be considered as a single clinical entity, tentatively defined as "PAFIYAMA".

Key words: exercise, arrhythmia, risk, treatment

RESUMEN

Introducción*: la fibrilación auricular es la arritmia cardíaca más común, cuyo riesgo suele aumentar con la edad. Esta condición se asocia comúnmente con las principales enfermedades cardiovasculares y daño estructural del corazón, mientras que rara vez se observa en personas jóvenes sanas.* ***Objetivo:*** *analizar la creciente evidencia relacionada con la fibrilación auricular paroxística en atletas de resistencia, jóvenes o de mediana edad y sanos.* ***Métodos:*** *se buscó la literatura sobre el tema de la fibrilación auricular asociada con el ejercicio de resistencia extenuante. Se encontraron unas 281 referencias con los términos incluidos, pero se tomaron en consideración los 50 artículos indexados que se consideraron más relevantes.* ***Análisis e integración de la información****: la creciente evidencia indica que la fibrilación auricular paroxística también puede ser iniciar en atletas de resistencia, en particular jóvenes o de mediana edad y por lo demás sanos. Se sugiere el uso de la definición de un nuevo síndrome basado en los datos acumulados en la literatura: fibrilación auricular relacionada con ejercicio físico extenuante bajo el acrónimo de "PAFIYAMA". El diagnóstico objetivo de esta afección se puede alcanzar cumpliendo una serie de criterios diagnósticos putativos, una vez que se han descartado los factores de riesgo comunes para la fibrilación auricular y otras causas subyacentes.* ***Conclusiones****: la creciente evidencia sugiere que la fibrilación auricular asociada con el ejercicio físico extenuante ahora puede considerarse como una entidad clínica única, que puede definirse tentativamente como "PAFIYAMA".*

Palabras clave*: ejercicio, arritmia, riesgo, tratamiento*

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RÉSUMÉ

Introduction: *La fibrillation auriculaire est l'arythmie cardiaque la plus fréquente, dont le risque augmente généralement avec l'âge. Cette affection est couramment associée à des maladies cardiovasculaires majeures et à des lésions structurelles du cœur, alors qu'elle est rarement observée chez les jeunes en bonne santé.* ***Objectif****: Analyser les preuves croissantes liées à la fibrillation auriculaire paroxystique chez les athlètes d'endurance jeunes ou d'âge moyen en bonne santé.* ***Méthodes****: La littérature sur le sujet de la fibrillation auriculaire associée à un exercice de résistance intense a été recherchée. Quelque 281 références ont été trouvées avec les termes inclus, mais les 50 articles indexés jugés les plus pertinents ont été pris en considération.* ***Analyse et intégration des informations****: De plus en plus de preuves indiquent que la fibrillation auriculaire paroxystique peut également apparaître chez les athlètes d'endurance, en particulier les athlètes jeunes ou d'âge moyen et par ailleurs en bonne santé. L'utilisation de la définition d'un nouveau syndrome basé sur les données accumulées dans la littérature est suggérée: la fibrillation auriculaire liée à un exercice physique intense sous l'acronyme "PAFIYAMA". Le diagnostic objectif de cette affection peut être atteint en répondant à un certain nombre de critères de diagnostic putatifs, une fois que les facteurs de risque courants de fibrillation auriculaire et d'autres causes sous-jacentes ont été exclus.* ***Conclusions****: Des preuves croissantes suggèrent que la fibrillation auriculaire associée à un exercice physique intense peut désormais être considérée comme une entité clinique unique, provisoirement définie comme "PAFIYAMA".*

Mots-clés: *exercice, arythmie, risque, traitement*

INTRODUCTION

Atrial fibrillation (AF) is the most common type of cardiac arrhythmia, [1] whose risk increases with age (it affects 9% of people over 65 years of age). In general, this condition is associated with major cardiovascular diseases, as well as structural damage to the heart. Its prevalence ranges from 0.5% in young patients to 5% in those aged 65 or over. [2] The prevalence of AF is usually higher in men than in women of the same age. [3] Low to moderate intensity resistance exercise (eg, brisk walking) prevents the onset of AF. [4] This type of exercise could also reduce AF-related symptoms, as well as mortality and morbidity in affected individuals. [5]

In contrast, long-term use of strenuous resistance exercise (eg, cycling, marathon running, cross-country skiing) has been associated with an increased risk of developing AF, particularly paroxysmal AF, in healthy young to middle-aged adults. [6] However, the prevalence of AF among athletes is quite variable, ranging from 0.3% to 12.8%, reflecting methodological biases such as differences between studies in subject age, training status or sports specialty. [7]

Another caveat in this field of research arises from the fact that the vast majority of studies have been conducted only in men. Although regular physical activity is associated with a lower risk of AF in women, [8] little is known about its prevalence among female endurance athletes. In a Swedish

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cohort, repeated participation in a 90 km cross-country ski race was associated with increased AF risk in men, but not in women, [9] and exercise induced hypertension in men could be a risk factor. However, a recent study in a Norwegian cohort suggested that prolonged strenuous physical exercise could also cause AF in women; those older than 40 years who engaged in strenuous physical exercise had a borderline-significant higher risk of self-reported AF compared with sedentary counterparts. [10]

AF is medically comparable to that occurring in the elderly and should not be treated equally. According to Turangam *et al.*, [11] although AF is the most frequent tachyarrhythmia in both groups: both endurance athletes and the general population (non-athletic), there are probable differences in the etiology and clinical presentation of this condition, likewise the treatment approach may also differ.

Hence, the purpose of this study is to review the growing evidence related to paroxysmal atrial fibrillation in healthy young to middle-aged endurance athletes.

SEARCH FOR EVIDENCE

The main scientific evidence search engines that included results in the last 5 years at the time of writing this document (2019-2023) were used. Some 281 references were found with the terms included, but the 50 indexed articles that were considered most relevant, in Spanish and English, were taken into consideration.

ANALYSIS AND INTEGRATION OF INFORMATION

Literature on the subject of AF associated with strenuous resistance exercise was searched and the definition of a new syndrome based on the accumulated data in the literature is proposed: AF related to strenuous physical exercise under the acronym of "PAFIYAMA" from its acronym *Paroxysmal Atrial Fibrillation In Young And Middle-Aged Athletes* .

Etiology

Although the pathophysiological mechanisms responsible for the arrhythmia present a number of cellular processes and bodily functions, including the heart, the occurrence of AF in athletes has not yet been clearly elucidated. Next, the available evidence (as far as the authors were able to review) on the responsible pathogenic substrate will be presented. A clear association between the increase in left atrial (LA) size induced by strenuous physical activity and the occurrence of AF has not been consistently reported. However, dilation/extension of the less muscular chambers of the atria potentially induced by long-term exposure to and higher blood pressure levels during exercise, with insufficient time for recovery between exercise bouts, could lead to microtrauma, inflammation and fibrosis, which are potential substrates for arrhythmias. [12]

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LA enlargement and atrial wall stretch could increase proportionally with lifetime training hours [13] or within the level of competition. There are implications of micro-RNAs in the pathogenic role of such arrhythmic events. Micro-RNAs (miRNAs) are small, non-coding regions of the genome that regulate a myriad of cellular processes and bodily functions, including the heart, and help rebuild ion channels by regulating gene expression in cardiomyocytes in the process of arrhythmia. Some miRNAs are important mediators of proarrhythmogenic remodeling (i.e., those involved in electrical and structural remodeling, atrial fibrosis, and calcium homeostasis, such as miR-1, miR-26a, miR-29b, miR-30a, and miR 133a), and therefore have been proposed as potential biomarkers of AF. [14]

Fibrosis could play a key role in the generation of supraventricular arrhythmias. [15] Although the development and progression of atrial fibrosis is considered the hallmark of structural remodeling in AF, and therefore a substrate for the perpetuation of AF; [16] This phenomenon has only been observed in two studies with animal models exposed to strenuous physical exercise (Wistar rats). [17] Sixteen weeks of strenuous physical exercise increased the expression of fibrosis, the levels of biomarkers in the atria and ventricles of rodents subjected to such activity compared to a control group. The fibrotic changes induced by strenuous physical exercise were reversed after an 8-day period of cessation of exercise. [17] Another study showed that 16 weeks of strenuous physical exercise increased atrial fibrosis in rats, a phenomenon that remained unchanged after detraining. [18]

Indirect evidence of strenuous exercise-induced atrial fibrosis in humans comes from a study by Lindsey *et al*., [19] which included 45 veteran athletes. Compared with sedentary controls, these individuals showed higher levels of three cardiac fibrosis biomarkers, propeptide plasma carboxyterminal type I collagen (PICP), telopeptide collagen carboxyterminus type I (CITP) and tissue inhibitor of matrix metalloproteinase type I (TIMP-1).

The link between cardiac inflammation and AF has been widely described. [20] Along with oxidative and metabolic stress, inflammation is, in fact, one of the major contributors to atrial electroanatomic remodeling and extracellular tissue formation, ultimately leading to the development of AF. [21] In particular, the migration of inflammatory cells and proinflammatory cytokines into the atrial myocardium can cause a proarrhythmogenic state which in turn could act as a precursor to AF. [22] Therefore, the circulating levels of proinflammatory markers and cytokines such as interleukin (IL)-1ÿ, IL-6, C-reactive protein (CRP) or tumor necrosis factor (TNF)-ÿ, among others, have been associated with AF, thus linking inflammation with its onset. [23]

In turn, both acute and marathon runners, as well as those subjected to (chronic) strenuous physical activity, also lead to an increase in the aforementioned biomarkers. [24] The risk of inflammation-related AF would be particularly higher in overtrained athletes. Conversely, low-moderate intensity resistance exercise would have the opposite beneficial effect. Aschar-Sobbi *et al*., [25] recently reported that TNFÿ is a key factor in the pathology of AF induced by strenuous physical exercise.

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In many athletes, AF occurs at night when vagal tone is most pronounced. This alteration is related to L-type calcium fibers. In addition, both the vagus nerve-induced decrease in refractory period and slower conduction shorten the excitation wavelength and therefore facilitate reentry, which induces the appearance of AF. [26]

In relation to the presence of atrial ectopia, particularly pulmonary vein ectopia, this behaves as a trigger for most episodes of paroxysmal AF in the general population, [27] its real participation in AF remains to be demonstrated. associated with strenuous physical activity, so more research is needed. Atrial and ventricular ectopy may increase as a consequence of regular strenuous physical exercise. [28]

Regarding the electrical remodeling of the sinoatrial node in athletes, to test the hypothesis that bradycardia induced by resistance training may be caused by an intrinsic change in the sinoatrial node, which in turn would be the result of a remodeling of the ion channels that govern the pacemaker, D'Souza *et al.,* [29] recently conducted an animal study comparing treadmill-trained versus sedentary rats, sedentary versus trained (swimming) mice, and sedentary controls; these used in vivo, in vitro, and transcriptomic analyses, as well as mechanistic approaches including ivabradine blockade of the most important component of the membrane clock, the funny current (If).

The authors demonstrated that training-induced bradycardia persisted after autonomic nervous system blockade in vivo in mice and in vitro in the denervated sinus node. They also demonstrated that widespread remodeling of pacemaker ion channels occurred with training, in particular a downregulation of cyclic nucleotide-activated hyperpolarization (HCN)4 and its corresponding ionic current, If. In turn, If blockade abolished the difference in heart rate between trained and sedentary animals in vivo and in vitro. Resistance training was also associated with the downregulation of miRNAs for various components of the calcium clock, such as RyR2. D'Souza 's Findings *et al.,* provide molecular insights into potentially pathological heart rate adaptation to regular resistance exercise that could lead to conditions such as atrial fibrillation. [29]

Atrial fibrillation, types and correlation with strenuous physical exercise

Atrial fibrillation can generally be divided into three forms: I) paroxysmal (automatically ending within 48 h); II) persistent (duration of at least 7 days or terminated by electrical or pharmacological cardioversion); and III) permanent (refractory to cardioversion). Athletes are more likely to suffer from the first condition. [30] One of the characteristics of paroxysmal AF episodes is that they occur at night or after meals in endurance athletes.

One of the first studies on AF in athletes was carried out by Karjalainen *et al*., [31] who reported a prevalence of AF of 5.3% in veterans who practiced strenuous physical exercise (age 47 ± 7 years). Grimsmo *et al*., [6] reported a higher prevalence of AF (12.8%) in older cross-country skiers (59-88 years). On the contrary, Peliccia *et al*., [12] reported a very low prevalence of paroxysmal AF of 0.3% in young elite endurance athletes (mean age 24 ± 6 years). Therefore, diagnostic efforts to identify

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AF associated with strenuous physical exercise should focus primarily on long-term endurance middle-aged (45-65 years) male athletes.

Endurance athletes also have greater ventricular diastolic chamber compliance and compliance than non-athletes, on the steep part of the Starling curve . [32] The aforementioned SEE-induced changes are currently considered to be physiological adaptations. Specifically, they tend to disappear with the cessation of training. In contrast to pathologic left ventricular hypertrophy, the septal wall thickness of healthy athletes decreases after only 3 months of cessation of training. Left ventricular (LV) cavity dimension returns to baseline levels after 1 to 13 years of cessation of physical training. [32]

Although LV end-diastolic diameter may remain elevated for up to 5 years after discontinuation of training, this dilation is not accompanied by impaired LV function or leads to adverse cardiac events. [33] Similarly, increased LV mass in athletes is almost always associated with a normal resting ejection fraction, whereas stroke volume is normal or increased. [34-37] These adaptations are known as the athlete's heart.

Although AF is commonly associated with a number of diseases, most of which can cause structural damage to heart tissue, such as hypertension or heart failure, when this condition affects middle-aged athletes, it is generally not associated with any structural heart disease known, but rather with the 'athlete's heart', as mentioned above.

The typical clinical profile of strenuous exercise-related AF is described as a condition affecting young to middle-aged male athletes (usually in their forties to fifties) with a long-term history of regular strenuous exercise period (more than 6 months) who are still doing this type of exercise regularly (more than 1 hour per day of running, cycling or cross-country skiing most, if not all, days of the week, usually at 60% intensity of maximum heart rate), or the metabolic equivalent (MET) of 8 METS.

This pattern of exercise based on what they practice is the central activity, and usually the favorite, of their free time, which creates a high degree of psychological dependence. AF is usually paroxysmal, that is, in the form of acute crises, initially very punctual and self-limited, which progressively increase in duration and intensity. AF episodes usually occur at night or after meals, revealing that AF may be related to increased vagal tone, although it can sometimes occur during strenuous physical exercise. Affected individuals present the typical characteristics of the athlete's heart [38] .

Since the main characteristics of these patients converge on a common profile, it is considered that paroxysmal AF in young and middle-aged athletes (acronym: PAFIYAMA). Unexplained AF in young and middle-aged athletes requires evaluation by expert specialists. A careful history should be taken of all possible contributing factors, including the frequency and duration of the episodes and their association with sporting activities. Medical conditions such as hypertension, coronary artery disease, hyperthyroidism, pericarditis, Wolff-Parkinson-White syndrome, hypertrophic or dilated

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cardiomyopathy, Brugada syndrome, long QT syndrome, or catecholaminergic ventricular tachycardia should be ruled out [39].

The use of alcohol, caffeine, supplements or sports performance-enhancing drinks (such as taurine-containing "energy" drinks), or drugs and sympathomimetics in cold medications should be investigated and eventually discontinued. Diagnostic testing should include thyroid function tests, echocardiography (to exclude structural heart disease) [38,39], and both baseline and exercise 12-lead electrocardiography (ECG).

In fact, treadmill tests can sometimes trigger AF, so ECG recording should continue for at least 6 minutes during the recovery period. Holter ECG is also recommended in cases of suspected but not yet documented AF episodes, as it has been estimated that the 7-day recording can document arrhythmia in approximately 70% of AF patients. [39] Once all the aforementioned tests have been carried out, if they are negative to make the diagnosis of PAFIYAMA syndrome, it is suggested to apply the following criteria.

PAFIYAMA diagnostic criteria [40]

Major criteria

1. Onset as paroxysmal AF

2. Age usually under 60 years, male

3. Prolonged practice of strenuous physical activity (ÿ 6 to 8 h/week with intensity greater than 60% of maximum heart rate, for more than 6 months)

4. Preserved ejection fraction (greater than 55%)

Minor criteria

1. ST segment elevation at J point (STE) ÿ0.1 mm in 2 leads 2. T wave inversion in 2 leads 3. Increased vagal tone (bradycardia

sinus syndrome, prolonged PQ time, first-degree AV block)

4. Left atrial enlargement by echocardiography

5. LV hypertrophy

6. Increased LV wall thickness and LV mass

7. Diastolic function normal or even higher than normal

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Absence of common AF risk factors

1. Not overweight (BMI N 25 kg/m2) or obese (BMI N 30 kg/m2)
2. No arterial hypertension at rest
3. No smoking habit
4. No diabetes

Other underlying causes to exclude

1. Metabolic or hormonal diseases (hyperthyroidism, pheochromocytoma).
2. Dilated or hypertrophic cardiomyopathy.
3. Pericarditis.
4. Coronary artery disease.
5. Wolff-Parkinson-White syndrome, Brugada syndrome, Long QT syndrome,

arrhythmogenic cardiomyopathy or catecholaminergic ventricular tachycardia.

6. 6. Performance-enhancing agents or use of illicit drugs.
7. Obstructive sleep apnea.
8. Electrolytic abnormalities.

Treatment

In the past it was recommended that the management of AF in athletes should follow similar principles to those used to manage AF in the general population. [38] However, previous studies demonstrated the reversibility of hypertrophic changes at the ventricular level in the hearts of athletes. Biffi *et al*., [41] showed a marked decrease in ventricular ectopy after cessation of sport. Therefore, pending more definitive data, it might also be useful to reduce strenuous exercise loads in order to minimize the substrate and triggers for physical activity-related AF. Thus, although not generally accepted by athletes, a reduction in exercise load (at least in terms of training volume) to reduce AF load [42] is accepted behaviour.

Furlanello *et al.*, [43] described a good response to sports abstinence in high-level athletes with AF. Monte *et al.* also noted that limiting exercise time appears to reduce the number of AF crises, particularly in those with recent onset and a minimally dilated atrium. Also Hoogsteen *et al*., [44] showed that up to 30% of athletes experienced fewer AF episodes with reduced sports activity. Therefore, the initial approach should probably be to recommend reducing levels of physical activity.

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According to the Sports Cardiology Study Group of the European Association for Cardiovascular Prevention and Rehabilitation [45] points out that Athletes with early-stage paroxysmal AF should stop training for 2 months to stabilize the rhythm and thus remain arrhythmia-free.

Reducing the volume of resistance training may not be enough to prevent AF and its recurrence in all cases. Based on recent American Heart Association (AHA) and American College of Cardiology (ACC) guidelines for eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities, [46] management options for AF in athletes include rate control or rhythm control. Rate control, while an option, may not be ideal for competitive athletes because this can obviously impair performance, which is the focus of more committed athletes, and also because of the inherent difficulty in ensuring adequate rate control speed during training and competition. Therefore, a rhythm control strategy is the preferred means of treatment in athletes. Class I and III antiarrhythmic drugs such as flecainide and amiodarone are also options for short-term benefit in symptomatic patients. While beta-blockers, Sotalol and Digoxin can be recommended in the long term. However, Propafenone is not recommended, as it could worsen the vagal reaction [46].

Antiarrhythmic drug therapy has efficacy, but also worrisome side effects, including proarrhythmic risk. Flecainide can be started as a ventricular rate control medication during exercise and is safe in the absence of structural heart disease [47].

The "pill in pocket" approach with class I drugs is usually used in athletes with paroxysmal AF, although sport activity should be limited in these patients until at least one half-life of the antiarrhythmic drug has elapsed, due to the proarrhythmic risk of events triggered by adrenergic hypertonia during exercise. Amiodarone may show greater efficacy in preventing AF episodes, but should be used with caution due to long-term side effects and impaired quality of life [47].

The possible long-term role of drugs used to prevent cardiac hypertrophy (converting enzyme inhibitors, angiotensin inhibitors, or beta blockers) remains to be elucidated, although angiotensin blockers appear to play a role in improving outcomes of cardioversion or ablation of AF [48].

Finally, the new drug Dronederone has yet to prove its effectiveness in athletes; however, due to its rate-slowing effect, it may not be as useful in this group of patients because it may impair athletic performance [48]. The need for anticoagulation is based on risk factors for stroke. In younger athletes without underlying structural heart disease, the risk of thromboembolism is low without treatment. The efficacy of aspirin for the primary prevention of stroke in relation to the risk of bleeding in AF alone has not been established. Long-term anticoagulation with a vitamin K antagonist, direct thrombin inhibitor, or activated factor X (FXa) inhibitor for the primary prevention of stroke in patients with isolated AF without risk factors for thromboembolism, especially in those athletes, is another option to assess [49].

For whom the risk of traumatic bleeding is high (such as in rugby players, cyclists, motor sports) the risks and benefits must be weighed or the sport activity changed. However, most athletes will be at

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low risk of systemic thromboembolism, as manifested by a low CHADS2 score or a CHA2DS2-VASc score of zero, so anticoagulation will rarely be necessary. If anticoagulants are used, athletes should be restricted from participating in high-impact contact sports due to the high risk of bleeding [49].

Because autonomic hyperarousal is important in the initiation and maintenance of AF in athletes, direct current electrical cardioversion may not be a definitive treatment and there may be a high risk of recurrence. Because autonomic hyperarousal is important in the initiation and maintenance of AF in athletes, the use of electrical cardioversion may not be a definitive treatment and there may be a high risk of arrhythmia recurrence [50].

Circumferential pulmonary vein ablation (PVAA) is increasingly viewed as the option with the most sustained benefit, particularly in those with paroxysmal AF in the absence of evidence of structural cardiac damage, which is generally for the majority of young and middle-aged athletes with AF [49]. However, longer-term observations are needed to determine the eventual sustainability of benefits over time. ACVP in healthy young patients and also in middle-aged people is highly effective and safe [50].

Recent data supports the efficacy of the ACVP strategy in athletes. Compared with controls, the endurance and non-endurance groups of athletes had a similar proportion of patients without arrhythmia assessed at 3 years after repeated ablation procedures. After a successful ACVP procedure and the absence of symptomatic recurrences for 3 months or more, the resumption of all sports activities seems justified, but athletes should be followed up every 6 months [50].

Some of them had undergone ACVP with a similar success rate to patients who do not practice resistance sports. furlanello *et al*., [50] described a highly successful ablation with 90% success after a mean of two ablation procedures in a series of 20 athletes without major complications. Apparently, the objective of the ACVP was to allow athletes aged between (44 ± 13 years) to restart their competitive activity.

The series reported may represent a selected series of patients since the majority presented AF induced by exercise, in contrast to the reported prevalence of vagal AF among endurance athletes.

Pulmonary vein catheter ablation, which prevents progression to persistent or even permanent AF, seems the best option for athletes with severe symptoms or for those who are determined to continue training for peak athletic performance.

Conclusions

Growing evidence suggests that AF associated with strenuous physical exercise can now be considered as a single clinical entity, which can be tentatively defined as "PAFIYAMA" ('paroxysmal AF in young and middle-aged athletes'). The objective diagnosis of this condition can be reached by fulfilling a series of putative diagnostic criteria (major and minor); once common risk factors for AF and other underlying causes have been ruled out. Due to its peculiar pathogenesis, clinical

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management may also differ from other forms of AF, and should basically consist of reducing the volume of exercise, the administration of antiarrhythmics such as flecainide and amiodarone, as well as catheter ablation in selected cases.

In general, whatever the final treatment, it should be considered that the therapy should be primarily aimed at antagonizing AF, but it should also be compatible with the possibility of maintaining at least a moderate degree of physical activity that produces an impact favorable in the physical and mental state, as well as the well-being of the affected subject.

The evidence available in Latin America on the subject is scarce, and more research should be carried out in this regard and clinical practice guidelines should be developed at the national level. Primary Care physicians must be related to this entity, which may be underestimated due to the age of presentation in patients, but with a potential for complications and morbidity and mortality that is not insignificant.

Abbreviations: AF: atrial fibrillation: AV, atrioventricular; BMI: body mass index; EF: ejection fraction; LA: left atrium; LV: left ventricle; SEE: strenuous resistance exercise; STE: ST segment elevation.

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CONFLICTS OF INTEREST

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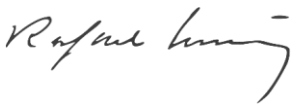 Rafael Guillermo Parales Strauch	https://orcid.org/0000-0002-7887-5611

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