Exploration of Cardiology

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Open Access Mini Review

Endurance sport and atrial fibrillation: a mini-review of a complex relationship

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Academic Editor: Hassane Zouhal, Rennes 2 University, France
Received: February 13, 2025 **Accepted:** April 14, 2025 **Published:** July 30, 2025

Cite this article: Costantini M, Elizari MV, Previtali M, Sciarra L, Luzza F, Costantini L, et al. Endurance sport and atrial fibrillation: a mini-review of a complex relationship. Explor Cardiol. 2025;3:101267. https://doi.org/10.37349/ec.2025.101267

Abstract

Scientific evidence seems to indicate that, in males, intense and prolonged endurance sport can favor the onset of atrial fibrillation. A plausible explanation may be the impact that intense endurance sports produce on the three vertices of Coumel's triangle. However, genetics is probably also involved in translating this impact into an arrhythmic phenotype. On a management level, the first task of the cardiologist is to exclude the presence of structural heart disease, channelopathy, endocrine and/or electrolyte disorders, and substance use. As for the treatment of arrhythmia, the "CARE" paradigm proposed by the latest ESC guidelines should probably be accompanied by detraining, although this suggestion is often rejected by the athlete. Anticoagulant therapy, where indicated, must take into account the risk of trauma that the sport entails, even if the particular pharmacodynamics/pharmacokinetics of DOACs should allow training/competition to take place when the anticoagulant effect of the previous administration has completely or almost completely worn off.

Keywords

Atrial fibrillation, endurance sport, Coumel's triangle

Introduction

Hypothesizing an etiological relationship between sport and atrial fibrillation (AF) may seem paradoxical, since strong evidence indicates that, in addition to bringing a significant reduction in cardiovascular risk factors and all-cause mortality [1], physical activity is a cornerstone of both primary and secondary prevention of AF [2–4]. Physical activity is even considered an integral part of treatment in subjects with permanent AF, since it assists in rate control, improves quality of life, reduces thrombogenesis, improves endothelial function, and promotes positive atrial remodeling [5, 6]. Yet, it should be noted that a substantial number of works appearing in the literature have associated endurance sports, characterized by isotonic-dynamic muscular activity, predominantly powered by aerobic energy (e.g., marathon running, cross-country skiing, cycling, swimming) with the risk of AF, and this has raised doubts about the real benefits of endurance with respect to the prevention of arrhythmia.

Available data

Studies by Karjalainen et al. [7], Mont et al. [8], Molina et al. [9], Elosua et al. [10], Heidbüchel et al. [11], and Baldesberger et al. [12] unanimously report that endurance sports can favor the onset of AF in middle-aged people. However, many of these studies have attracted criticism over the small number of subjects, because they were mostly retrospective, or because they were flawed by questionable enrollment criteria [13]; not to mention the fact that other studies [2, 14–16] have yielded opposite results. Evidence from subsequent important studies substantially supported the observation that intense physical activity could, in fact, act as a risk factor for AF. These include an American study based on data from the Physician Health Study, which enrolled approximately 17,000 males. The results showed a correlation between the dose of physical exercise and the incidence of AF [17]. In another study, Calvo et al. [18] demonstrated that the incidence of lone AF is higher in athletes with an intense endurance curriculum exceeding 2,000 hours. A Swedish study that enrolled 1.1 million male subjects, following them for approximately 26 years, demonstrated a U-shaped trend in the risk of AF as the level of physical activity increased [19]. A meta-analysis by Newman et al. [20] showed that in athletes and non-athletes without cardiovascular disease risk factors (i.e., diabetes mellitus and hypertension), athletes had a significantly greater relative risk of AF and that younger athletes (< 55 years) had a significantly higher relative risk of AF than older athletes. Another meta-analysis by Ayinde et al. [21] confirmed that the risk of AF is significantly higher in younger athletes (< 54 years) than in older athletes. What is striking about these studies is that the subjects enrolled are almost always male. Surprisingly, studies [22, 23] and meta-analyses [24, 25] including equal numbers of men and women reveal completely opposite trends for the two sexes: the incidence of AF continues to decrease in females even when they perform intense physical activity. Accepted by the guidelines of various scientific societies [26, 27], these data are part of the many substantial arrhythmological differences between males and females (see Table 1).

Table 1. Arrhythmological differences between the two sexes

In females ↑	In males ↑
Basal sinus rate	Atrio-ventricular reentry tachycardia
Inappropriate sinus tachycardia	Atrial fibrillation in athletes
AV nodal re-entry tachycardia	Distal AV block
Aquired long QT syndrome	Sudden cardiac death
Torsade de pointes	
Ischemic stroke risk related to atrial fibrillation	
Sick sinus syndrome	

The most significant arrhythmological differences between the two sexes are reported. In females, the “basal” sinus frequency is generally higher than in males. Inappropriate sinus tachycardia is an arrhythmological condition almost exclusively of the female sex. In women, the incidence of AV nodal reentry tachycardia is higher than in men, as is the incidence of acquired long QT syndrome and—consequently—the risk of torsade de pointes. In women affected by atrial fibrillation, the risk of ischemic stroke is higher than in men. Finally, the incidence of sick sinus syndrome is higher in women than in men. In men, the incidence of atrioventricular reentry tachycardia involving an accessory pathway, atrial fibrillation in athletes, distal atrioventricular block, and sudden cardiac death is higher

Mechanisms involved

The higher incidence, shown in the data and accepted by the scientific community, of AF in males who practice intense endurance requires a scientifically plausible explanation. It is well documented that AF is an arrhythmia due to the chaotic multiple reentry of the impulse within the atrial musculature [28], propitiated by a critical level of interaction between a substrate (the atrial muscle mass) and by modulation of the same substrate by functional factors (vagus/sympathetic), in the presence of a trigger mechanism (generally represented by atrial ectopic beats, often starting from the pulmonary veins). AF, therefore, paradigmatically embodies Coumel's concept of the "arrhythmia triangle" [29], and it has been scientifically demonstrated that intense endurance sport consistently impacts all three vertices of this iconic triangle:

1) It impacts the substrate (atrial dilation, atrial fibrosis, inflammation, cellular damage, and electrical remodeling [30–32]).

2) It impacts the functional modulation of the same substrate (increased vagal activity at rest; increased sensitivity to acetylcholine; reduction of refractory periods of atrial cells; dispersion of atrial refractoriness, and increased sympathetic tone during exercise [4, 33]).

3) It impacts trigger factors (increased atrial ectopias; often other atrial arrhythmias can trigger AF [4, 33]).

It should also be considered that, according to recent studies, vigorous and prolonged endurance leads to an increase in the level of circulating microRNA [34]. As is known, microRNA is a regulator of gene expression and takes an active part in cellular differentiation, growth, and apoptosis [35], while from the cardiovascular point of view, it is closely involved in physiopathological processes such as hypertrophy, fibrosis, and myocellular damage [36]. Changes in microRNA levels, demonstrated in endurance athletes, can favor atrial fibrosis, making the substrate more favorable to the onset of AF [34].

Another recent acquisition that could be important in this context is the demonstrated presence of an electromechanical feedback cycle in the human heart, on the basis of which excitability, conduction and activity of membrane ion channels are influenced by the "mechanical environment" in which they are situated [37]. The medium of this feedback is the "mechano-gated ion channels", at the same time sensors of mechanical activity and effectors of electrical responses, by means of interactions with the ion channels responsible for the electrical activity of the membrane. The strong impact of intense physical activity on the "mechano-gated ion channels" can modulate proarrhythmic electrical feedback and thus favor the onset of AF [38].

Finally, the risk of AF related to endurance is probably modulated by genetic factors that interact in various ways with structural and electrical remodeling, more or less markedly favoring the onset of arrhythmia, as hypothesized by Fatkin et al. [39].

As for the rationale for the relationship between gender and different propensity to AF in subjects who practice intense endurance, the available studies highlight a difference in atrial remodeling, both chronic and acute, in the two sexes, which could perhaps provide a key to understanding. In male athletes, chronic left ventricular hypertrophy and diastolic dysfunction prevail; in female athletes, there is a different modality of acute remodeling, which reversibly impacts the right sections, while chronic left ventricular hypertrophy and diastolic dysfunction are less evident [40, 41].

Therapeutic management and eligibility for competitive sports

When faced with an athlete with AF, the first task of the doctor is to properly frame the subject from a cardiological point of view, excluding structural heart disease, channelopathies, thyroid disorders, alcohol abuse and use of performance-enhancing drugs. In the absence of dedicated studies, the therapeutic choices should refer to the current guidelines: those just published by the European Society of Cardiology [42] introduce the CARE paradigm as the basis of the approach to AF [C: comorbidity and risk factor treatment; A: avoid stroke; R: reduce symptoms (rate and rhythm control); E: evaluation and dynamic reassessment], which can also be a valuable guide here. However, there are some important peculiarities to take into consideration with patients who are athletes:

1) The treatment, whatever it is, must not significantly impact their physical performance.
2) The treatment must be well tolerated.
3) The treatment must be shared and accepted by the athlete.
4) Any drugs used must not be included in the list of "prohibited" substances (such as beta blockers in some sports).
5) If anticoagulant treatment is considered, the traumatic risk that the sport may entail must be considered.

But above all, a fifth letter must be added to the four letters of the CARE paradigm: the "D" of "detraining". Experimental and human studies demonstrate that detraining alone can significantly reduce the incidence of AF in athletes who are affected by it [33, 43, 44]. A common clinical recommendation is to have the athlete with symptomatic AF reduce the duration and intensity of exercise for up to 3 months to assess the relationship of exercise to AF [33], but this approach is based solely on anecdotal evidence and expert opinions [33], and the first randomized controlled trial on this specific topic is still ongoing [45]. However, many athletes refuse detraining: a completely understandable attitude, which must be respected. In such cases, or when detraining does not produce significant results, the "R" of the CARE paradigm must be brought into play, with the patient being managed by controlling rate or rhythm. In the past, the choice between the two options represented a major dilemma for the cardiologist: today, the two strategies are seen as complementary and adopting one or the other is a decision that must always be re-evaluated in follow-up, as the recently published guidelines themselves indicate [42]. Of course, in athletes, rate control (leaving the arrhythmia to itself and simply controlling the ventricular rate) is an option that does not give satisfactory results [33, 41]: the drugs used for this purpose (beta blockers and non-dihydropyridine Ca-antagonists) are often not well tolerated, are sometimes prohibited (beta blockers), or are not very effective, while it is known that digitalis does not safely/effectively control the ventricular rate of AF in athletes [33, 41]. Rhythm control (trying to restore and maintain sinus rhythm) is not much better: class IC drugs are effective, but if the arrhythmia arises during a race or training, flecainide or propafenone can cause the AF to transform into atrial flutter, which can be conducted to the ventricles at a very rapid rate, even with 1:1 conduction, with emergency or catastrophic consequences [33, 41, 46]. In case of drug failure or as a first-line option in selected cases, there is catheter ablation, the results of which seem to be superimposable on those for the general population [46, 47]. With regard to antithrombotic prophylaxis, it seems obvious that, in the athlete, the risk assessed with CHA2DS2-VA is often zero, but the problem should not be underestimated, given that some evidence indicates a non-negligible incidence of stroke in senior athletes affected by AF [48, 49]. In the event that anticoagulant therapy is started, the attitude so far has been to prohibit contact sports or in any case sports with a traumatic risk [26, 33]. The particular pharmacodynamics and pharmacokinetics of direct-acting oral anticoagulants (DOAC) could perhaps introduce some innovations in this field: theoretically, the athlete could be allowed, by calibrating the time of administration of the DOAC [50, 51], to perform competitions/training limited to the period in which the effect of the drug is minimal or null.

The last but not least important question is precisely that of the eligibility for competitive sports of athletes with AF. A recent document submitted to the literature by an Italian expert committee states that in the absence of Wolff-Parkinson-White (WPW), eligibility should be granted under the following conditions [52]:

- Absence of heart disease incompatible with competitive sports and absence of major symptoms.
- Any identifiable triggering cause has been identified and removed.
- There is no cause-effect relationship between sports activities and arrhythmia.
- The AF episode is infrequent, is not high frequency and is of limited duration.

If AF is permanent, eligibility could be granted for low or moderate cardiovascular commitment sports activities, provided there is no heart disease or significant symptoms, and the heart rate during exercise test and electrocardiogram (ECG) monitoring does not exceed the maximum heart rate (HR) for the athlete's age [52]. Italian guidelines tend to be more specific and restrictive than European ones: the latter indicate good practice for subjects engaging in physical activity at various levels [53]. As has already been observed, athletes on anticoagulant therapy have so far been excluded from participating in sports activities with a high risk of trauma [52, 53].

Conclusions

In conclusion, intense endurance sports increase the risk of AF in males, especially in middle-aged ones. In the management of arrhythmia, the "D" of detraining is added to the CARE paradigm. The topic deserves further study to better define mechanisms, subjects at risk and management. Finally, we believe that generating doubts about the beneficial effect of physical activity can be somewhat dangerous and is not scientifically justified.

Abbreviation

AF: atrial fibrillation

DOAC: direct-acting oral anticoagulants

Declarations

Acknowledgments

We are grateful to Dr. Claire Archibald for her support and guidance.

Author contributions

MC: Conceptualization, Writing—original draft. MVE: Writing—review & editing. MP: Conceptualization, Writing—review & editing. LS: Writing—review & editing. LC: Writing—original draft. FL: Data curation, Writing—review & editing. VC: Writing—review & editing. MM: Writing—review & editing. All authors read and approved the submitted version.

Conflicts of interest

The authors declare that there are no conflicts of interest.

Ethical approval

Not applicable.

Consent to participate

Not applicable.

Consent to publication

Not applicable.

Availability of data and materials

Not applicable.

Funding

Not applicable.

Publisher's note

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