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Atrial fibrillation in endurance athletes. Can you have too much of a good thing?

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Take Home Messages

- Endurance athletes appear to be at increased risk of atrial fibrillation (AF).
- Morphological cardiovascular adaptions to exercise in the left atrium may be critical to the development of AF in this group.
- Whilst advocated by international guidelines, there is little data to guide individualised treatment strategies in this group.

Introduction.

Atrial fibrillation (AF) is the most common cardiac arrhythmia with a prevalence of 2.5% in the United Kingdom [1]. Physical activity markedly reduces the risk of AF [2]. However, these benefits plateau with increasing exercise dose and contemporary data suggests a paradoxical rise in AF risk in athletes who participate in sport performed over long durations of time (termed endurance exercise) [3]. In a study of >50,000 cross-country skiers, the risk of AF was associated a greater number of race participations (HR 1.29, 95%CI 1.04-1.61)[4]. These findings have been replicated in other studies and a recent meta-analysis showed athletes to be at a higher risk of AF compared to non-athletes (OR 2.46, 95%CI 1.73-3.51) [3].

Here, we will explore the physiological pathways that could explain the excess risk of AF observed in athletes and the unique challenges faced by this population.

The Athletes Heart: a substrate for AF.

Morphological adaptations to endurance exercise have been extensively reported as part of the athlete's heart and are considered physiological and reversible. However, these adaptations may increase the risk of AF. Here, we describe these as part of Coumel's Triangle, a conceptual framework that describes three fundamental processes for arrhythmogenesis: a pro-arrhythmogenic substrate, modulator and trigger [5].

Endurance exercise simulates a transient pressure- and volume-overload-like state which evokes a proportionate dilatation of all four cardiac chambers. These changes are typically observed after many months to years of endurance training[6]. Atrial adaptations are integral to the pathophysiological process of AF and they may be more susceptible to the acute and chronic insults of exercise when compared to the ventricles. Atrial walls are thinner and elliptical in shape [7]. Therefore, the thin flat walls are less able to modulate shear wall stress [7]. As proposed by the 'dam wall theory,' time in diastole is relatively shortened during exercise [8]. Consequently, the mitral valve remains closed for longer periods which increases back pressure into the left atrium (LA) [8]. These processes cause atrial stretch. The greater susceptibility of the atria to stretch evokes a stronger adaptive signal and may explain the excess in atrial arrhythmias and why ventricular arrhythmias are rarely observed in otherwise healthy athletes.

Over time, atrial stretch evokes morphological adaptations. A meta-analysis found LA diameter to be 13% (4.1 ml/m^2) greater in athletes when compared with sedentary controls [9]. In the general population, every 5mm increase in LA size is associated with a 39% increased risk of developing AF of any type [10, 11]. Surprisingly, the atria of athletes may be more susceptible to scar. In 20 otherwise healthy athletes undergoing

cardiac magnetic resonance imaging, the investigators reported a significantly higher proportion of LA fibrosis in highly trained athletes [12]. Myocardial fibrosis is thought to be pro-arrhythmogenic by disrupting muscle bundle conduction causing adjacent areas of myocardium to be out-of-phase [13].

Bradycardia is seen as a marker of cardiorespiratory fitness and reflects a shift in autonomic balance towards parasympathetic tone. This may be a critical step in the pathogenesis of exercise-induced AF. Animal studies have shown inhibition of parasympathetic tone to suppress AF inducibility [14]. Increasing parasympathetic tone shortens the atrial refractory period and allows re-entry of electrical conduction into maladaptive conduction pathways [7]. Atrial ectopy is a trigger for AF in the general population and small studies have shown that rates of atrial ectopy appeared to be associated with lifetime training volume [15].

What is unique about this population?

Stroke is a major cause of morbidity in AF, but athletes are typically thought to be at low risk of stroke due to the favourable effects of exercise on risk factors used to calculate stroke risk. However, in an international survey, athletes with AF had an increased risk of stroke, even in those who had a CHA_2-DS_2-VASc of 0/1 (OR 4.20, 95%CI 1.83-9.66) [16]. As risk scores do not capture pro-stroke phenotypes such as atrial dilatation, it may be that the risks are underestimated [17].

Athletes with AF may have distinct treatment goals. The prescription of anticoagulation in endurance athletes comes with challenging shared-care decisions over bleeding risk, where the risk of impact related bleeding may be significant [18]. Pharmacological rate and rhythm control strategies are pillars in the management of AF but may be less favourable choices for athletes due to potential performance limitations, with many continuing with competition into older age [18]. Radiofrequency catheter ablation may

be effective in athletes, but its role in those with irreversible structural adaptions is less clear [19]. In some, exercise limitation may be advised but this should be weighed against the mental benefits of exercise.

Conclusions.

Atrial fibrillation is now a well-recognised consequence of long-term endurance exercise. Future research should look to assess individualised treatment strategies in this population.

Disclosures

I have no conflict of interests to declare.

Figure(s)

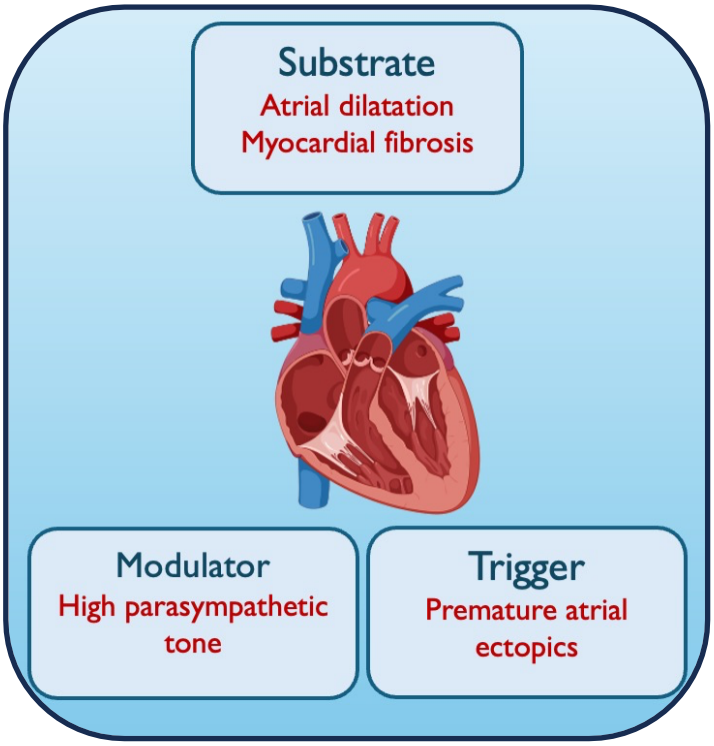

Figure 1. Cardiovascular adaptions that occur in response to long-term endurance training which may promote atrial fibrillation. Categorised by Coumel's Triangle of Arrhythmogenesis.

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