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Protecting against sedentary lifestyle, left atrial enlargement and atrial fibrillation

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Physical inactivity in contemporary obesogenic environments induces cardiac maladaptations, which causes a higher risk of atrial fibrillation (AF).[1] Light-to-moderate physical activity (PA), however, is associated with a lower incidence of AF.[1–3] Conversely, the long-term practice of strenuous endurance exercise increases the risk of AF in younger, middle-aged and older populations, particularly in highly competitive athletes,[4] depicting a U-shaped curve (see figure 1).[1 5] The precise causative mechanism/s of exercise-induced AF is still unknown.[6] This type of exercising produces structural and functional cardiac adaptations, higher levels of oxidative stress, inflammation and adrenergic activation, ultimately predisposing to atrial fibrosis and AF.[1]

Left atrium (LA) dilation or LA enlargement (LAE) is thought to be a potential causative mechanism of exercise-related AF.[7 8] In addition, there are likely differences in the aetiology and clinical presentation of AF between endurance athletes and the general (non-athletic) population, that is, 'classical AF',[9 10] so much so that we described that 'exercise-induced' AF, which we coined as 'Paroxysmal Atrial Fibrillation in Young and Middle-Aged Athletes' syndrome, usually shows a number of features that are common to most affected athletes.[9]

Some studies have previously demonstrated more pronounced LAE in the most trained athletes, especially in long-term strenuous endurance exercisers.[11 12] We evaluated LA volumes with late gadolinium enhancement MRI (LGE-MRI) in former elite endurance athletes (n=10) and sedentary control subjects (n=5).[11] The values of LA volumes corrected for body surface area were significantly higher in athletes compared with control subjects (58±14 mL/m^2 vs 39±14 mL/m^2, respectively; p=0.026). Recently, Trivedi *et al* also reported altered LA function between athletes with and without AF, as well as the difference between groups in LA/left ventricle (LV) ratio.[13] Importantly, LAE has been independently associated with an increased risk of all-cause mortality in patients with normal LV filling pressure and preserved LV ejection fraction.[14 15]

Heitmann and colleagues,[16] from the University of Norway, as part of the Tromsø Study 1994–2016, reported that moderately active individuals had 32% lower AF risk than inactive, while those with LAE had 38% higher AF risk compared with participants with normal LA size (figure 1). However, the increased AF risk with LAE was attenuated by PA; compared with inactive participants with LAE, the AF risk was 45% lower among active with LAE (see figure 1). AF risk in active participants with LAE did not differ from active with normal LA size. Noticeably, the authors also observed a 79% higher risk of AF in males than in females, as well as those participants ≥65 years had a 2.6-fold higher risk of AF compared with participants<65 years.

There is growing evidence that long-term strenuous aerobic (or 'endurance') exercise (eg, marathon/ultra-marathon running) is associated with higher risk of AF (particularly lone AF) compared with age-matched inactive referents[17–21] and such increased risk also affects young people.[17 22] The association might be particularly strong in elite athletes, although there is still some controversy.[12 23] However, the risk of exercise-induced AF might decrease as the population ages[17 22] and is overall offset by known beneficial effects of vigorous exercise, that is, attenuation of cardiovascular (CV) disease (CVD) risk factors[17] and improved CV health.[24] Potential causative factors for de novo development of AF in previously healthy long-term exercisers include LAE/fibrosis, increased parasympathetic tone and inflammation.[25–28] Yet more mechanistic research is needed to unveil the actual cause–effect relationships. In contrast, light-moderate PA/exercise (eg,

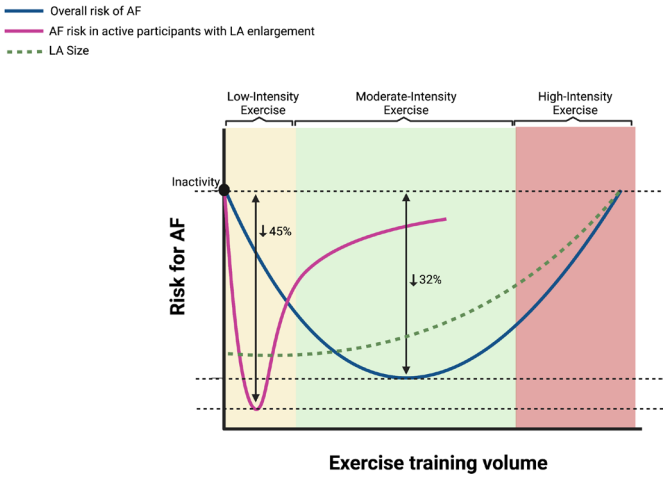

Figure 1 Legend dose–response association between physical activity volume, AF risk, and left atrium enlargement based on Heitmann *et al* results. AF, atrial fibrillation; LA, left atrium.

walking/brisk walking) would have an opposite, beneficial effect on AF risk, particularly in old adults.[22 29 30]

Overall, in agreement with the results reported by Heitmann and colleagues, LAE is one important factor associated with the onset of AF. The study conducted by Heitmann and coworkers increases the evidence on how physical activity or exercise intensity can be implicated in LAE, and hence, in preventing or increasing the risk of suffering AF. Until now, it has not been demonstrated that there is a genetic association of exercise with AF but only that it is related to CV adaptations and/or maladaptations in response to long-term and high-intensity training and/or competition. It seems reasonable to monitor cardiac remodelling and be aware of AF symptoms in these individuals. Finally, there is little evidence to date on the adverse CVD complications, including stroke, of AF in highly trained athletes, with or without LAE, that are well-known complications in many typical patients with AF.

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REFERENCES

1 O'Keefe EL, Sturgess JE, O'Keefe JH, *et al*. Prevention and treatment of atrial fibrillation via risk factor modification. *Am J Cardiol* 2021;160:46–52.
2 Santos-Lozano A, Sanchis-Gomar F, Barrero-Santalla S, *et al*. Exercise as an adjuvant therapy against chronic atrial fibrillation. *Int J Cardiol* 2016;207:180–4.
3 Mehta NK, Strickling J, Mark E, *et al*. Beyond cardioversion, ablation and pharmacotherapies: risk factors, lifestyle change and behavioral counseling strategies in the prevention and treatment of atrial fibrillation. *Prog Cardiovasc Dis* 2021;66:2–9.
4 Iskandar A, Mujtaba MT, Thompson PD. Left atrium size in elite athletes. *JACC Cardiovasc Imaging* 2015;8:753–62.
5 Schnohr P, O'Keefe JH, Lavie CJ, *et al*. U-shaped association between duration of sports activities and mortality: Copenhagen City heart study. *Mayo Clin Proc* 2021;96:3012–20.
6 Sanchis-Gomar F, Lucia A. Pathophysiology of atrial fibrillation in endurance athletes: an overview of recent findings. *CMAJ* 2016;188:E433–5.
7 Sanchis-Gomar F, Lucia A, Levine BD. Editorial commentary: Relationship between strenuous exercise and cardiac "morbimortality": Benefits outweigh the potential risks. *Trends Cardiovasc Med* 2016;26:241–4.
8 Sanchis-Gomar F, Pérez LM, Joyner MJ, *et al*. Endurance exercise and the heart: friend or foe? *Sports Med* 2016;46:459–66.
9 Sanchis-Gomar F, Perez-Quilis C, Lippi G, *et al*. Atrial fibrillation in highly trained endurance athletes - Description of a syndrome. *Int J Cardiol* 2017;226:11–20.
10 Sanchis-Gomar F, Guía-Galipienso Fdela, Lavie CJ. Atrial fibrillation in athletes and non-athletes: evidence of different causative mechanisms. *Eur Heart J Cardiovasc Imaging* 2021;22:723.
11 Sanchis-Gomar F, Garatachea N, Catalán P, *et al*. LA size in former elite athletes. *JACC Cardiovasc Imaging* 2016;9:630–2.
12 Pelliccia A, Maron BJ, Di Paolo FM, *et al*. Prevalence and clinical significance of left atrial remodeling in competitive athletes. *J Am Coll Cardiol* 2005;46:690–6.
13 Trivedi SJ, Claessen G, Stefani L, *et al*. Differing mechanisms of atrial fibrillation in athletes and non-athletes: alterations in atrial structure and function. *Eur Heart J Cardiovasc Imaging* 2020;21:1374–83.
14 Patel DA, Lavie CJ, Gilliland YE, *et al*. Prediction of all-cause mortality by the left atrial volume index in patients with normal left ventricular filling pressure and preserved ejection fraction. *Mayo Clin Proc* 2015;90:1499–505.
15 Patel DA, Lavie CJ, Milani RV, *et al*. Association of left ventricular geometry with left atrial enlargement in patients with preserved ejection fraction. *Congest Heart Fail* 2012;18:4–8.
16 Heitmann KA, Løchen M-L, Stylidis M, *et al*. Associations between physical activity, left atrial size and incident atrial fibrillation: the Tromsø study 1994–2016. *Open Heart* 2022;9:e001823.
17 Aizer A, Gaziano JM, Cook NR, *et al*. Relation of vigorous exercise to risk of atrial fibrillation. *Am J Cardiol* 2009;103:1572–7.
18 Elosua R, Arquer A, Mont L, *et al*. Sport practice and the risk of lone atrial fibrillation: a case-control study. *Int J Cardiol* 2006;108:332–7.
19 Molina L, Mont L, Marrugat J, *et al*. Long-term endurance sport practice increases the incidence of lone atrial fibrillation in men: a follow-up study. *Europace* 2008;10:618–23.
20 Mont L, Sambola A, Brugada J, *et al*. Long-lasting sport practice and lone atrial fibrillation. *Eur Heart J* 2002;23:477–82.
21 Myrstad M, Nystad W, Graff-Iversen S, *et al*. Effect of years of endurance exercise on risk of atrial fibrillation and atrial flutter. *Am J Cardiol* 2014;114:1229–33.
22 Drca N, Wolk A, Jensen-Urstad M, *et al*. Atrial fibrillation is associated with different levels of physical activity levels at different ages in men. *Heart* 2014;100:1037–42.
23 Furlanello F, Bertoldi A, Dallago M, *et al*. Atrial fibrillation in elite athletes. *J Cardiovasc Electrophysiol* 1998;9:S63–8.
24 Sanchis-Gomar F, Lucia A, Levine BD. Relationship between strenuous exercise and cardiac "morbimortality": Benefits outweigh the potential risks. *Trends Cardiovasc Med* 2016;26:241–4.
25 Guasch E, Benito B, Qi X, *et al*. Atrial fibrillation promotion by endurance exercise: demonstration and mechanistic exploration in an animal model. *J Am Coll Cardiol* 2013;62:68–77.
26 Wilhelm M, Roten L, Tanner H, *et al*. Atrial remodeling, autonomic tone, and lifetime training hours in nonelite athletes. *Am J Cardiol* 2011;108:580–5.
27 Turagam MK, Velagapudi P, Kocheril AG. Atrial fibrillation in athletes. *Am J Cardiol* 2012;109:296–302.
28 Turagam MK, Velagapudi P, Alpert MA. Does exercise cause atrial fibrillation? *Int J Cardiol* 2015;181:245–6.
29 Mozaffarian D, Furberg CD, Psaty BM, *et al*. Physical activity and incidence of atrial fibrillation in older adults: the cardiovascular health study. *Circulation* 2008;118:800–7.
30 Levine BD. Can intensive exercise harm the heart? The benefits of competitive endurance training for cardiovascular structure and function. *Circulation* 2014;130:987–91.