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Differing mechanisms of atrial fibrillation in athletes and non-athletes: alterations in atrial structure and function

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Aims Atrial fibrillation (AF) is more common in athletes and may be associated with adverse left atrial (LA) remodelling. We compared LA structure and function in athletes and non-athletes with and without AF.

Methods and results Individuals (144) were recruited from four groups (each $n = 36$): (i) endurance athletes with paroxysmal AF, (ii) endurance athletes without AF, (iii) non-athletes with paroxysmal AF, and (iv) non-athletic healthy controls. Detailed echocardiograms were performed. Athletes had 35% larger LA volumes and 51% larger left ventricular (LV) volumes vs. non-athletes. Non-athletes with AF had increased LA size compared with controls. LA/LV volume ratios were similar in both athlete groups and non-athlete controls, but LA volumes were differentially increased in non-athletes with AF. Diastolic function was impaired in non-athletes with AF vs. non-athletes without, while athletes with and without AF had normal diastolic function. Compared with non-AF athletes, athletes with AF had increased LA minimum volumes (22.6 ± 5.6 vs. 19.2 ± 6.7 mL/m^2, $P = 0.033$), with reduced LA emptying fraction (0.49 ± 0.06 vs. 0.55 ± 0.12, $P = 0.02$), and LA expansion index (1.0 ± 0.3 vs. 1.2 ± 0.5, $P = 0.03$). LA reservoir and contractile strain were decreased in athletes and similar to non-athletes with AF.

Conclusion Functional associations differed between athletes and non-athletes with AF, suggesting different pathophysiological mechanisms. Diastolic dysfunction and reduced strain defined non-athletes with AF. Athletes had low atrial strain and those with AF had enlarged LA volumes and reduced atrial emptying, but preserved LV diastolic parameters. Thus, AF in athletes may be triggered by an atrial myopathy from exercise-induced haemodynamic stretch consequent to increased cardiac output.

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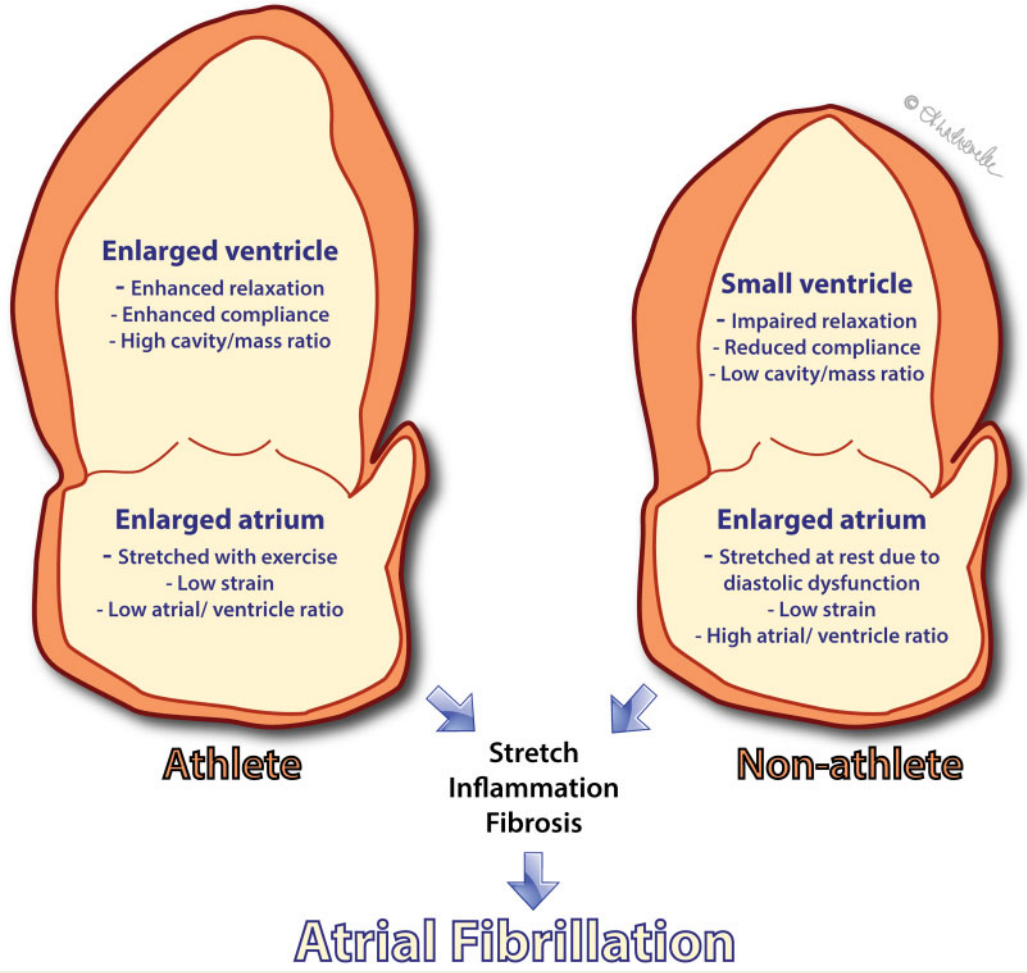
Graphical abstract



Keywords athletes • athlete's heart • atrial fibrillation • left atrium • strain • diastolic function

Introduction

There is evidence of an increased risk of cardiac arrhythmias, particularly atrial fibrillation (AF), in endurance athletes.[1,2] Data suggest a 'U-shaped' relationship between exercise dose and cardiac outcomes, where higher exercise doses confer overall health benefits, but are associated with a higher AF risk.[1,2]

However, the mechanisms responsible for 'exercise-induced' AF remain unclear. An established risk factor for AF is left atrial (LA) enlargement,[3] typically secondary to diastolic dysfunction and hypertension. In athletes, LA remodelling (enlargement) is a common physiologic adaptation to endurance training,[4] though affecting only a minority. The relationship between atrial size and AF risk has not been established in athletes. In this study, we investigated whether changes in atrial structure and function could differentiate between athletes with and without AF. In addition, comparisons were performed with non-athletic individuals with and without AF, to unravel the pathophysiological mechanisms underpinning the development of AF with exercise training. We hypothesized that, relative to non-athletes, there would be significant atrial remodelling in athletes, with greater differences evident in athletes with AF.

Methods

Study population

Thirty-six endurance athletes with paroxysmal AF were identified from the ProAFHeart trial, a prospective multicentre study initiated in 2017

(ACTRN12618000711213), comprising of former elite rowers and endurance athletes with AF, meeting the following criteria: (i) previous or current competition in endurance sport (distance running, road or mountain cycling, rowing or triathlon) at a national or international level, (ii) training for endurance competition for at least 5 years, and (iii) no other cardiovascular disease or symptoms. From a cohort of 249 elite endurance athletes, 47 athletes were identified with at least 1 documented AF episode requiring medical intervention; of these, 9 were excluded because of chronic AF, one for hypertrophic cardiomyopathy and another for moderate aortic stenosis. Each athlete with AF was age- and gender-matched (±5 years) to an athlete in the ProAFHeart trial without a history of AF (including an exercise test and 72-h Holter monitor), and any other cardiac abnormalities.

Non-athletic individuals were fit and active but did not participate in competition (i.e. only recreational sports) and were not engaged in any kind of routine training programme. Non-athletes with AF were recruited from cardiology outpatient clinics, and healthy controls were recruited from hospital volunteers. Written informed consent was obtained from all subjects and the study protocol was approved by the Western Sydney Local Health District Human Research Ethics Committee and the Baker Heart and Diabetes Institute Ethics Committee.

There were 144 individuals across 4 groups ($n = 36$ in each): (i) endurance athletes with paroxysmal AF, (ii) endurance athletes without AF, (iii) non-athletes with paroxysmal AF, and (iv) non-athletic healthy controls.

Standard echocardiographic assessment

A comprehensive transthoracic echocardiogram was performed using commercially available ultrasound machines (Vivid E95, GE Healthcare, Chicago, IL) by experienced medical professionals or cardiac sonographers, blinded to patient information. With patients in left-lateral decubitus position, parasternal, apical, and subcostal images were obtained. 2D, colour and Doppler images were obtained; 3–5 consecutive cardiac cycles were saved in cine format. Measurements were obtained according to the American Society of Echocardiography recommendations[5] with offline analysis performed using dedicated software (EchoPac 113, General Electric-Vingmed).

LA maximal volume (LA_{max}) was measured at end-systole[5] and LA minimum volume (LA_{min}) end-diastole by modified Simpson's biplane method of discs from the apical four- and two-chamber views. An average of three values was obtained and indexed to body surface area (BSA).

LA stroke volume was calculated as LA_{max} volume–LA_{min} volume. LA emptying fraction was calculated as $(LA_{max}–LA_{min}/LA_{max}) \times 100$. LA expansion index was calculated as $(LA_{max}–LA_{min}/LA_{min}) \times 100$. All atrial volumes were indexed to BSA.

LV mass, LV end-diastolic volume (EDV), LV end-systolic volume (ESV), and LV stroke volume were calculated and indexed to BSA as recommended.[5] Biplane LVEF was also calculated.

The ratio of LA_{max} volume to LVEDV (LA/LV ratio) was assessed to verify whether atrial remodelling was disproportionate to LV remodelling.

Pulsed-wave Doppler mitral inflow velocities were obtained to assess LV diastolic filling, as recommended,[6] including peak E-wave and A-wave, and the E/A ratio.

Pulsed tissue Doppler imaging was performed and septal and lateral annular peak systolic (s'), early diastolic (e'), and late diastolic (a') annular velocities were obtained.[6] The E/e' was calculated using an average of septal and lateral e'.[6]

Two-dimensional strain echocardiography

LV and LA strain measurements were obtained from 2D images (acquired at >60 fps) using software permitting semi-automated analysis (EchoPac version BT 13, General Electric, Horten, Norway).[7] In brief, LA endocardial borders were manually traced in four- and two-chamber views. After adjustment of the tracked area and reducing the region of interest for the thin walled LA, the software divided the atrium into six segments (*Figure 1*); the tracking quality for each segment was automatically scored, with the possibility for further manual correction. Peak atrial reservoir and contractile strain were obtained using R-R wave gating. Right atrial strain was measured in a similar manner, with strain measurements limited to six segments from the apical four-chamber view.

LV global longitudinal strain (GLS) was calculated as the average longitudinal strain from 18 segments obtained from apical four-, two-, and three-chamber views, with the software automatically subdividing each LV wall into three segments (basal, mid, and apical). Peak LV systolic strain was defined as the peak negative strain during systole.

LA stiffness estimation

LA stiffness was assessed as E/e'/LA reservoir strain as previously described.[8]

Statistical analysis

Analyses were performed using IBM SPSS version 25 (IBM, Inc., New York, NY). Continuous variables are summarized using mean ± SD, and categorical variables using frequencies and percentages. Plots of the mean and its associated 95% CI by AF status (present vs. absent) and athletic status (athlete vs. non-athlete) illustrate the distribution of parameters of interest. Two-tailed tests with a significance level of 5% were used. Repeated measures analysis of variance was used to test for an interaction between the effect of AF status (present vs. absent) and athletic status (athlete vs. non-athlete), accounting for matching. A statistically significant interaction signified that the difference in a parameter measurement in athletes due to AF status differed significantly from that seen in non-athletes. Independent-samples *t*-tests were used to test for differences by AF status within athletes and non-athletes, as well as between athletes and non-athletes.

Results

Baseline characteristics

Demographic characteristics of the study population are presented in Table 1.

Non-athletes

Non-athletes with AF had higher BMI than those without AF (mean difference 2.4 kg/m^2, 95% CI 0.4–4.4 kg/m^2, $P = 0.018$). Non-athletes with AF had higher heart rates compared with non-athletes without AF (mean difference 3.9 bpm, 95% CI 0.2–7.6 bpm, $P = 0.04$).

Athletes

BMI was comparable in athletes with and without AF. Heart rates in athletes were lower than in non-athletes, with no difference between those with and without AF. Amongst individuals with AF, the proportion of non-athletes on antiarrhythmic therapy was higher than in athletes. Only one athlete without AF was on a beta blocker for hypertension.

LA parameters

Table 2 summarizes the LA parameters and the effect of AF status within non-athletes and athletes. The difference in echocardiographic

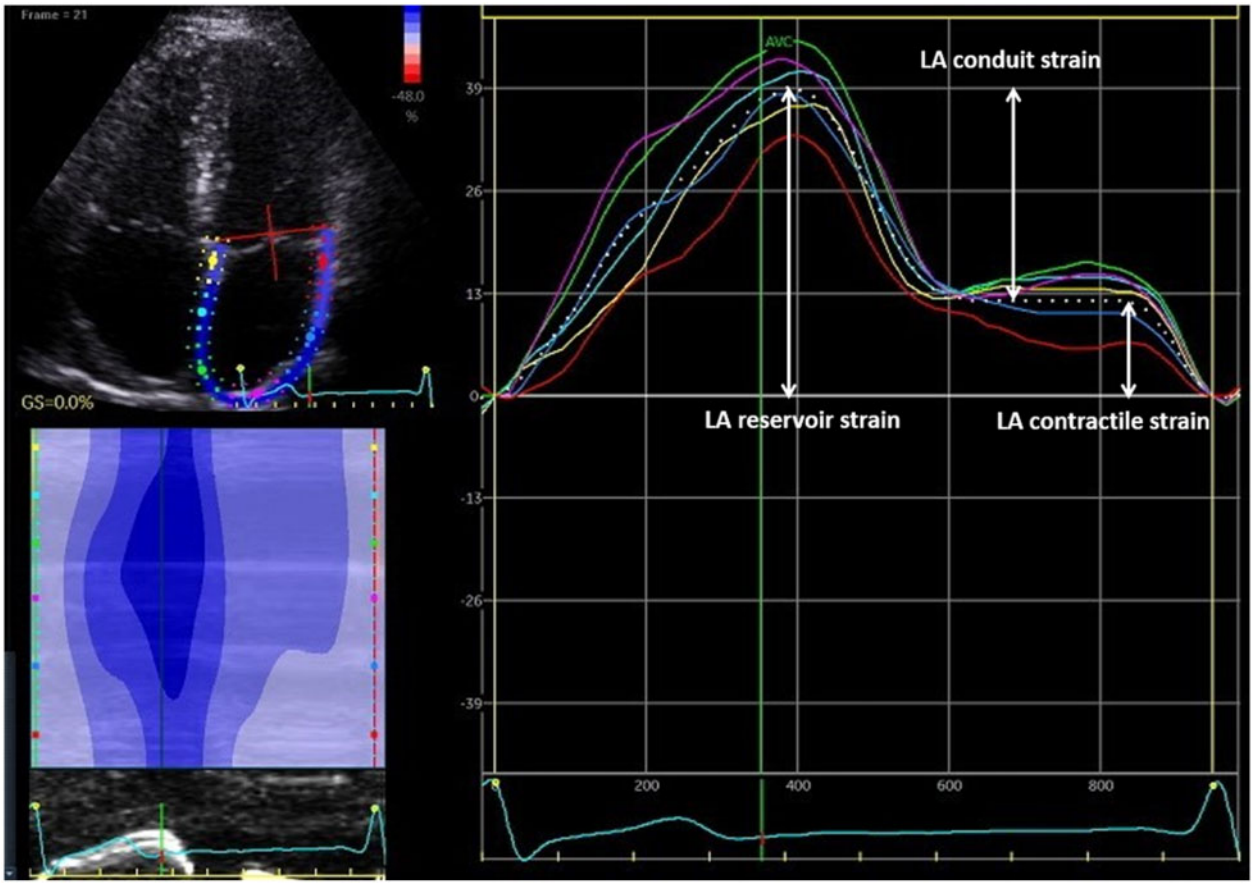


Figure 1 LA strain measurement (apical 4-chamber view) from six segments (different colours), with corresponding strain curves, and average strain curve (dotted line).

variables by AF status is presented separately for athletes and non-athletes and reports the *P* value for interaction.

Overall, in both athlete and non-athlete groups, those with AF had greater LA_{min} compared with those without AF (*Figure 2*). LA stiffness was reduced in athletes compared with non-athletes.

Non-athletes

Within the non-athletic cohort, LA stiffness was increased in those with AF (*Figure 3*).

Athletes

Athletes with AF had significantly lower LA emptying fraction and LA expansion index compared with athletes without AF (*Figure 2*). Athletes with and without AF had similar LA stiffness.

LA strain

All athletes, as well as non-athletes with AF had reduced LA reservoir (*Figure 4*) and contractile strain compared with non-athletes without AF. The ratio of LA contractile strain (measured in end-diastole) to LA_{min} was significantly lower in both athletes and non-athletes with AF compared with their matched non-AF controls (*Figure 4*).

LV parameters

Table 3 summarizes LV parameters and the effect of AF status within non-athletes and athletes. All athletes had larger LVEDV compared with non-athletes (*Figure 5*). In both non-athlete and athlete groups, those with AF had larger LVEDV with a trend towards significance. The LA/LV ratio, as a measure of disproportionate LA-to-LV remodelling, was increased in non-athletes with AF, but was similar between controls and both athlete groups (*Figure 5*). Non-athletes with AF had greater LV mass, and diastolic parameters including E/e′ (*Figure 5*) were significantly impaired, compared with non-athletes without AF. In contrast, both athlete groups had normal diastolic function.

As detailed in *Table 3*, there was no difference in any of the measured LV parameters between athletes with and without AF. Overall, LV GLS was lower in athletes compared with non-athletes ($P < 0.001$).

Discussion

This cross-sectional cohort study, with an age- and gender-matched comparator population for endurance athletes, highlights potential differences in LA structural and functional characteristics between athletes and non-athletes, thereby suggesting different causative mechanisms for the development of AF. The

Table 1 Baseline characteristics of the study population

Variables	Non-athletes		Athletes	
	No AF	AF	No AF	AF
Age (years)	55.3 ± 13.5	54.5 ± 13.9	56.4 ± 12.7	56.5 ± 13.2
Males, *n* (%)	34 (94%)	34 (94%)	34 (94%)	34 (94%)
Height (m)	1.74 ± 0.09	1.75 ± 0.06	1.84 ± 0.08	1.82 ± 0.09
Weight (kg)	80 ± 17	87 ± 12	85 ± 12	82 ± 13
Body mass index (kg/m^2)	26.0 ± 4.5	28.4 ± 3.7	25.0 ± 2.7	24.7 ± 2.1
Body surface area (m^2)	1.94 ± 0.2	2.02 ± 0.15	2.07 ± 0.18	2.04 ± 0.20
Heart rate (beats/min)	62 ± 10	68 ± 9	53 ± 8	51 ± 9
Hypertension, *n* (%)	0.0	10 (28)	3 (8)	5 (14)
SBP (mmHg)	118 ± 11	147 ± 10	130 ± 11	137 ± 23
DBP (mmHg)	76 ± 8	88 ± 9	73 ± 9	73 ± 11
Smoking, *n* (%)	0.0	0.0	0.0	0.0
Diabetes mellitus, *n* (%)	0.0	0.0	0.0	1 (3)
Antiarrhythmic medications				
Beta-blocker, *n* (%)	0.0	8 (22)	1 (3)	4 (11)
Calcium-channel blocker, *n* (%)	0.0	4 (11)	0.0	1 (3)
Cardiac glycoside, *n* (%)	0.0	3 (8)	0.0	0.0
Sotalol, *n* (%)	0.0	14 (39)	0.0	2 (6)
Flecainide, *n* (%)	0.0	3 (8)	0.0	3 (8)
Amiodarone, *n* (%)	0.0	7 (19)	0.0	1 (3)

Values are expressed as mean ± SD or *n* (%).
DBP, diastolic blood pressure; SBP, systolic blood pressure.

Table 2 Matched analysis of the effect of AF status within non-athletes and athletes for LA echocardiographic parameters

Variables	Non-athletes		*P* value for non-athletes	Athletes		*P* value for athletes	*P* value athletes vs. non-athletes	*P* value for interaction between athletic status and AF status
	No AF	AF		No AF	AF			
Minimum LA volume indexed (mL/m^2)	13.0 ± 4.3	19.9 ± 8.2	<0.001	19.2 ± 6.7	22.6 ± 5.6	0.022	<0.001	0.10
Maximum LA volume indexed (mL/m^2)	27.3 ± 7.9	37.8 ± 11.2	<0.001	43.2 ± 11.7	44.5 ± 10.7	0.61	<0.001	0.011
LA stroke volume indexed	14.3 ± 5.8	18.0 ± 7.4	0.022	23.9 ± 10.0	21.9 ± 6.6	0.30	<0.001	0.050
LA emptying fraction indexed	0.51 ± 0.19	0.48 ± 0.15	0.53	0.55 ± 0.12	0.49 ± 0.06	0.011	0.30	0.52
LA expansion index indexed	1.2 ± 0.5	1.1 ± 0.6	0.48	1.2 ± 0.5	1.0 ± 0.3	0.009	0.71	0.39
LA stiffness	0.24 ± 0.09	0.37 ± 0.20	0.001	0.22 ± 0.09	0.22 ± 0.07	0.93	<0.001	0.001
LA reservoir strain (%)	33.2 ± 7.1	27.9 ± 8.4	0.005	28.2 ± 3.7	27.2 ± 4.5	0.32	0.008	0.032
LA conduit strain (%)	16.6 ± 6.3	14.9 ± 5.5	0.22	14.4 ± 4.0	14.2 ± 4.5	0.83	0.09	0.26
LA contractile strain (%)	16.6 ± 3.1	13.0 ± 5.1	0.001	13.8 ± 3.6	13.0 ± 3.1	0.32	0.032	0.026
LA contractile strain/LA minimum volume indexed ratio	1.4 ± 0.5	0.9 ± 0.6	<0.001	0.8 ± 0.4	0.6 ± 0.2	0.021	<0.001	0.006

Values are expressed as mean ± SD, and mean difference and its 95% CI.

key findings of our study are that athletes, including those with AF, have normal diastolic parameters, with atrial enlargement proportional to LV remodelling. However, athletes with AF have altered atrial function relative to athletes without AF. In contrast, AF in non-athletes is associated with diastolic dysfunction and disproportional atrial enlargement.

LA size and function

As a consequence of repeated, acute haemodynamic alterations with increased cardiac output, endurance training results in chronic adaptive changes in cardiac structure and function.[4] All four cardiac chambers increase in volume and mass, the extent of which is proportional

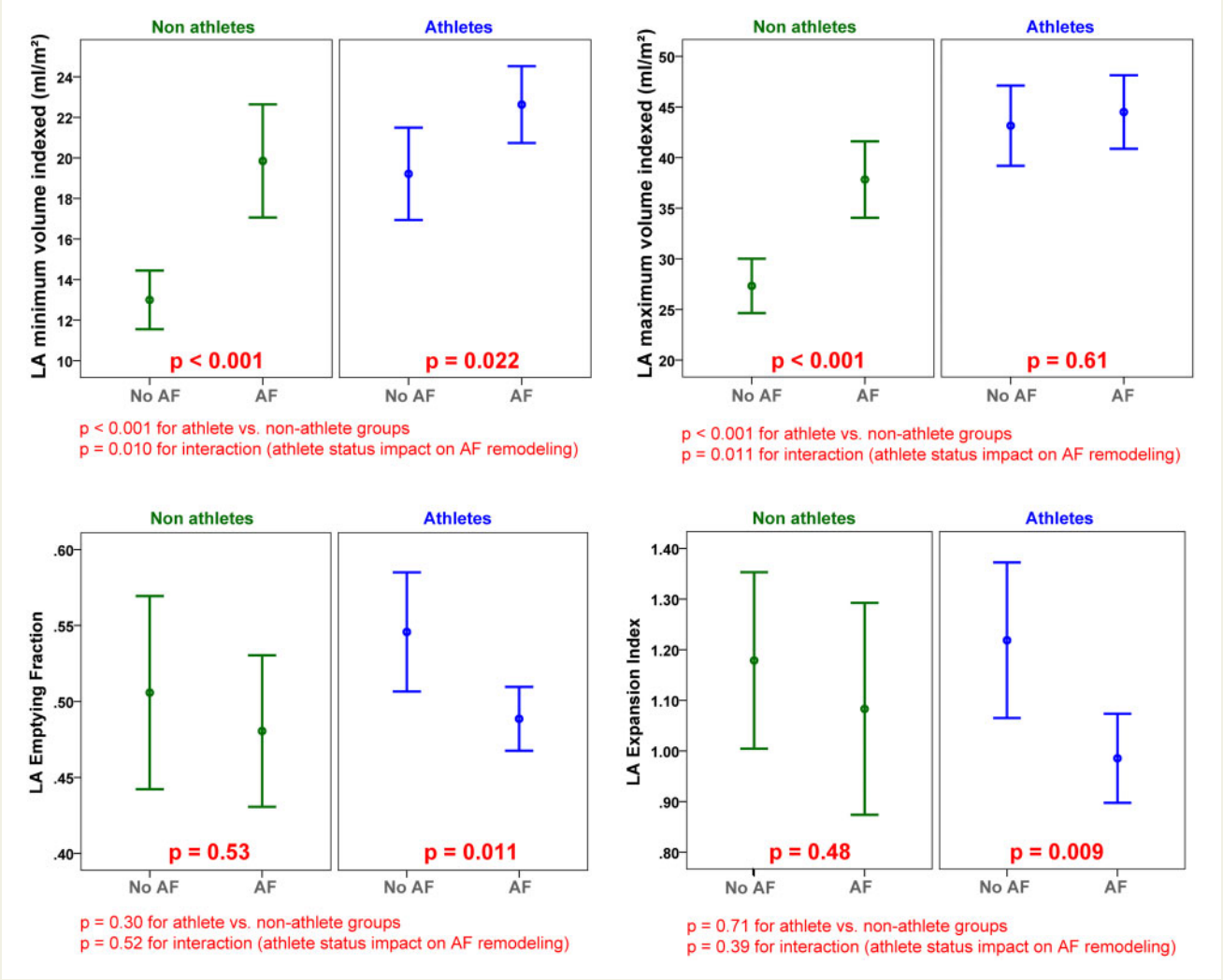


Figure 2 Mean and 95% CI by AF status (present vs. absent) and athletic status (athlete vs. non-athlete) for indexed LA minimum and maximum volume, LA emptying fraction, and LA expansion index.

to the level of fitness.[9] There is recent interest in the LA and the right ventricle (RV) due to their thin-walled structure and disproportionate increase in wall stress during exercise.[10,11] Perhaps as a consequence, these chambers appear to be a common origin of arrhythmias in athletes.[12,13] LA enlargement is an established risk factor for the development of AF in non-athletes,[14] but at the same time LA enlargement is common even in healthy athletes. Although atrial enlargement is associated with AF in the general population, its contribution to AF development in athletes has yet to be determined. Consistent with expectations, we observed increased LA volumes (both LA_{min} and LA_{max}) in athletes as compared with healthy non-athletes. The LA/LV ratio provides a particularly useful index because it expresses LA remodelling relative to LV remodelling. Consistent with descriptions of athletic cardiac remodelling,[4] we observed symmetrical enlargement of all four cardiac chambers. As a result, even though atrial volumes in athletes were greater than healthy non-athletes, the LA/LV ratio was similar. However, athletes with AF had a similar LA/LV ratio to athletes without AF, whereas non-athletes with AF had disproportionately enlarged atria (greater LA/LV ratio), probably due to raised LV pressures. We speculate that the lack of an increase in the LA/LV ratio in athletes with AF may be due to the lack of LV diastolic impairment. Moreover, it is likely that the effects of exercise-induced LA remodelling are relatively profound and 'dilute' any additional remodelling due to AF.

It was notable that LA_{min} was greater in endurance athletes with AF than in athletes with no AF. LA_{min}, measured in end-diastole, occurs when the LA is directly exposed to the LV end-diastolic pressure and is a more sensitive surrogate for LA dysfunction than LA_{max}.[15] In non-athletic populations LA_{min} has been demonstrated to be a sensitive marker of LV diastolic dysfunction that is closely related to the E/e′ ratio and a stronger prognostic indicator of adverse outcomes than LA_{max}.[16] However, this does not readily explain the predisposition to AF in endurance athletes, given that LV diastolic function parameters were similar in athletes with and without AF as

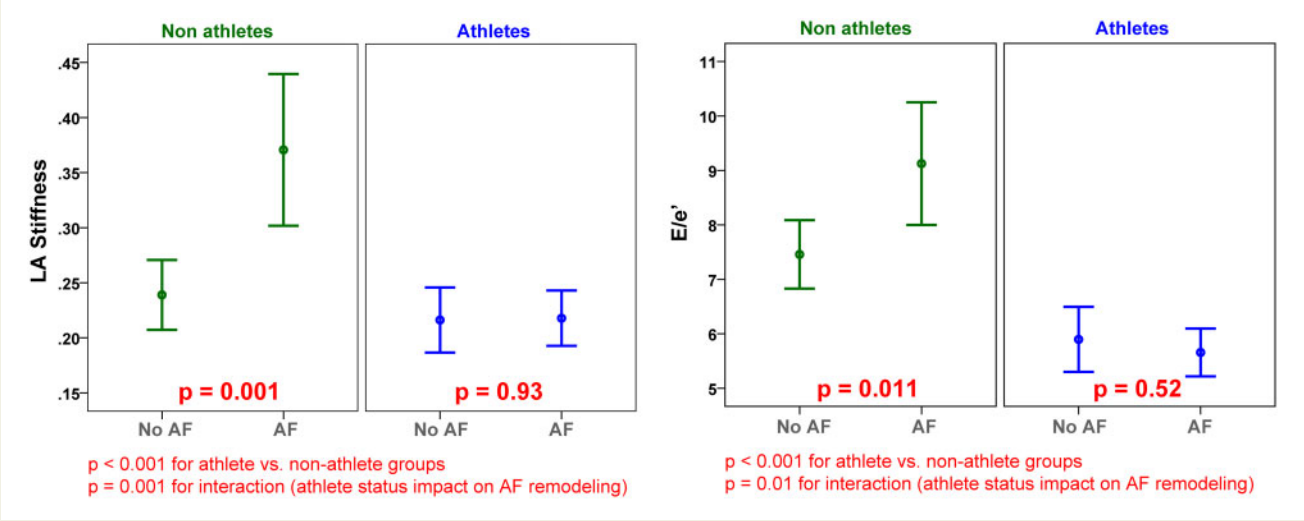


Figure 3 Mean and 95% CI by AF status (present vs. absent) and athletic status (athlete vs. non-athlete) for LA stiffness and E/e′.

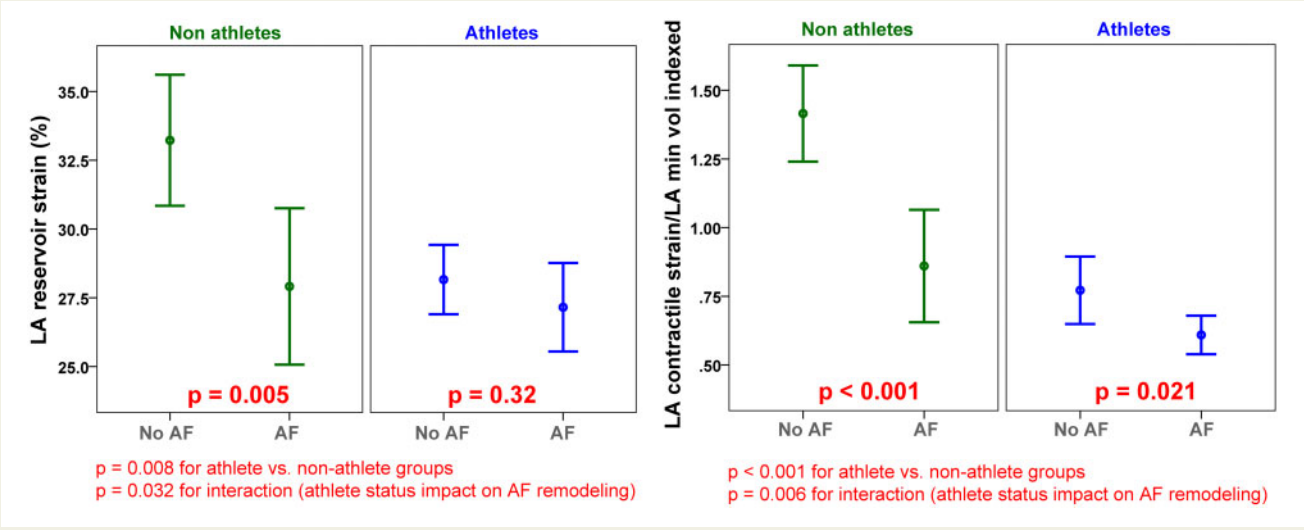


Figure 4 Mean and 95% CI by AF status (present vs. absent) and athletic status (athlete vs. non-athlete) for LA reservoir strain and ratio of LA contractile strain/LA minimum volume indexed.

well as healthy controls. Hence, there is likely an associated atrial myopathy in some endurance athletes.

Moreover, the development of AF may be multifactorial, including the development of atrial fibrosis, autonomic imbalance, and increased ectopic trigger activity. Thus, while an increase in LA_{max} in athletes represents the adaptive change to the increase in stroke volume from exercise, the increase in LA_{min} may be a marker of reduced atrial contractility and reflect an 'atrial myopathy'. This would also align with previous observations of prolonged p-wave duration on the ECG of athletes with AF that has been postulated to be an electrical marker of atrial dysfunction.[17]

Relative atrial remodelling

The key novel finding in this study is the between group difference in LA/LV ratio. This simple ratio expresses differential LA remodelling. If atrial size is considered in isolation, one might expect an even greater incidence of AF in endurance athlete populations; however, larger atrial volumes were observed in athletes without AF than in non-athletes with AF. The corollary is not directly addressed in this study, but it stands to reason that an increased LA/LV ratio may also be associated with AF in subjects with small stiff ventricles, whereby even mild increase in atrial volume would represent significant enlargement.

LA stiffness is a relatively novel echocardiographic parameter. Filling pressure estimates (E/e′) and myocardial deformation (LA reservoir strain) are combined and have been validated against invasively measured LA stiffness.[8,18] LA stiffness predicts maintenance of sinus rhythm after cardioversion for AF,[19] and predicts AF recurrences after pulmonary vein isolation (PVI).[8] In the context of physiological remodelling, the assessment of LA stiffness in athletes has

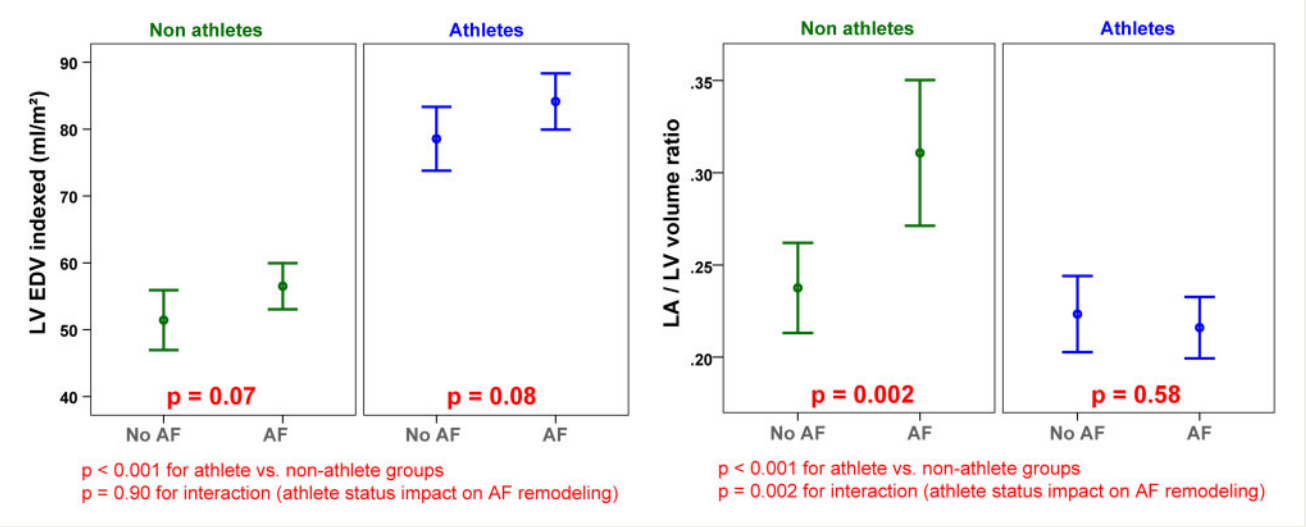


Figure 5 Mean and 95% CI by AF status (present vs. absent) and athletic status (athlete vs. non-athlete) for LV EDV indexed, and ratio of indexed LA/LV volume.

Table 3 Matched analysis of the effect of AF status within non-athletes and athletes for LV echocardiographic parameters

Variable	Non-athletes		P value for non-athletes	Athletes		P value for athletes	P value athletes vs. non-athletes	P value for interaction between athletic status and AF status
	No AF	AF		No AF	AF			
LVESV indexed (mL/m^2)	27.0 ± 33.0	25.0 ± 8.0	0.68	34.0 ± 7.0	35.0 ± 6.0	0.50	0.002	0.16
LVEDV indexed (mL/m^2)	51.4 ± 13.2	56.5 ± 10.2	0.07	78.6 ± 13.9	84.2 ± 12.1	0.08	<0.001	0.90
LV ejection fraction (%)	58.0 ± 8.0	56.0 ± 10.0	0.41	56.0 ± 4.0	58.0 ± 4.0	0.15	0.80	0.10
LV mass indexed (g/m^2)	78.0 ± 15.0	94.0 ± 27.0	0.003	90.0 ± 19.0	94.0 ± 14.0	0.29	0.11	0.09
Indexed LA/LV ratio	0.24 ± 0.07	0.31 ± 0.12	0.002	0.22 ± 0.06	0.22 ± 0.05	0.58	<0.001	0.002
Peak E (cms^{-1})	66.0 ± 10.0	71.0 ± 19.0	0.13	55.0 ± 12.0	57.0 ± 13.0	0.50	<0.001	0.47
Peak A (cms^{-1})	61.0 ± 15.0	54.0 ± 18.0	0.09	47.0 ± 11.0	48.0 ± 13.0	0.77	<0.001	0.08
E/A	1.2 ± 0.4	1.5 ± 0.7	0.013	1.3 ± 0.5	1.3 ± 0.5	0.90	0.64	0.023
Average e′ vel (cms^{-1})	9.0 ± 2.0	8.0 ± 2.0	0.09	10.0 ± 3.0	10.0 ± 2.0	0.49	0.003	0.025
E/e′	7.5 ± 1.9	9.1 ± 3.3	0.011	5.9 ± 1.8	5.7 ± 1.3	0.52	<0.001	0.010
LV GLS (%)	21.7 ± 2.9	21.0 ± 3.1	0.34	18.9 ± 2.1	19.2 ± 1.7	0.58	<0.001	0.20

Values are expressed as mean ± SD, and mean difference and its 95% CI.

clarified exercise-induced atrial adaptation, demonstrating that, despite a greater LA size, remodelling is accompanied by low LA stiffness,[20] contrary to that found in patients with cardiomyopathies or AF. This suggests preserved compliance of the LA despite increased atrial size, which is primarily mediated via the increased stroke volume by exercise. The increased LA stiffness in non-athletes with AF would highlight that the alterations in the LA are a consequence of both LA remodelling as well as functional reduction in LA compliance.

Strain parameters

We observed a lower LV GLS in athletes compared with non-athletes. It is possible that, as a result of increased LV volume due to athletic remodelling, reduced myocardial deformation is required to obtain the same stroke volume; hence reduced LV GLS in athletes defines an adaptive change rather than subclinical myocardial dysfunction. This observation of reduced GLS in athletes is consistent with a recent study of Dores *et al.*[21] who observed a high prevalence of reduced GLS in athletes that was more common in those who were elite, had a higher volume of training and had greater ventricular and atrial volumes. In a similar manner in the RV, we have demonstrated reduced resting RV deformation in endurance athletes but normal RV contractile reserve during exercise, suggesting that lower RV deformation at rest may reflect physiological remodelling and chamber enlargement.[22] Certainly, this observation of reduced LV GLS in athletes

warrants further assessment. Indeed, recent meta-analyses and systematic reviews of athletes in different sports have shown marked heterogeneity in results, demonstrating greater, similar or even reduced GLS relative to non-athletic controls.[23–25]

In our study, LA reservoir and contractile strain were both reduced in athletes as well as non-athletes with AF. A recent meta-analysis (403 athletes and 297 controls) found that LA reservoir strain was generally reduced in athletes.[26] The larger LA volumes in athletes implies that they require less deformation at rest to eject similar volumes and it is also possible that athletes have the ability to augment strain to a greater extent during exercise.[27] However, this potential benefit to atrial performance is obtained at the cost of higher wall stress[28] that could potentially be a trigger for inducing atrial fibrosis, a known risk factor for development of AF.[29] Reduced LA reservoir strain in non-athletic populations has been shown to be a strong predictor of AF occurrence.[30] A reduction in LA contractile function is also associated with increased risk of AF.[31] Whether these changes in athletes are only benign adaptations to intensive training or represent areas of significant LA structural alterations that are potential substrates for AF needs to be explored further.

Within athletes, we did not identify any differences in LA strain parameters in those with or without AF. To the best of our knowledge, there is only one previous study by Hubert *et al.*[32] that has evaluated LA strain in athletes with regards to AF. Twenty-seven male endurance veteran athletes with AF were compared with 30 control endurance athletes without AF, with similar training level, age, and risk factors. The authors concluded that veteran male endurance athletes with AF had impaired LA reservoir strain compared with those without AF. However, there are some differences in our study, with younger participants enrolled with more stringent endurance training criteria. Our study also demonstrated increased LA and LV volumes in athletes. It is possible that athletes in the study by Hubert *et al*. were at earlier stages of athletic remodelling compared with athletes in our study. The impaired LA strain in AF athletes at earlier stages of remodelling may represent stretch-induced alterations in myocardial deformation. With increasing intensity and duration of athletic training, as in our study, athletes likely undergo greater 'athletic' remodelling, possibly nullifying the effects of AF on myocardial deformation. This may suggest that the definition of an endurance athlete or the degree of athletic remodelling may play a significant role in modulating the effects of AF on LA function in athletes.

Possible mechanisms of AF in athletes

A study of LA structural and functional remodelling in athletes provides important mechanistic insights of the group in which AF is more prevalent. Our study demonstrates that atrial dilation occurs in athletes, proportional to the LV and is consequent to the demands of an increased cardiac output; however, unlike AF in non-athletic populations, is independent of diastolic dysfunction and atrial stiffening. However, we observed several measures of atrial contractile function that are impaired in both athletic and non-athletic subjects with AF. Thus, we conclude that atrial contractile dysfunction is an endpoint associated with AF that is mediated by impaired diastolic relaxation in non-athletes but not in athletes. We speculate that in athletes, a co-existent atrial myopathy may be a consequence of frequent exposure to elevated atrial wall stress during exercise (Graphical Abstract). Although these two differing mechanisms in athletes and non-athletes likely contribute to LA remodelling and fibrosis in distinct ways, the final consequence in both scenarios appears to be clinically similar. It is intriguing to speculate that the different remodelling mechanisms may manifest as different effects on atrial electrophysiology. However, we are yet to identify many differences in the clinical behaviour of AF in athletes and the initial experience suggests that PVI is similarly efficacious in athletic and non-athletic populations.[33–35]

Study limitations

Most of our participants were males and given the increasing involvement of females in endurance sports, additional studies are required in female athletes. Athletes were selected from a single centre, and future multicentre studies would be valuable to validate the current findings. AF athletes and non-athletes had paroxysmal AF, and hence were at an earlier stage of AF pathogenesis. This attenuated changes in LA remodelling and function due to the AF itself. Further studies in athletes and non-athletes with more advanced forms of the disease, such as persistent AF, may provide further insight into progressive LA remodelling. Furthermore, a longitudinal study is required to determine if the markers associated with AF in this study can predict athletes at risk of developing AF. Finally, a greater percentage of individuals with AF were on antiarrhythmic medications (both athletes and non-athletes) in our study. We accept that medication use can affect LA and LV function; however, after excluding individuals on medications, our small sample size did not allow performing a sensitivity analysis to assess this further.

Conclusions

This cross-sectional study highlights mechanistic and functional differences in athletes and non-athletes with AF, suggesting distinct pathophysiological mechanisms. Diastolic dysfunction, atrial dilation, and reduced LA strain defined non-athletes with AF. In athletes, atrial enlargement and reduced atrial strain were universal with only subtle further changes in those with AF. It is therefore important to recognize that atrial measures considered abnormal in the general population are common in athletes. Despite this, differences in atrial contractile function are appreciable in athletes with AF and raise the possibility that endurance training can promote an atrial myopathy in a subset of athletes as opposed to the non-athletic population where diastolic dysfunction appears to be the key driver for AF.

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Data availability

The data underlying this article will be shared on reasonable request to the corresponding author.

Conflict of interest: none declared.

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