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Atrial remodeling and ectopic burden in recreational athletes: Implications for risk of atrial fibrillation

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Background: Atrial remodeling, vagal tone, and atrial ectopic triggers are suggested to contribute to increased incidence of atrial fibrillation (AF) in endurance athletes. How these parameters change with increased lifetime training hours is debated.

Hypothesis.: Atrial remodeling occurs in proportion to total training history, thus contributing to elevated risk of AF.

Methods: We recruited 99 recreational endurance athletes, subsequently grouped according to lifetime training hours, to undergo evaluation of atrial size, autonomic modulation, and atrial ectopy. Athletes were grouped by self-reported lifetime training hours: low (<3000 h), medium (3000–6000 h), and high (>6000 h). Left atrial (LA) volume, left ventricular (LV) dimensions, and LV systolic and diastolic function were assessed by echocardiography. We used 48-hour ambulatory electrocardiographic monitoring to determine heart rate, heart rate variability, premature atrial contractions, and premature ventricular contractions.

Results: LA volume was significantly greater in the high (+5.1 mL/m^2, 95% CI: 1.3–8.9) and medium (+4.2 mL/m^2, 95% CI: 0.2–8.1) groups, compared with the low group. LA dilation was observed in 19.4%, 12.9%, and 0% of the high, medium, and low groups, respectively (P = 0.05). No differences were observed between groups for measures of LV dimensions or function. Minimum heart rate, parasympathetic tone expressed using heart rate variability indices, and premature atrial contraction and premature ventricular contraction frequencies did not differ between groups.

Conclusions: In recreational endurance athletes, increased lifetime training is associated with LA dilation in the absence of increased vagal parameters or atrial ectopy, which may promote incidence of AF in this cohort.

KEYWORDS

Arrhythmias, Heart Rate, Sports Cardiology, Training History

1 | INTRODUCTION

There are well-documented benefits of exercise for the maintenance of cardiovascular health, such that even the long-term practice of endurance exercise reduces all-cause mortality.[1] However, a heightened risk of atrial arrhythmias (eg, atrial fibrillation [AF]) associated with long-term endurance training has been described in a number of independent cohorts.[2–4]

Endurance exercise induces a series of structural, functional, and electrophysiological adaptations within the heart, including biatrial enlargement and eccentric hypertrophy of the ventricles with little to no change in systolic function.[5] Ventricular compliance is improved

likely contributing to an improvement in ventricular filling.[6] In trained individuals, enhanced parasympathetic activity[7] with sinus bradycardia is one of the more established cardiovascular adaptations. In addition there is evidence for an alteration in the intrinsic electrophysiological properties of the heart.[8]

Despite the performance improvement that accompanies the "athlete's heart," certain maladaptations may mediate the increased AF risk in this cohort.[9,10] Increased occurrence of premature atrial contractions (PACs) has been reported in athletes with greater accumulated training hours,[11] which may represent a potential trigger for the onset of atrial arrhythmias.[12] This trigger can initiate a sustained AF episode dependent on the presence of an arrhythmogenic atrial substrate.[13] The atrial substrate for AF is characterized by atrial dilatation and fibrosis predisposing to atrial reentry, whereas increased vagal tone shortens the atrial refractory period, thus facilitating reentry and potentially maintaining AF.[14]

Although numerous studies have examined the electrocardiographic (ECG) and echocardiographic characteristics of highly trained athletes,[15,16] few have examined the dose–response of endurance exercise and the potential pathophysiological mechanisms underlying an increased risk of AF in a cohort of recreational endurance-sport participants. In >50 000 endurance-race participants, the risk of AF was highest in those with the most previously completed races,[2] thus indicating a role of training history in mediating AF risk. Importantly, the excess of AF among those who engage in endurance training is not typically confined to those in elite sports.[17] Therefore, we hypothesize that atrial remodeling may occur more profoundly in endurance-sports participants, thus lending to an elevated AF risk in this cohort.

2 | METHODS

We recruited 99 participants from the local sporting community. The inclusion criteria were endurance-sports participants age 18 to 80 years, engaging in ≥5 hours per week of endurance exercise for at least the past 12 months, and free of known cardiovascular disease. Participants with known hypertension, type 2 diabetes mellitus, or hypercholesterolemia were excluded. Each participant completed a health questionnaire declaring known medical issues or medications known to alter heart rhythm. All participants were further screened regarding their lifetime exercise training history, where they were asked to declare the number of years they have been training and the average number of training hours per week during this time. Lifetime training hours were subsequently calculated according to the following formula: Lifetime training hours = (52 × training hours per week) × number of years training. Using this data, participants were subsequently separated into 3 groups: low, 0 to 2999 hours; medium, 3000 to 5999 hours; and high, >6000 hours. The study was approved by the Human Research Ethics Committee at the University of Adelaide.

All patients were reviewed in the ambulatory cardiac clinic. After completing study questionnaires, participants undertook ambulatory Holter monitoring for a 48-hour period and within 7 days a standardized echocardiographic examination. Three-lead Holter monitoring (leads I, II, and III; sample rate: 256 Hz) was performed over a 48-hour period using Medilog AR12 monitors (Schiller AG; Baar, Switzerland). The recording was imported into Medilog Darwin analysis software (Schiller AG) and analyzed by 2 investigators blinded to training history. R-R intervals were used as a surrogate for atrial coupling, as they closely reflect atrial coupling during ectopy.[18] ECG periods with significant movement/muscle artifact were removed. Premature atrial contractions (PACs) were detected automatically as a minimum 25% reduction in the R-R interval compared with the previous normal R-R coupling. All automatically detected PACs were manually verified for inappropriate beat annotation. Following the establishment of an ectopic beat, classification of the beat as atrial or ventricular in origin was based on the visible presence of atrial activity preceding the ventricular activation and/or the absence of a change in the QRS duration and morphology to sinus rhythm. In the presence of a change in QRS duration or morphology the beat was classified as ventricular in origin. Mean, minimum, and maximum heart rates were determined for each recording in addition to time-based heart rate variability (HRV) parameters, including the standard deviation of normal-to-normal intervals and root mean square of successive differences. All PACs and PVCs, as well as the following normal beat, were excluded from HRV analyses. All participants were asked to refrain from exercise in the 12 hours preceding and during each monitoring period.

All participants underwent a transthoracic echocardiography using a Vivid 7 Dimension echocardiograph (GE Vingmed Ultrasound, Horten, Norway) according to most recent American Society of Echocardiography (ASE) guidelines.[19] An experienced sonographer, blinded to training group, performed all echocardiograms. Left ventricular (LV) dimensions and ejection fraction were obtained using 2-dimensional and M-mode echocardiography. Peak early (E) and late diastolic filling (A) velocities were obtained using pulsed-wave Doppler from samples at the tips of the mitral valve. Mitral annular tissue velocities were sampled using tissue Doppler imaging of the septal and lateral walls, respectively. Interventricular septum diameter was assessed using 2-dimensional scans taken from the parasternal long-axis view. LV mass was calculated according to Devereux formula.[20] Left atrial (LA) volume was assessed and averaged from the apical 2-chamber and 4-chamber views, respectively. Where applicable, all cardiac dimensions were indexed to body surface area.

All data were analyzed with SPSS 22 (SPSS Inc., Chicago, IL). Data are presented as mean ±SD unless otherwise stated. For normally distributed continuous variables, differences between the 3 training groups were compared by analysis of variance with Sidak-adjusted pairwise comparisons. Non–normally distributed data were compared between the 3 groups using the Kruskal-Wallis test, followed by pairwise Mann–Whitney *U* tests when required. Categorical data were compared using the χ^2 statistic. Multivariate regression analysis was used to determine the independent predictors of LA volume that differed between the 3 groups. A *P* value of <0.05 was considered statistically significant.

3 | RESULTS

At baseline, groups were well matched with regard to height and body mass (Table 1). Participants in the lower-training-hours group were

TABLE 1 Baseline characteristics of groups according to lifetime training history

	Low Group, <3000 h	Medium Group, 3000–6000 h	High Group, >6000 h	*P* Value
No.	30	31	35	
Age, y	45 ± 9	51 ±9	54 ± 10	0.001
Height, cm	174 ±9	173 ±9	173 ±9	0.97
Body mass, kg	73 ±10	73 ±11	70 ±10	0.47
BMI, kg/m^2	24.2 ±2.4	24.1 ±2.5	23.3 ±2.5	0.33
Mean lifetime training hours	1684 (560–2912)	4533 (3000–5850)	12 696 (6240–37 000)	<0.001
Lifetime training years	6 ±3	15 ±7	24 ±10	<0.001

Abbreviations: BMI, body mass index; IQR, interquartile range; SD, standard deviation Data are presented as mean ± SD or median (IQR).

younger than those in the medium and high groups ($P < 0.001$; Table 1). Participants in the high-training-hours group engaged in a greater number of training years vs those in the medium and low groups, respectively (24.3 ±10.5 vs 15.2 ±7.2 vs 6.4 ±3.2 years, $P < 0.001$; Table 1).

There was a significantly greater LA volume (Figure 1) in both the high (34.3 ±7.3 mL/m^2) and medium (33.4 ±6.1 mL/m^2) training groups when compared with the low training group (29.3 ±4.7 mL/m^2). The percentage of athletes meeting the definition of moderate to severe atrial dilation[19] (>42 mL/m^2) was 19.4% and 12.9% in the high and medium training groups, respectively, with no athletes in the low training group meeting the atrial dilation criteria ($P = 0.05$). Lifetime training hours was a significant predictor of LA size, even after adjustment of baseline differences in age ($P = 0.006$). There was no difference in the P-wave duration between groups, even after age adjustment ($P = 0.87$).

There were no significant differences between groups for LV dimensions, wall thickness, or mass (Table 2). As expected, LV systolic function was normal in all participants with no between-group differences. There was a trend toward a significantly greater E/A ratio in the high training group, although this did not reach statistical significance ($P = 0.08$). There were no between-group differences for E/e' ($P = 0.58$).

The mean heart rate (HR) across the entire 48-hour recordings tended to be lower in the high training group, although this did not reach statistical significance ($P = 0.07$). The minimum HR, expressed as the lowest HR throughout nighttime hours, did not differ between groups, although the lowest hourly average HR was seen for the high training group (53 ±7, 52 ±5, and 49 ±5 bpm for the low, medium, and high groups, respectively; $P = 0.05$). No such episodes were recorded in any other athlete. To determine autonomic contributions, we compared 3 time-based measures of HRV between groups. There were no significant differences with regard to standard deviation of normal-to-normal intervals, root mean square of successive differences, or pNN50 measures recorded over the 24-hour period (Table 2).

There was no significant difference between the PAC or PVC counts for each group (Table 2). Likewise, the number of athletes with >100 PACs or PVCs per 24-hour recording period was similar between groups ($P = 0.2$).

Of all athletes, there were 3 significant clinical findings. One athlete was detected to have asymptomatic nocturnal bradycardia (HR, 23 bpm) and an asymptomatic nocturnal sinus pause of 8 seconds. A short period of detraining resolved these observations without need for further intervention, perhaps suggestive of a reversible vagal involvement. One athlete had significant ventricular ectopy burden

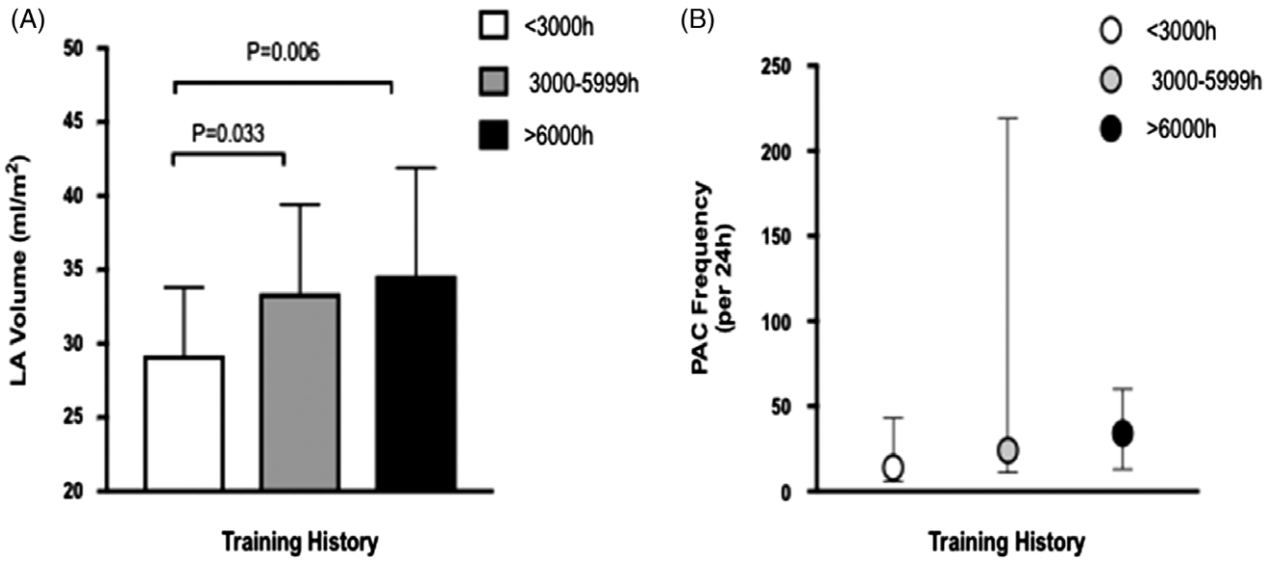

FIGURE 1 (A) LA indexed volume and (B) PAC frequency classified according to lifetime training hours. Abbreviations: LA, left atrial; PAC, premature atrial contraction

TABLE 2 LV structure and function and ambulatory Holter findings by training group

	Low Group, <3000 h	Medium Group, 3000–6000 h	High Group, >6000 h	*P* Value
Echocardiography				
LA volume, mL/m^2 (indexed)	29.3 ± 4.7	33.4 ± 6.1	34.3 ± 7.3	0.005
LA diameter, cm	3.5 ± 0.4	3.7 ± 0.4	3.7 ± 0.4	0.3
LV dimension, cm/m^2 (indexed)	2.7 ± 0.3	2.7 ± 0.3	2.8 ± 0.2	0.13
LV mass, g/m^2 (indexed)	86 ± 16	93 ± 18	95 ± 18	0.08
LV IVSd, cm	0.9 ± 0.1	1.0 ± 0.1	1.0 ± 0.1	0.08
LV PWd, cm	0.9 ± 0.1	0.9 ± 0.1	1.0 ± 0.1	0.30
RAA	16.1 ± 2.6	16.6 ± 2.4	17.6 ± 3.3	0.1
E/A	1.5 ± 0.5	1.5 ± 0.5	1.7 ± 0.6	0.09
E/E′	7.4 ± 2.3	8.0 ± 2.0	7.7 ± 2.3	0.58
48-h Ambulatory Holter monitoring				
Mean HR, bpm	67 ± 8	65 ± 8	62 ± 7	0.14
Minimum hourly HR, bpm	53 ± 7	52 ± 5	49 ± 5	0.02
PAC burden, per 24 h	14 (6–43)	25(11–219)	34(13–60)	0.1
PVC burden, per 24 h	2 (1–12)	2 (1–79)	3 (1–12)	0.98
SDNN, ms	173 ± 38	163 ± 35	167 ± 34	0.57
RMSSD, ms	49 ± 15	46 ± 19	42 ± 9	0.28

Abbreviations:A, late mitral filling velocity;E, early mitral filling velocity; E′, early annular motion velocity; HR, heart rate; IQR, interquartile range; IVSd, interventricular septum diameter end-diastole; LA, left atrial; LV, left ventricular; PAC, premature atrial contraction; PVC, premature ventricular contraction; PWd, posterior wall diameter end-diastole; RAA, right atrial area; RMSSD, root mean square of successive differences; SD, standard deviation; SDNN, standard deviation of normal-to-normal intervals.
Data are presented as mean ± SD or median (IQR).

(17 000 PVCs per 24 hours), and subsequently underwent PVC ablation. In this case, the PVCs were of a right-sided origin and were terminated by ablation in the posterior papillary muscle region. There were no features of adverse RV or LV remodeling on cardiac magnetic resonance imaging. One athlete had a single 2-minute asymptomatic episode of AF detected on Holter monitoring and was referred for further management. All 3 athletes were in the high training group.

4 | DISCUSSION

A growing body of evidence supports the view that endurance exercise training promotes an increased risk of AF. However, the identification of the at-risk athlete remains elusive. This study provides insight into pathophysiological changes that may predispose to AF. There are 3 important findings from this study: (1) LA size appears to increase with lifetime training hours, and this appears somewhat independent of the degree of LV remodeling; (2) more profound bradycardia occurs in those with greater lifetime training history as compared with lesser amounts. The lack of difference in HRV raises the possibility that this may be independent of vagal influences; and (3) exercise history, within the range studied, has minimal impact on the frequency of atrial or ventricular ectopic burden.

Recent data show an accumulation of risk with more prolonged exercise training histories,[21] whereby a lifetime training history >2000 hours results in an almost 4-fold elevation in risk of incident AF. Given that elite endurance athletes achieve annual training volumes >800 hours per year,[22] this issue is likely to extend to the more recreational athlete training at more modest volumes over several years.

Exercise training is associated with biatrial enlargement[23] that can create an arrhythmogenic substrate potentially contributing to the maintenance and progression of AF. Our data show that LA enlargement occurs even with moderate training histories >3000 hours. Importantly, lifetime training history remained a significant predictor of LA volume, even after adjustment for age. Our data are consistent with that reported previously in which runners with more extensive training histories reported the largest atrial size.[11] Likewise, the percentage of athletes showing LA enlargement in our study (12%–20% among the medium and high training groups) closely resembles the 20% reported in a study of >1700 competitive athletes previously.[24] Increased LA size has been reported as an important predictor of AF occurrence among endurance-sports participants.[4] Although we did not observe longer atrial activation times determined by P-wave duration, additional factors such as the development of atrial fibrosis are likely to contribute to the arrhythmogenic substrate by atrial local conduction disturbances in endurance athletes. Indeed, preclinical data show the development of atrial fibrosis concomitant with increased AF inducibility in exercise-trained rats.[25] Intriguingly, there was a strong temporal relationship between the development of atrial fibrosis and the inducibility of AF, suggesting that atrial remodeling with more extensive fibrosis is necessary for the promotion of AF with endurance exercise.

One of the hallmark changes associated with endurance training is sinus bradycardia. We showed evidence of more profound bradycardia among the high training group, which may be significant given the recent interest in bradycardia as an additional independent risk factor for AF.[26] Orthodox teaching would argue that bradycardia in athletes is attributed to enhanced parasympathetic tone and these autonomic changes may contribute to the development of AF by

shortening of the atrial refractory period, thus facilitating reentry.[14,27] However, some preclinical and human data have challenged the view that bradycardia is explained by excess vagal tone, raising speculation that bradycardia may be due to more permanent structural remodeling of the sinoatrial node and its ion-channel constituents.[8,28] Further longitudinal studies are required to evaluate autonomic shifts and intrinsic electrophysiology of athletes throughout extended periods of endurance training.

PACs have been implicated as a potent trigger for AF. In athletes, the frequency of PACs is reportedly higher in those with longer training histories.[11] However, we found no group differences with regard to PAC frequency. Moreover, the overall burden of PACs was low. Although the presence of high PAC burden in athletes with AF cannot be ruled out, this evidence suggests that exercise training does not critically contribute to an excess of PACs. The cause of the disparity in findings between our study and others published previously with regard to the training effect on ectopic burden is unclear, although potentially due to the slightly older participants in our study and the modestly higher burden of atrial ectopy in our study compared with that reported previously.[11] Future studies in athletes of different age ranges will be critical in confirming the findings reported here. Furthermore, insights from continuous ECG monitoring of elite endurance athletes may reveal a different burden of atrial ectopy from that seen in recreational athletes.

Although our aim was to evaluate the effect of lifetime training history on potential arrhythmogenic risk factors in athleteswe observed only 1 episode of AF in our training groups. It isthereforenot possible to directly assess any causal link between the cardiac adaptations observed here and AF risk. Howeverthis data suggest that atrial enlargement is an early adaptive response to exercise training that potentially explains the increased risk of AF among recreational endurance athletes. Interestinglythe atrial adaptations observed here occurred in isolation with minimal influence of training history on LV function or structure. Given the strong correlation between LV size and exercise performance,[29] this finding underscores the clinical observation that training-induced AF is not confined only to elite athletes or those with the highest cardiorespiratory fitness.

4.1 | Study limitations

Several limitations should be considered when interpreting this study. Our recruitment focused on recreational athletes in the absence of a control group consisting of age-matched nonathletes. Calculation of lifetime training hours was performed as per previous studies[11] using recall and estimation of training history. Additionally, we did not determine training intensity and therefore cannot make any inferences regarding training load and cardiac changes with exercise training. The study included a small sample size, which may have led to type II error for some measures. Furthermore, our study population included predominantly males, which may limit the application of these findings to a female cohort. We also used echocardiography to determine cardiac structure, which may produce findings that differ with other imaging techniques such as cardiac magnetic resonance. Finally, we indirectly assessed autonomic function using HRV indices, which may limit the interpretation of the vagal indices described in this study.

5 | CONCLUSION

In amateur endurance-sports participants, increased lifetime training hours is associated with atrial dilation in the absence of significant differences in vagal tone, atrial ectopic trigger burden, or LV size. Future prospective studies should examine these measures in response to chronic training in a long-term follow-up to further elucidate the mechanisms underlying the increased AF risk in middle-aged endurance athletes.

Conflicts of interest

R.M. has received lecture and/or consulting fees and research funding from St. Jude Medical and Medtronic. P.S. has received lecture and/or consulting fees from Biosense Webster, Medtronic, St. Jude Medical, and Boston Scientific; has received research funding from Medtronic, St. Jude Medical, Boston Scientific, Biotronik, and Sorin; and has served on the advisory boards of Biosense Webster, Medtronic, St. Jude Medical, Boston Scientific, and CathRx. The authors declare no other potential conflicts of interest.

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