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FULL RESEARCH PAPER
Sports cardiology

Exercise blood pressure response does not differentiate endurance athletes with paroxysmal atrial fibrillation

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Aims Atrial fibrillation (AF) is more prevalent among endurance athletes than in the general population. An exaggerated blood pressure (BP) response to exercise may contribute to atrial pressure excess and AF risk. We hypothesized that higher exercise BP would be associated with left atrial enlargement and AF prevalence in endurance athletes.

Methods and results One hundred seventy-eight endurance-trained athletes, 89 with paroxysmal AF [median age 55 (IQR: 46–62 years), 92% male] and 89 age- and sex-matched athletes without AF, underwent resting echocardiography and cardiopulmonary exercise testing with automated BP measurements. Athletes with hypertension were excluded. Absolute BP responses [maximal systolic (SBP_{max}) and diastolic (DBP_{max})] and BP responses relative to exercise workload (W) (SBP/W-slope, DBP/W-slope, and the SBP_{max}/W-ratio) were compared between groups. Both groups had similar years of training ($P = 0.83$) and peak oxygen uptake ($P = 0.34$). Athletes with AF had significantly larger left atrial volumes (LAVi; 48 mL/m^2 vs. 42 mL/m^2, $P < 0.001$), higher mean resting SBP (132 ± 13 mmHg vs. 127 ± 14 mmHg, $P = 0.018$), and DBP (76 ± 10 mmHg vs. 73 ± 11 mmHg, $P = 0.090$) compared with athletes without AF. There were no differences in SBP_{max} (221 ± 23 mmHg vs. 219 ± 26 mmHg, $P = 0.54$) or DBP_{max} ($P = 0.84$) in athletes with and without AF, respectively. In athletes with AF, SBP/W-slope was lower, whilst DBP/W-slope and SBP_{max}/W-ratio were similar to athletes without AF ($P = 0.022$, $P = 0.63$, and $P = 0.82$, respectively). Furthermore, there was no difference in exercise BP responses in those athletes with and without severely dilated LAVi.

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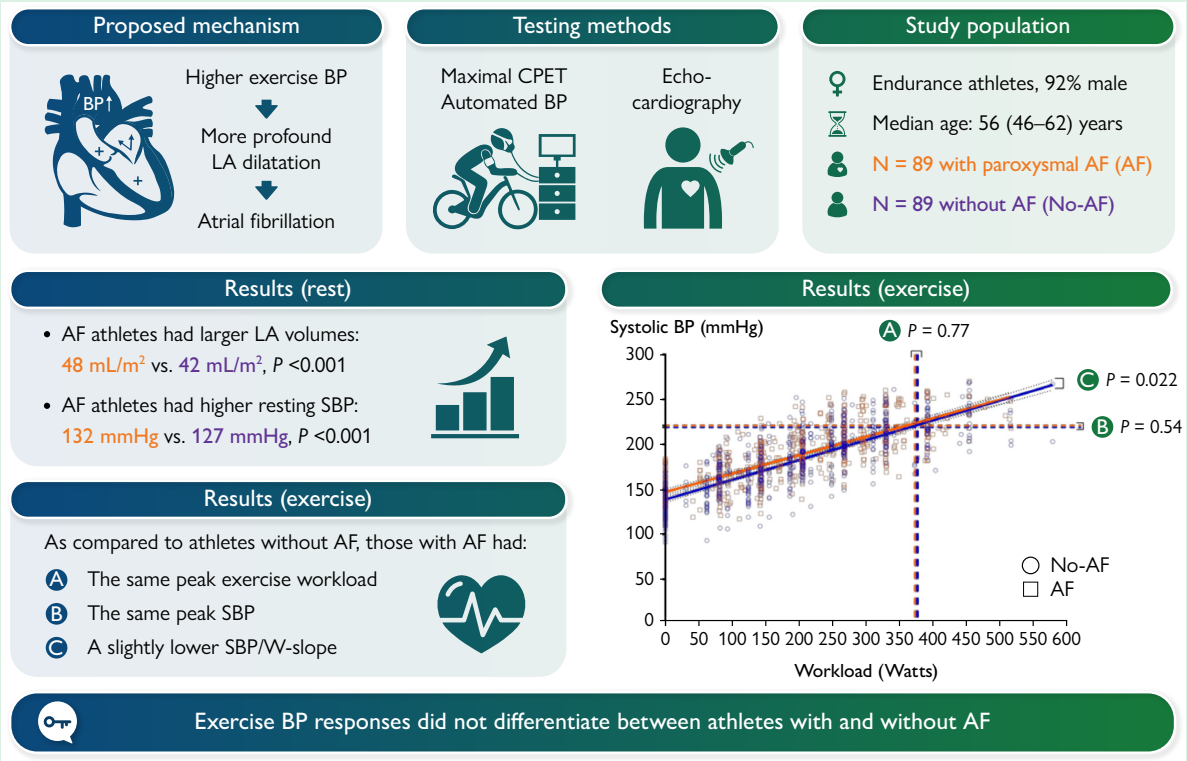
Conclusion In endurance athletes, neither AF nor left atrial dilation was associated with higher exercise BP responses.

Lay summary Atrial fibrillation is the most common cardiac arrhythmia in endurance athletes, but the underlying mechanisms remain unknown. We hypothesized that high BP during exercise may cause atrial dilatation and potentially explain why otherwise healthy endurance athletes develop AF. In this study, we evaluated and compared the BP response to exercise in 178 endurance athletes, half of whom were diagnosed with paroxysmal AF, to see if those athletes with AF exhibited an exaggerated BP response during exercise.

- All athletes reached high maximal systolic BP during exercise, but there was no difference in maximal BP between groups.
- When assessed relative to exercise workload, athletes with AF had a slightly *lower* SBP/W-slope.
- Atrial dilation was common among athletes, more so in those with AF, but was not associated with BP during exercise.
- Overall, we found no evidence of an association between exercise BP responses and the presence of AF, indicating that the heightened risk of AF in athletes may be attributable to other factors.

Graphical Abstract



Keywords Athlete • Atrial fibrillation • Blood pressure • Cardiopulmonary exercise testing • Endurance exercise • Hypertension

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Introduction

Atrial fibrillation (AF) is the most prevalent sustained arrhythmia in both the general population and in endurance-trained athletes. As compared with non-athletes, several cohort studies have revealed a two- to seven-fold increased risk of AF in individuals practising endurance sports such as running, rowing, and cross-country skiing.[1–4] Whilst AF in the general population is often associated with cardiovascular risk factors,[5] these are often absent in athletes who develop AF. The mechanisms underpinning this excess risk of AF in athletes remain speculative. However, increased susceptibility to AF has been associated with exercise-induced atrial remodelling comprising enlargement,[6,7] fibrosis and inflammation,[8] increased vagal tone,[9,10] and genetic predisposition.[1,11,12]

In the general population, hypertension is a well-established risk factor for AF,[13] likely as a consequence of left atrial (LA) remodelling caused by afterload-induced chronic left ventricular

pressure overload and resultant increases in chamber stiffness. Although most endurance athletes are normotensive at rest, the contribution of haemodynamic stress, particularly pressure overload during exercise, to AF risk in this population remains under-explored.

Recent evidence suggests that endurance exercise can impose a significant pressure load on the heart, particularly during high-intensity efforts.[14] Some athletes have markedly elevated systolic blood pressure (SBP) during exercise, both at maximal (i.e. SBP_{max}) and at submaximal workloads (i.e. steep SBP-to-workload slopes).[14] These findings have led to speculation that repetitive pressure loading accumulated over multiple years of athletic training and racing drives atrial remodelling in athletes. Given the association between exaggerated BP responses to exercise and masked hypertension in non-athletes,[15–17] a comparable haemodynamic stressor may be disproportionately prevalent among athletes with AF.

Therefore, we hypothesized that a higher SBP_{max} and a steeper BP-to-workload slope would be observed in (i) endurance athletes with AF as compared with endurance athletes without AF and (ii) in athletes with severe LA dilation as compared with those without severe LA dilation. To interrogate this hypothesis, we compared automated BP measures throughout graded exercise testing between endurance athletes with and without paroxysmal AF.

Methods

Study design and participants

This case-control analysis included data from three international, multicentre studies: The ProAFHeart study (ACTRN12618000711213), the NEXAF Detraining study (NCT04991337), and the Master@Heart study (NCT03711539). The ProAFHeart study is a prospective cohort study investigating atrial remodelling and arrhythmia risk in endurance athletes. NEXAF Detraining is a randomized controlled trial assessing effects of training adaptation in endurance athletes with paroxysmal AF and no cardiovascular comorbidities. Master@Heart is a prospective cohort study evaluating the long-term cardiovascular effects of endurance training, including coronary atherosclerosis, AF, and myocardial fibrosis. Full protocols for NEXAF Detraining and Master@Heart have been published, and procedures for the ProAFHeart study were identical to those described in the Pro@Heart protocol.[18–20]

Ethical approval for the three studies was obtained from the local Ethics Committee at each participating site, the Alfred Hospital Ethics Committee, Melbourne, Australia (484/16, 470/21), for ProAFHeart and NEXAF Detraining; the Regional Committee for Medical and Health Research Ethics, Norway (212748), for NEXAF Detraining; and the UZ/KU Leuven Research Committee, Belgium (S61336), for Master@Heart. All participants provided written informed consent in accordance with the Declaration of Helsinki.

Participants were recruited and underwent testing at five sites: St. Vincent's Hospital and the Baker Heart and Diabetes Institute (Melbourne, Australia); Bærum Hospital (Gjettum, Norway); St. Olav's Hospital (Trondheim, Norway); and University Hospital Leuven (Leuven, Belgium).

All participants were aged 16–80 years and met sport-specific endurance training criteria: ≥5 h/week of running or rowing or ≥8 h/week of cycling, cross-country skiing, or triathlon. Athletes were classified as Tier 2–5 (trained to world-class level) using the participant classification framework.[21] All assessments were conducted when the participants were in sinus rhythm. Study specific inclusion and exclusion criteria are detailed in Supplementary material online, *Table S1*.

Case definition (AF cohort)

The cases were endurance athletes with paroxysmal AF confirmed by 12-lead electrocardiogram. Participants were either athletes referred to specialist cardiology services for AF management or retired elite rowers with a documented history of paroxysmal AF from the ProAFHeart study.[1] The AF cohort also included individuals with recurrent AF after pulmonary vein ablation and those free of AF recurrence for >5 years after successful pulmonary vein ablation and athletes with documented AF who had undergone previous right atrial (RA) ablation for atrial flutter.

Control definition (no-AF cohort)

Controls were endurance-trained athletes with no prior history of AF, matched 1:1 to AF cases by age (±5 years) and sex.

Exclusion criteria for both cohorts were permanent AF, diagnosed arterial hypertension requiring antihypertensive therapy, body mass index (BMI) > 30 kg/m^2, cardiomyopathy, or frequent or sustained ventricular arrhythmias.

Study protocol

Cardiovascular profile and training history

Participants completed a questionnaire assessing medical history, cardiovascular risk factors, sports and exercise history (including weekly training hours, sport type, and years of participation), and regular daily medication use at the time of their study evaluation.

Resting blood pressure and heart rate measurement

Resting office BP was recorded in the supine position following 5–10 min of rest. When three measurements were obtained, the first measurement was discarded, and the mean of the subsequent two measurements was used. Blood pressure was measured using validated digital automatic BP machines (Omron HEM-907XL Pro BP Monitor or Omron model M6W, Omron, Kyoto, Japan, or Tango M2 BP Monitor, Suntech Medical Inc., NC, USA) with an appropriately sized cuff. Resting heart rate was taken from a resting 12-lead electrocardiogram. Office hypertension was defined as a systolic BP (SBP) ≥ 140 mmHg and/or diastolic BP (DBP) ≥ 90 mmHg.[22]

Cardiopulmonary exercise testing

All centres used the same cardiopulmonary exercise test (CPET) protocol and comparable exercise equipment. A maximal CPET was conducted on an electronically braked bicycle ergometer (LODE Excalibur Sport or LODE Corival, Groningen, The Netherlands, or Avantronic Cyclus 2, Leipzig, Germany) to assess BP responses during exercise and peak oxygen uptake ($\dot{V}O_{2peak}$). Two minutes of passive resting data on the bike were obtained after which the participants performed a 1–3-min warm-up at an initial resistance of 40–60 Watts (W). Thereafter, the workload increased progressively at a rate of 20–30 W/min until volitional fatigue. Maximal workload (W_{max}) was defined as the highest power output achieved. Peak heart rate (HR_{peak}) was recorded from continuous 12-lead electrocardiography and was defined as the maximal heart rate in sinus rhythm at peak exercise. Continuous gas exchange data were collected using a calibrated metabolic cart (Vyntus CPX Metabolic Cart, Vyaire Medical, GmbH, Germany, or Cortex Metalyzer 3b, Leipzig, Germany). Peak oxygen uptake was determined as the highest value from a 30-s rolling average of 5-s averaged data. Individual percentage of predicted $\dot{V}O_{2peak}$ was calculated using the FRIEND equation.[23]

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Blood pressure during exercise

Blood pressure during CPET was measured using a validated auscultatory automated BP device incorporating R-wave gating from QRS-complexes and a noise reducing signalling processing system to facilitate accurate detection of K-sounds (Tango® M2 ECG-gated Automated BP Monitor, Suntech Medical Inc., NC, USA).[24] In accordance with guideline recommendations, measurements were taken whilst seated at rest on the bike and subsequently every 2 min throughout the test.[25] Maximal SBP (SBP_{max}) was defined as the highest recorded SBP during exercise, and the corresponding DBP was defined as the maximal DBP (DBP_{max}). To define the relationship between BP and exercise workload, the corresponding power output was recorded for each BP measurement. Individual linear regression analysis, using the multiple BP measurements and corresponding workloads, was used to determine the SBP/W- and DBP/W-slope.[14] The SBP_{max}/W-ratio was calculated by dividing the last measured SBP during exercise by the corresponding workload.

Echocardiography

A resting transthoracic echocardiogram (TTE) was performed (Vivid E9 or E95 ultrasound system, GE Healthcare, Chicago, IL, USA) to assess atrial and ventricular volumes. Images were saved digitally for offline analysis using the same analysis methods and software (EchoPAC version 13, EchoPAC version 206, GE Vingmed Ultrasound, Horten, Norway). Body surface area (BSA) was calculated using the Du Bois formula. Left ventricular end diastolic volume (LVEDV) was measured by biplane disk summation. The LA volume was measured using the biplane method of discs at ventricular end-systole. Diastolic filling measurements included the mitral peak early inflow velocity (E) and the mitral peak late inflow velocity (A). The early diastolic mitral annular velocity (e′) was evaluated through pulsed-wave tissue Doppler, and the (E/e′) was calculated using the average of septal and lateral e′. The RA area was measured from an apical four-chamber view at end-systole, where the RA chamber was at its greatest dimension prior to tricuspid valve (TV) opening. The measurement was by planimetry of the inner endocardial border, excluding the area under the TV annulus and the RA appendage. Pulmonary artery systolic pressure (PASP) was measured non-invasively using Doppler echocardiography by measuring the tricuspid regurgitation (TR) velocity and assuming a right atrial pressure (RAP) of 3 mmHg for all measures. Where appropriate measurements were indexed to BSA.

$$PASP = (4 \times TR\ velocity^2) + RAP$$

Statistical analysis

Normality of distribution was assessed using the Shapiro–Wilk test. Data are presented as mean ± standard deviation (SD) for normally distributed variables, median [interquartile range (IQR)] for non-normally distributed variables, or counts and percentages for categorical variables. Fisher's exact test was used to compare categorical variables. Continuous variables were compared between groups using independent t-tests for parametric data or the Mann–Whitney U test for non-parametric data. To account for the repeated SBP measurements, a linear mixed-effects model was used with SBP as the dependent variable, workload and AF status and their interaction as fixed effects, and participant ID as a random effect. In addition, cumulative SBP exposure during exercise was quantified using the area under the SBP/W curve calculated for each participant using the trapezoidal method. Group difference in cumulative SBP exposure was assessed using independent t-tests. To assess the robustness of our findings, a sensitivity analysis was performed, excluding participants with office hypertension criteria (resting SBP ≥140 mmHg and/or DBP ≥90 mmHg) as well as individuals taking beta-blockers for rhythm control, given the antihypertensive properties of these agents. An additional sensitivity analysis was performed excluding athletes on beta-blockers as well as antiarrhythmic medications as both can have an influence on BP and heart rate. To evaluate the association between exercise BP responses and LA dilatation, independent of AF status, athletes with a severely dilated LA indexed to BSA (LA_i >48 mL/m^2)[26] were compared with athletes with a LA_i <48 mL/m^2. As a further sensitivity analysis, we investigated whether there were differences in exercise BP responses across the full spectrum of LA size from normal (16–34 mL/m^2), mildly enlarged (35–41 mL/m^2), moderately enlarged (42–48 mL/m^2), or severely enlarged (>48 mL/m^2). Statistical significance was set at *P*-value <0.05 (two-tailed). Statistical analyses were performed using IBM SPSS version 30.0 (IBM SPSS Inc., Chicago, IL, USA), and figures created using GraphPad Prism version 8.0.1 (GraphPad Software, San Diego, CA, USA).

Results

Participant characteristics

The study population included 178 athletes with median age of 55 (IQR: 46–62) years of which 92% were male (*Table 1*). Baseline characteristics were well matched between groups, including BMI and cardiovascular risk factors (*Table 1*). Two participants in the AF group had a permanent pacemaker to treat symptomatic bradycardia. In the AF group, the predominant sport was cycling (34%). The most common types of sport in the No-AF group were cycling (40%) and rowing (40%). The training load at the time of study was significantly less in athletes with AF when compared with the athletes without AF [8 (5–10) h vs. 10 (7–15) h, *P* = 0.005], despite similar years of regular endurance training (*P* = 0.83). Approximately 90% of both groups were current competitive athletes.

AF group characteristics

Eighty-four athletes (94%) had current symptoms of AF, 16 athletes (18%) had undergone at least one AF ablation procedure of which 14 athletes (16%) had recurrent AF after ablation, and seven athletes (8%) had a previous atrial flutter ablation. Oral anticoagulation was used by 15 athletes (17%), beta-blockers by two athletes (2%), and rhythm control medication by eight athletes (9%), including Class Ic agents by seven athletes (8%) and Class III agents (sotalol) by one athlete (1%) (*Table 1*).

In the No-AF group, one person was taking a beta-blocker, but otherwise, no athletes (cases or controls) were taking BP lowering medications (*Table 1*).

Resting office blood pressure

Mean resting SBP was significantly higher in athletes with AF compared with those without AF (132 ± 13 mmHg vs. 127 ± 14 mmHg, *P* = 0.018), and there was a similar trend towards higher resting DBP among those athletes with AF (76 ± 10 mmHg vs. 73 ± 11 mmHg, *P* = 0.090). However, the prevalence of office hypertension (BP ≥140/90 mmHg) did not differ significantly between groups (*P* = 0.17).

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Table 1 Baseline participant characteristics and comparison between endurance athletes without atrial fibrillation (AF) (no-AF) and with paroxysmal AF (AF)

Variable	No-AF (*n* = 89)	AF (*n* = 89)	*P*-value
Age (years)	56 (46–62)	55 (46–63)	0.97
Male, *n* (%)	82 (92.1)	82 (92.1)	1.00
Height (cm)	180.3 ± 7.8	181.8 ± 7.3	0.17
BMI (kg/m^2)	23.8 (22.1–25.8)	24.1 (22.6–25.5)	0.35
Overweight, (BMI >27 kg/m^2), *n* (%)	6 (6.7)	11 (12.4)	0.31
Resting SBP (mmHg)	127 ± 14	132 ± 13	0.018
Resting DBP (mmHg)	73 ± 11	76 ± 10	0.090
Office BP ≥140/90 mmHg, *n* (%)	12 (13.5)	20 (22.5)	0.17
Resting heart rate (b.p.m)	52 ± 9	51 ± 8	0.28
Cardiovascular Risk factors, *n* (%)			
Antihypertensive therapy[a]	0 (0.0)	0 (0.0)	1.00
Stroke	1 (1.1)	3 (3.4)	0.62
Coronary artery disease	0 (0.0)	3 (3.4)	0.25
Lipid treatment	3 (3.4)	7 (7.9)	0.33
Diabetes treatment	0 (0.0)	0 (0.0)	1.00
Previous smoking	9 (10.1)	14 (15.7)	0.37
Sport and training, *n* (%)			
Cycling	36 (40.4)	30 (33.7)	0.44
Rowing	36 (40.4)	13 (14.6)	<0.001
Running	7 (7.9)	15 (16.9)	0.11
Cross-country skiing	0 (0.0)	11 (12.4)	<0.001
Triathlon	10 (11.2)	11 (12.4)	1.00
Other	0 (0.0)	9 (10.1)	0.003
Current athlete	79 (88.8)	83 (93.2)	0.43
Former athlete	10 (11.2)	6 (6.7)	0.43
Years regular training (years)	35.0 (22.0–44.0)	37.0 (19.5–46.0)	0.83
Average hours/week exercise training	10.0 (6.5–15.0)	8.0 (5.0–10.0)	0.005
Rate control medication, *n* (%)			
Beta-blocker	1 (1.1)	2 (2.2)	1.0
Calcium channel blocker	0	0	N/A
Digoxin	0	0	N/A
Rhythm control medication, *n* (%)			
Class III agents (sotalol, amiodarone, dronedarone)	0	1 (1.1)	1.0
Flecainide	0	7 (7.9)	0.007
Antithrombotic medication, *n* (%)			
Antiplatelet agents	2 (2.2)	4 (4.5)	0.68
Anticoagulants	0	15 (16.9)	<0.001

Values are median (IQR), mean ± SD, or *n* numbers (%). The *P*-value is for comparison between AF status.
AF, atrial fibrillation; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure.
[a]Excluding beta-blockers.

Echocardiographic parameters

Indexed LA volumes were moderately enlarged, on average, in both groups but were significantly larger in athletes with AF compared with those without AF [47.6 (40.6–53.9) mL/m^2 vs. 42.0 (34.2–49.9) mL/m^2, $P < 0.001$]. Right atrial area did not differ significantly between groups [24.7 (20.9–28.5) cm^2 vs. 25.1 (20.8–29.0) cm^2, $P = 0.87$] for athletes with and without AF, respectively. The athletes with AF had significantly smaller LVEDV, including indexed LVEDV [76.8 (65.8–88.8) mL/m^2 vs. 85.0 (72.0–100.2) mL/m^2, $P = 0.002$], and the left atrioventricular (LA:LV) volume ratio was increased in athletes with AF [0.61 (0.51–0.75) vs. 0.49 (0.41–0.57), $P < 0.001$]. Left ventricular ejection fraction was higher in athletes with AF (59.2 ± 5.3% vs. 56.4 ± 4.6%, $P < 0.001$). Mitral valve E/A-ratios were within normal limits in both groups but were higher in the AF group [1.45 (1.19–1.87)] compared with the No-AF group [1.23 (0.89–1.50)], $P < 0.001$ (*Table 2*). PASP was normal in both groups but significantly higher in the group with AF compared with those without AF (*Table 2*).

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Table 2 Echocardiography measures and comparison by atrial fibrillation (AF) status, either without AF (no-AF) and with paroxysmal AF (AF)

Variable	No-AF (*n* = 89)	AF (*n* = 89)	*P*-value
BSA (m^2)	1.98 ± 0.16	2.01 ± 0.16	0.10
LVEDV (mL)	168.1 ± 35.3	155.5 ± 36.6	0.021
$LVEDV_i$ (mL/m^2)	85.0 (72.0–100.2)	76.8 (65.8–88.8)	0.002
LV EF (%)	56.4 ± 4.6	59.2 ± 5.3	<0.001
LA (mL)	83.0 (65.5–99.0)	92.9 (79.7–116.1)	<0.001
LA_i (mL/m^2)	42.0 (34.2–49.9)	47.6 (40.6–53.9)	<0.001
LA:LV volume ratio	0.49 (0.41–0.57)	0.61 (0.51–0.75)	<0.001
RA area (cm^2)	25.1 (20.8–29.0)	24.7 (20.9–28.5)	0.87
E/e'	5.6 (4.8–7.1)	6.0 (4.8–6.7)	0.70
MV E/A ratio	1.23 (0.89–1.50)	1.45 (1.19–1.87)	<0.001
PASP (mmHg)[a]	21 ± 5	24 ± 4	<0.001

Values are mean ± SD or median (IQR).
AF, atrial fibrillation; BSA, body surface area; LVED, left ventricular end-diastolic volume; $LVEDV_i$, LVEDV indexed to BSA; LV EF, left ventricular ejection fraction; LA, left atrial volume; LA_i, LA indexed to BSA; RA, right atrial; E/e', mitral inflow to annular early diastolic velocity; MV E/A ratio, mitral valve early (E) to late (A) diastolic flow velocity ratio; PASP, pulmonary artery systolic pressure.
[a]*n* = 82 for No-AF and *n* = 69 for AF.

Table 3 Comparison of exercise testing characteristics and blood pressure response to exercise in endurance athletes without atrial fibrillation (AF) (No-AF) and with paroxysmal AF (AF)

Variable	No-AF (*n* = 89)	AF (*n* = 89)	*P*-value unadjusted
$\dot{V}O_{2peak}$ (L/min)	3.64 ± 0.81	3.68 ± 0.79	0.75
$\dot{V}O_{2peak}$ (mL/kg/min)	47.2 ± 10.1	45.8 ± 8.4	0.34
$\dot{V}O_{2peak}$ (% predicted)	130 ± 19	128 ± 18	0.53
Peak heart rate (b.p.m)	170 ± 16	168 ± 15	0.31
Peak workload (W)	364 ± 83	361 ± 70	0.77
RER	1.21 (1.16–1.29)	1.23 (1.18–1.31)	0.13
SBP seated on bike[a] (mmHg)	139 (130–150)	142 (134–152)	<0.001
DBP seated on bike[a] (mmHg)	85 ± 10	87 ± 10	0.11
SBP_{max} (mmHg)	219 ± 26	221 ± 23	0.54
DBP_{max} (mmHg)	83 (77–91)	84 (75–91)	0.84
SBP/W-slope (mmHg/W)	0.26 ± 0.09	0.23 ± 0.08	0.022
DBP/W-slope (mmHg/W)	0.000 (−0.030–0.030)	−0.010 (−0.020–0.020)	0.63
SBP_{max}/W-ratio (mmHg/W)	0.66 (0.57–0.79)	0.65 (0.56–0.81)	0.82

Values are median (IQR) or mean ± SD.
AF, atrial fibrillation; $\dot{V}O_{2peak}$, peak oxygen uptake; RER, respiratory exchange ratio; SBP, systolic blood pressure; SBP_{max}, maximal SBP; DBP_{max}, maximal diastolic blood pressure; SBP/W-slope, SBP to workload slope from individual linear regression; W, Watts; DBP/W-slope, DBP to workload slope from individual linear regression.
[a]*n* = 86 for No-AF and 73 for AF.

Exercise testing

There were no significant differences between groups in cardiorespiratory fitness, with a relative $\dot{V}O_{2peak}$ of 45.8 ± 8.4 mL/kg/min for athletes with AF compared with 47.2 ± 10.1 mL/kg/min for athletes without AF ($P = 0.34$). Both groups achieved similar peak workload ($P = 0.77$) and peak heart rate ($P = 0.31$) (*Table 3*).

Blood pressure responses

Figure 1 and *Table 3* show the BP responses during exercise testing. There were no significant differences in SBP_{max} or DBP_{max} between groups {SBP_{max}: 221 ± 23 mmHg vs. 219 ± 26 mmHg, $P = 0.54$; DBP_{max}: [84 (75–91) mmHg vs. 83 (77–91) mmHg, $P = 0.84$], for AF and No-AF groups, respectively}. The effect size was negligible [SBP_{max} Cohen's d = −0.09, 95% CI

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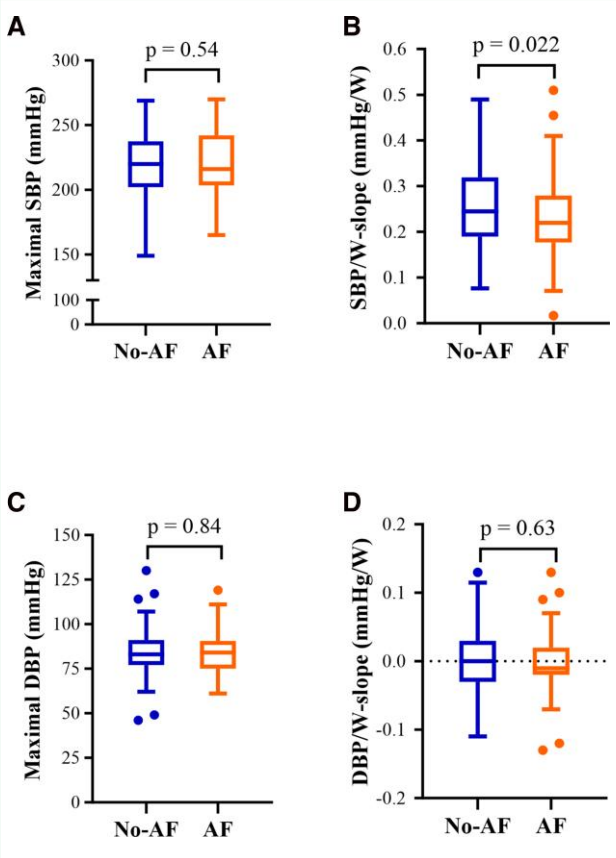

Figure 1 Comparison of exercise blood pressure metrics in athletes without atrial fibrillation (No-AF, $n = 89$) and with paroxysmal atrial fibrillation (AF, $n = 89$). (*A*) Maximal systolic blood pressure (SBP_{max}), (*B*) the SBP to workload-slope (SBP/W-slope), (*C*) the maximal diastolic blood pressure (DBP_{max}), and (*D*) the DBP to workload-slope (DBP/W-slope). Boxplots show the median line and interquartile range (IQR), with whiskers extending to 1.5× IQR. No statistically significant differences were observed between groups for SBP_{max}, DBP_{max} or the DBP/W-slope. The SBP/W-slope was *lower* in the AF group compared to the No-AF group.

(−0.39, 0.20) and DBP_{max} Cohen's d = −0.02, 95% CI (−0.32, 0.27)]. The SBP/W-slope was significantly higher in athletes without AF ($P = 0.022$) (*Figure 2*). There were no between-group differences in DBP/W-slope ($P = 0.63$, r = −0.49) or the SBP_{max}/W-ratio ($P = 0.82$, r = −0.02). No significant AF status × sport discipline interactions were observed for SBP_{max} ($P = 0.08$), SBP/W-slope ($P = 0.36$), DBP/W-slope ($P = 0.09$), and SBP_{max}/W-ratio ($P = 0.72$). However, there was a significant interaction for the DBP_{max} ($P = 0.030$).

The linear mixed-effects model showed that a substantial proportion of the SBP variance is attributable to inter-individual variability (ICC = 0.64) and a significant workload-by-group interaction ($P = 0.035$). The athletes without AF had a significantly, albeit modestly, higher SBP/W-slope ($\Delta\beta = 0.01$; 95% CI 0.001–0.027; $P = 0.035$) compared with the athletes with AF (SBP/W-slope = 0.23 mmHg/W vs. 0.22 mmHg/W) for athletes without and with AF, respectively. Cumulative SBP during exercise, quantified as the area under the SBP/W-curve, did not differ between athletes with and without AF (mean difference −107 mmHg·W, 95% CI −1577 to 1363; $P = 0.89$, with a negligible effect size (Cohen's d = −0.006, 95% CI −0.087 to 0.076), representing <0.2% of total SBP exposure.

A sensitivity analysis excluding athletes with resting office BP ≥140/90 mmHg or taking a beta-blocker or on antiarrhythmic medications provided similar results and effect sizes (see Supplementary material online, *Tables S2* and *S3*).

To assess the potential for an interaction between exercise BP and atrial remodelling, we compared athletes with and without severe atrial enlargement (LA_i >48 mL/m^2). When comparing the 67 athletes with a severely dilated LA_i to the 111 athletes without severe LA_i enlargement, no significant between-group differences were seen in resting BP, pre-exercise BP, or maximal BP. However, both the SBP/W-slope and SBP_{max}/W-ratio were significantly lower in athletes with a severely dilated LA_i compared with those without (SBP/W-slope: 0.23 ± 0.08 mmHg/W vs. 0.26 ± 0.09 mmHg/W, $P = 0.025$; SBP_{max}/W-ratio: 0.65 ± 0.14 vs. 0.70 ± 0.18, $P = 0.049$) (see Supplementary material online, *Table S4*). When comparing the exercise BP responses across the full spectrum of LA size, no significant differences were seen in the SBP/W-slope, DBP/W-slope, or SBP_{max}/W-ratio between the groups. Furthermore, no significant difference was seen in the resting SBP (both at rest and pre-exercise) nor in the DBP_{max}. In a *post hoc* Bonferroni test, there was a significant difference in SBP_{max} between the athletes with a normal LA_i volume and those with a moderately enlarged LA_i volume ($P = 0.017$). SBP pre-exercise was significantly higher in athletes with moderately and severely enlarged LA_i ($P = 0.002$ and $P < 0.001$, respectively) compared with normal and mildly enlarged LA_i (see Supplementary material online, *Table S5*).

Discussion

This relatively large case-control study is the first to evaluate BP responses to exercise in endurance athletes with and without AF. We found no differences in BP responses between athletes with and without AF and no differences in exercise BP between athletes with and without severe LA enlargement. Whilst the increased volume and pressure load of exercise are likely involved in the causation of both atrial remodelling and AF in athletes, our data suggests that the individual variation in BP responses are not strong predictors of AF prevalence. Thus, an exaggerated BP response to exercise is unlikely to be a major contributing factor to the development of AF in otherwise healthy endurance athletes.

Atrial remodelling and AF pathophysiology

In the general population, LA enlargement is a powerful biomarker indicating impaired ventricular and atrial compliance, high filling pressures, and impaired atrial function and is an established risk factor for AF.[27] Chronic hypertension can exacerbate each of these factors and is strongly associated with incident and prevalent AF in the general population. By contrast, LA enlargement is almost universal in endurance athletes who exhibit supranormal ventricular and atrial compliance, low filling pressures, and superior atrial and ventricular function. Whilst this enlargement in athletes is generally considered physiological, some athletes develop AF. One proposed mechanism is that the remarkably high systemic BP observed in athletes during

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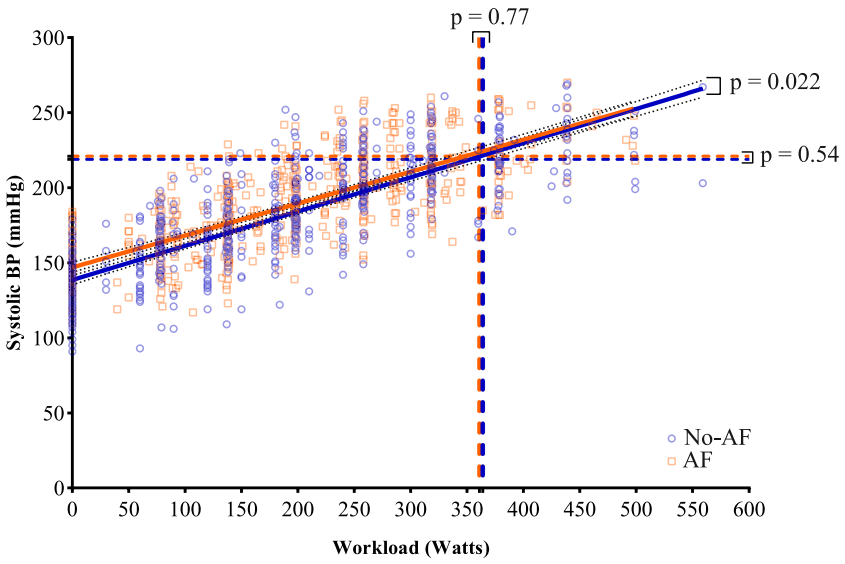

Figure 2 Scatterplot of multiple individual systolic bood pressure (SBP) measurements during graded bicycle testing with linear regression lines for SBP indexed to workload stratified by atrial fibrillation (AF) status (n = 178). No-AF (n = 89, circles) and AF (n = 89, squares). Horizontal dotted line indicating mean maximal SBP for No-AF and AF cohort (P = 0.54) and the vertical dotted line indicating mean maximal workload for No-AF and AF (P = 0.77). Linear regression lines (P = 0.022).

exercise leads to atrial stretch and dilatation, with the most exaggerated responses contributing to proarrhythmic remodelling and AF risk. As cardiovascular fitness is strongly driven by LV remodelling, the LA:LV volume ratio has emerged as a clinical index of proportionality.[28] The significantly higher LA:LV volume ratio observed in the AF group suggests disproportional atrial enlargement not captured by LA volumes alone.

However, our findings partially challenge the assumption that excessive rises in BP differentiate AF risk. Both AF and control athletes had enlarged atria, and marked increases in BP were observed in both groups. However, there was no difference in exercise BP responses in athletes with and without AF. This suggests that whilst the pressure and volume load of exercise may be associated with atrial remodelling and AF, exaggerated BP responses (sometimes termed exercise-related hypertension) do not differentiate between those athletes with and without AF. Similarly, SBP_{max} did not differ between the athletes with severely dilated atria and those without severe atrial enlargement. As we have demonstrated previously,[14] and again in the current analysis, BP increases relative to intensity during exercise. Thus, the combination of training time and intensity is likely to determine overall atrial load to a greater extent than any individual variability in BP response. Furthermore, the high BP during exercise might be offset by the BP during the many more hours that the participants spend not exercising. Our prior findings demonstrated that the 24-h ambulatory BP in those with a peak exercise >220 mmHg is most often normal and can often be quite low.[29]

The relationship between BP responses during exercise in athletes and non-athletes

Earlier investigations have shown that the BP responses to exercise in athletes is primarily determined by heightened cardiac output rather than vascular stiffness or impaired endothelial function.[14] Indeed, the youngest and fittest athletes generate the highest SBP_{max}.[14] Thus, it is entirely predictable that very high BP readings are observed during exercise in highly trained athletes.[30] Consistent with this expected physiological response of a high cardiac output during exercise, we observed high SBP_{max} values across our cohort and did not identify a heightened SBP response in athletes with AF, supporting the notion that high BP observed in athletes during exercise is merely an indicator of high flow necessary to meet the high oxygen demands of the working muscle.

Blood pressure responses in the general population, who achieve far lower cardiac outputs, may be more indicative of abnormal vascular responses and pathology. Emerging evidence from the general population suggests that an exaggerated BP response to exercise is associated with an increased risk of AF.[31] In a large retrospective study of 17 617 individuals with 7 years of follow-up, Aker *et al.*[31] found a modest association between AF incidence and a peak exercise SBP >170 mmHg (hazard ratio 1.21; 95% CI, 1.01–1.45) compared with a peak SBP ≤150 mmHg. A similar threshold of >170 mmHg as a risk marker would capture almost all athletes. One-third of the sample studied by Aker *et al.*[31] had a peak SBP >170 mmHg, compared with 174 out of 178 (98%) participants in our athlete sample, including 86 (97%) controls without AF. This highlights the need for athlete specific cut-offs or approaches that index BP to workload to identify abnormal BP responses to exercise in athletic populations, rather than extrapolating from general population data. Given the ubiquity of high SBP_{max}, we hypothesized that indexing BP to workload may better identify abnormal exercise BP responses associated with LA dilatation or AF, but similarly, the DBP/W-slope and the SBP_{max}/W-ratio were not different between athletes

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with and without AF, and paradoxically the SBP/W-slope was higher in athletes without AF.

Implications for understanding AF in athletes

A key finding of this study is that the more pronounced LA enlargement in athletes with AF was not associated with high exercise BP. One alternate explanation could be reverse causation, whereby AF itself may promote progressive atrial remodelling, particularly in older athletes or those with longer-standing arrhythmia, thus contributing to a self-perpetuating cycle of AF leading to structural change which begets more AF. However, athletes with exercise-related AF often present with a low AF burden,[32] probably reducing the likelihood of profound atrial remodelling secondary to AF. Alternatively, a genetic predisposition or neurohormonal factors may underlie a maladaptive atrial response to repetitive volume and pressure stimuli, resulting in disproportionate remodelling despite similar haemodynamic stress.[1,11,33]

It is important to acknowledge that BP responses observed during laboratory-based testing may not fully capture the haemodynamic demands of prolonged, high-intensity exercise performed under real-world outdoor conditions. In a review by Palatini,[34] intra-arterial recordings showed that cyclists achieved much higher BPs during outdoor cycling compared with laboratory testing. Conventional CPET may therefore underestimate the influence of the training environment on cardiovascular load and the observed lack of association between the BP responses, and AF could reflect constraints inherent to the measurement context. The BP response may also be influenced by the type of exercise. Buchan *et al.*[35] demonstrated that, after an initial BP increase with intensity, there was a reduction and flattening of BP responses during prolonged steady-state exercise. A more granular understanding of cumulative haemodynamic and training load is therefore needed to further understand why some endurance athletes develop AF. Wearable technologies to monitor BP during both daily activities and training sessions could help estimate the total pressure load to which the atria and vasculature are exposed. This may help identify distinct cumulative pressure and training load characteristics that are missed with the single snapshot of information on acute BP responses and self-reported training load assessments[36] derived using our methodology, although the current wearable devices lack sufficient accuracy for scientific use.[37]

Resting BP as a risk factor

Although endurance athletes generally have a lower prevalence of hypertension compared with non-athletic populations,[38,39] our findings and those from previous cohorts suggest that hypertension remains a relevant clinical consideration in this group.[40,41] In this present study, athletes with AF had slightly higher resting BPs, including pre-exercise BP measurements seated on the bike. These modest elevations in resting BP may be clinically relevant, as even pre-hypertensive BP levels (SBP 130–139 mmHg) have been associated with an increased risk of AF[42,43] and may similarly play a role in endurance athletes. This also highlights that the concept of total pressure load is likely to be more important than the extent to which BP rises during the minority of time when an athlete exercises strenuously. It is worth remembering that 1 h of exercise represents only 4% of exposure time in any given day. These findings underscore the importance of diagnosing and managing elevated BP even in highly trained athletes and are consistent with current AF guidelines,[44] which emphasize the importance of identification and treatment of hypertension as a modifiable risk factor. Close monitoring of mildly elevated resting BP in athletes is warranted, as it may contribute to AF risk. Athletes with elevated resting BP may therefore benefit from 24-h ambulatory BP monitoring or repeated home-based BP monitoring to assess cumulative haemodynamic load and for the diagnosis of masked hypertension.[22]

Limitations

The cross-sectional case-control design allowed us to determine the association between paroxysmal AF and exercise BP responses but limits our ability to determine any causal inferences as to whether exaggerated BP responses precede AF onset. Another limitation is the demographic composition of the cohort, which was predominantly White and male. Although females were included, their representation was limited (8%), restricting our ability to perform sex-specific analyses. Given that female participation in endurance sports is increasing and that athletic females might have an increased risk of AF compared with their non-athletic peers,[45] future studies with greater female representation are warranted. Training history was assessed using a self-reported questionnaire, which may not accurately capture true training volume or intensity; therefore, training-related variables should be interpreted with caution and considered descriptive. Despite these limitations, the study has several notable strengths including comprehensive clinical phenotyping with detailed cardiac imaging, CPET, and multiple exercise BP measurements on each participant (mean 7 ± 2 measurements) using a validated BP device. The auscultatory automated BP device has been assessed against invasive intra-arterial BP measurements during both supine and treadmill exercise (mean difference vs. intra-arterial measures 3.7 and 4.8 mmHg, respectively).[24] Furthermore, the cohort includes a large sample of endurance athletes with paroxysmal AF, who were well matched to controls for key confounders.

Conclusions

Our findings demonstrate that exercise BP characteristics do not distinguish endurance athletes with AF from those without AF. These findings suggests that an exaggerated BP response to exercise does not explain the increased risk of AF observed in endurance athletes and that exercise BP assessment has limited value in identifying endurance athletes at increased risk for AF.

Supplementary material

Supplementary material is available at *European Journal of Preventive Cardiology*.

Author contributions

Kristel Janssens (Conceptualization, Resources, Software [supporting], Data curation, Formal analysis, Writing—original draft, Writing

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—review & editing [lead], Investigation, Methodology, Project administration [equal]), Turid Apelland (Investigation, Project administration, Writing—review & editing [supporting]), Amy M Mitchell (Investigation, Project administration, Writing—review & editing [supporting]), Jarne De Paepe (Investigation, Writing—review & editing [supporting]), Stephen J Foulkes (Supervision [equal], Writing—original draft [supporting]), Jon Magne Letnes (Writing—review & editing [supporting]), Andreas Berg Sellevold (Writing—review & editing [supporting]), Steve Enger (Writing—review & editing [supporting]), Youri Bekhuis (Writing—review & editing [supporting]), Christophe Dausin (Investigation, Project administration, Writing—review & editing [supporting]), Luke Spencer (Writing—review & editing [supporting]), Stephanie J Rowe (Writing—review & editing [supporting]), Paolo D'ambrosio (Writing—review & editing [supporting]), Evelyn B Parr (Supervision [equal], Writing—review & editing [supporting]), Rik Willems (Methodology, Writing—review & editing [supporting], Supervision [equal]), Hein Heidbuchel (Writing—review & editing [supporting]), Guido Claessen (Methodology, Supervision, Writing—review & editing [supporting]), Marius Myrstad (Conceptualization [lead], Methodology, Writing—original draft [supporting], Supervision [equal]), and Andre La Gerche (Conceptualization [lead], Methodology, Writing—original draft [supporting], Supervision [equal])

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Conflict of interest: M.M. has received lecture fees from AstraZeneca, Bayer, Boehringer-Ingelheim, Bristol Myers Squibb, MSD, and Pfizer unrelated to this study.

Data availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Appendix

The Master@Heart consortium

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