BMJ Open Sport & Exercise Medicine

Atrial remodelling associated with sporting discipline, sex and duration in elite sports: a cross-sectional echocardiographic study among Danish elite athletes

Mikkel Aaroee ,[1,2] Susanne Glasius Tischer,[3] Robin Christensen,[2,4] Christian Have Dall ,[1,5,6] Jens Jakob Thune,[1,5] Hanne Rasmusen [1,5]

To cite: Aaroee M, Tischer SG, Christensen R, *et al*. Atrial remodelling associated with sporting discipline, sex and duration in elite sports: a cross-sectional echocardiographic study among Danish elite athletes. *BMJ Open Sport & Exercise Medicine* 2024;**10**:e001880. doi:10.1136/bmjsem-2023-001880

Accepted 15 April 2024

For numbered affiliations see end of article.

Correspondence to
Dr Mikkel Aaroee;
mikkel.aaroee@regionh.dk

ABSTRACT

Background Elite endurance training is characterised by a high-volume load of the heart and has been associated with atrial fibrillation (AF) in middle-aged men. We compared left atrial (LA) remodelling among elite athletes engaged in sports, categorised as having low, intermediate, and high cardiac demands.

Methods This cross-sectional echocardiographic study of healthy elite athletes evaluated LA size and function measured as LA maximum volume (maxLAVi) and contraction strain. Athletes were grouped according to the cardiac demands of their sport (low, intermediate, high). Morphological measures were indexed to body surface area and reported as least square means; differences between groups were reported with 95% CIs.

Results We included 482 elite athletes (age 21±5 years (mean±SD), 39% women). MaxLAVi was larger in the high group (28.4 mL/m^2) compared with the low group (20.2 mL/m^2; difference: 8.2, CI 5.3 to 11.1 mL/m^2; p<0.001), where measurements in men exceed those in women (26.4 mL/m^2 vs 24.7 mL/m^2; difference 1.6 mL/m^2; CI 0.3 to 2.9 mL/m^2; p=0.0175). In the high group, LA contraction strain was lower compared with the low group (−10.1% vs −12.9%; difference: 2.8%; CI 1.3 to 4.3%; p<0.001), and men had less LA contraction strain compared with women (−10.3% vs −11.0%; difference 0.7%; CI 0.0 to 1.4%; p=0.049). Years in training did not affect maxLAVi or LA contraction strain.

Conclusion MaxLAVi was higher while LA contraction strain was lower with increased cardiac demands. MaxLAVi was larger, and LA contraction was lower in men compared with women. Whether these sex-based differences in LA remodelling are a precursor to pathological remodelling in male athletes is unknown.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Endurance sports increase the risk of atrial fibrillation in veteran male athletes, with very limited knowledge of sex-specific differences in the long-term consequences of elite endurance training.

WHAT THIS STUDY ADDS

⇒ Individual measures of left atrial function are frequently found outside reference limits even for athletes of comparable sports and experience. Our results suggest athletic-induced reductions in left atrial contraction may be specific to men.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Clinical interpretation of individual measures of atrial function is insufficient to determine possible pathology. A longitudinal study of left atrial function is needed to determine if reductions in left atrial contraction strain are specific to male athletes and may be a precursor to later pathological remodelling and atrial fibrillation.

INTRODUCTION

Cardiac size and function are primarily determined by body size (body surface area, BSA) but are also influenced by factors such as age, ethnicity, sex and fitness level.[1–4] Elite endurance training is characterised by repeated high-volume/high-intensity training causing benign physiological adaptations collectively referred to as the 'athlete's heart'.[5] Training-induced left atrial (LA) dilation is a benign feature of the 'athlete's heart' and is frequently observed among young healthy athletes.[6 7] In the background population, enlarged LA is linked to an elevated risk of atrial fibrillation (AF). However, depending on the mechanism of LA dilation, the consequences of an enlarged LA may be vastly different. Benign LA remodelling, as observed in most athletes, is characterised by a reversible increase in volume and compliance, contributing to an overall improvement in function.[8] Even in severe cases of physiological remodelling, as seen in an uncomplicated pregnancy, in which LA size may increase up to 40%, complete remission to prepregnancy size is seen within

3 months postpartum with no long-term increase in the risk of cardiac arrhythmias.[9]

AF reduces exercise capacity and increases the risk of thromboembolic complications and is, therefore, important to prevent.[8] More recent markers of LA function by strain could be a tool for early recognition of conversion from physiological to pathological remodelling of LA. Pathological LA enlargement is characterised by myocardial inflammation, reduced LA function and potential long-term formation of LA fibrosis and cardiac rhythm disorders.[8] Emerging evidence suggests that excessive exercise may contribute to pathological LA remodelling.[5 10] Vigorous exercise has been linked to a higher incidence of AF in veteran male, but not female, endurance athletes,[11] though some studies have found a reduced risk of AF in non-elite moderate physically active men.[12] Additionally, some large population studies have suggested moderate or even vigorous physical activity may reduce the risk of AF in physically active females.[13 14] However, this sex-specific difference in elite endurance athletes has been questioned due to very few studies on female athletes.[15] Female athletes have been noted to exhibit a higher capacity for athletic remodelling compared with their male counterparts.[16] However, the occurrence of pathological remodelling and elevated risk of AF appear to be primarily a male phenomenon with no established explanation for this perceived sex-based difference.[17] This raises the question of whether women possess a greater capacity for athletic remodelling and do so without experiencing pathological adaptation in cardiac structure.

To our knowledge, we present the largest single sample echocardiographic investigation of Scandinavian elite athletes to determine the extent of LA remodelling in men and women across the full spectrum of cardiac demands (CD) in sport. The objective was to compare the LA remodelling of elite athletes in low cardiac demand sporting disciplines relative to athletes in intermediate and high cardiac demand sports regarding LA volume and function. Secondary objectives included investigating the impact of sex and years in elite training (training age) on LA remodelling.

Materials and methods

Study design and methods

In this cross-sectional study, we analysed echocardiographic data from 'the Danish athletes' heart cohort' (Data published in 2016).[18] We invited active athletes competing in the national Danish elite sports organisation (Team Danmark) and professional football clubs aged 12–35 years to participate. Athletes with prior known cardiac conditions were excluded. All participants were instructed to avoid training on the day of inclusion. Participants received a clinical examination from the designated physician, who also performed an interview on medical and training history, after which a transthoracic echocardiographic examination (TTE) was performed according to standard guidelines[19] (GE vivid E9 and GE Vivid S6, Vingmed, Horten, Norway).

Expanded analysis of TTE raw data was performed according to current guidelines[20] (analysing software: GE Viewpoint cardiology V.6.12, EchoPAC suite V.204). All cardiac size measures were indexed for BSA. LA maximum and minimum volume (maxLAVi; minLAVi) and left ventricle end-diastolic volume (LVEDVi) were measured manually as biplane average. The upper limit of normal maxLAVi was defined as 34 mL/m^2 following the current guidelines.[20] The upper reference limit for minLAVi was defined as 12 mL/m^2 according to a study of 276 healthy untrained volunteers by Badano *et al*.[21] The atrial strain was measured as biplane average in a standard two and four-chamber view for each phase of the atrial cycle.[22] Measures were deemed unreliable, if no clear strain-curve could be achieved, regardless of image quality. Strain measures were only performed on images of >60 frames per second, in accordance with manufacturer guidelines. Reduced LA reservoir strain was defined as <34.6, and reduced LA contraction strain was defined as <11.6, according to a meta-analysis of 408 athletes by Cuspidi *et al*.[23] Athletes were grouped according to the Mitchell classification of sports into CD groups of low (Mitchell class 1A, for example, rifling, curling), intermediate (Mitchell class 2–3A, 1-2-3B, eg, football, handball) and high cardiac demands (Mitchell class 1-2-3C, eg, triathlon, rowing).

The study was approved by the regional ethics committee (H-21043707), and data collection was approved by the Danish Data Protection Agency (P-2021–722). Before inclusion, all participants signed an informed consent form after receiving a verbal and written study outline. All data were stored in an encrypted online-based database (REDCap) according to Danish Data Protection Agency guidelines.

STATISTICAL METHODS

Using analysis of covariance models, we analysed the difference between the CD groups based on crude (unadjusted) models and adjusted for sex, the duration of elite-level training and age. Linear regression and Pearson correlations were used to determine associations between selected variables. Missing data were <10% in all four primary variables in any group. All p values and 95% CIs are two sided. We did not apply explicit adjustments for multiplicity; rather, we analysed, reported and interpreted the secondary objectives in a prioritised order.

Results

In total, 1347 athletes were invited, and n=516 were included in the initial cohort (figure 1). TTE data from 34 participants were lost. Therefore, the population available for the present cross-sectional sample was n=482 (39% women), representing 30 different sporting disciplines.

Demographics stratified by the cardiac demand of sporting discipline groups

The low CD-group consisted of 31 athletes (54% women) from skill-based sports, with 343 (38% women) in the

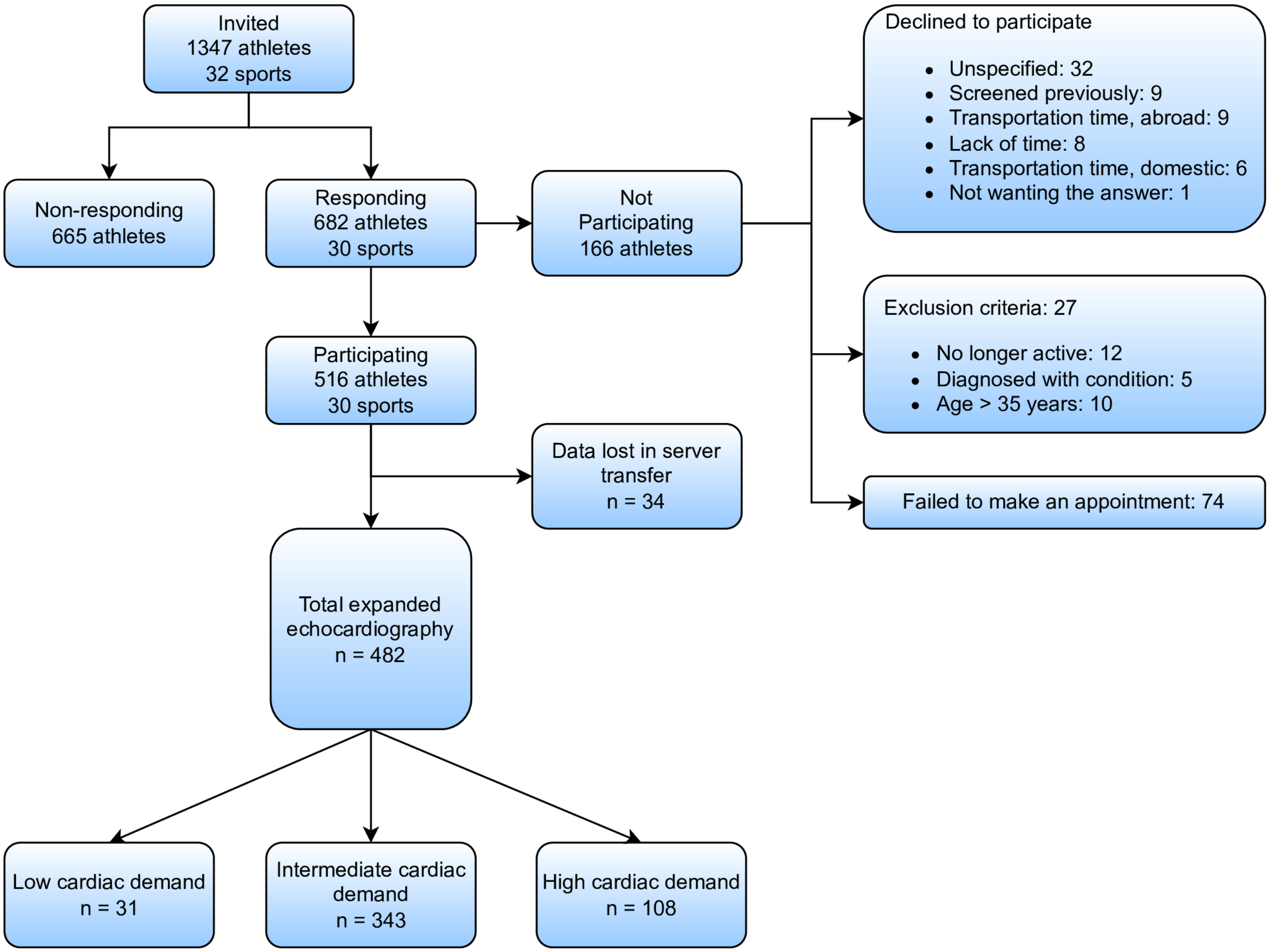

Figure 1 Flow diagram of the recruitment process. Low cardiac demand: defined as Mitchell class 1a. Intermediate cardiac demand: defined as Mitchell class 2-3 a, 1-2-3B. High cardiac demand: defined as Mitchell class 1-2-3c.

intermediate CD-group and 108 (37% women) in the high CD-group. All athletes were highly active, with ~7 years of experience at the elite level. The intermediate CD-group was younger than the other groups, while the high CD-group had higher training age and weekly training hours (table 1).

Statistical associations between sporting discipline and echocardiographic findings

The atrial filling phase is described by maxLAVi and LA reservoir strain in size and function, while minLAVi and LA contraction strain describes the atrial contraction phase.

LA filling phase

Athletes competing in sports characterised by high or intermediate CD demonstrated significantly larger maxLAVi compared with athletes from the low CD-group (table 2, and figure 2A). We found no interaction between sex and CD-group on maxLAVi (p for interaction=0.930). Adjusted for CD-group and age, maxLAVi was higher for men than for women (men 26.4 mL/m^2 vs women 24.7 mL/m^2; difference 1.6 mL/m^2; (CI 0.3 to 2.9 mL/m^2; p=0.018).

LA function in the filling phase, measured as LA reservoir strain, was reduced in the high and intermediate CD-groups compared with the low CD-group (table 2, and figure 2B), with no interaction between sex and CD-group on LA reservoir strain (p=0.963). However, LA reservoir strain adjusted for CD-group and age was lower in men (40.0% vs 43.4%) compared with women (difference: 3.6% (CI 1.9% to 5.2%); p<0.001).

LA contraction phase

MinLAVi was larger in the high CD-group than in the intermediate or low CD-groups. However, we found no significant difference between intermediate and low CD-groups (table 2 and figure 2C). The effect of CD-group on minLAVi was not affected by sex (p for interaction=0.900), though minLAVi was larger in male athletes (men 11.1 mL/m^2 vs women 9.4 mL/m^2) compared with their female counterparts (difference: 1.6 mL/m^2 (CI 0.8 mL/m^2 to 2.5 mL/m^2); p<0.001).

Table 1 Descriptive statistics of all variables stratified by cardiac demands groups

	Low cardiac demand n=31	Intermediate cardiac demand n=343	High cardiac demand n=108
Demographics			
Age, years	24.4±0.7	20.4±0.3	23.8±0.4
Training age, years	6.9±0.7	6.7±0.2	7.8±0.4
Weekly training, hours	17.1±1.2	16.3±0.4	19.9±0.7
BSA, m^2	1.9±0.03	1.9±0.01	1.9±0.02
Left atrium			
maxLAVi, mL/m^2	20.7±0.8	25.1±0.4	29.1±0.8
LA reservoir strain strain, %	46.3±1.6	40.9±0.5	40.4±0.9
minLAVi, mL/m^2	8.4±0.8	9.9±0.2	12.4±0.5
LA contraction strain, %	−13.2±0.7	−10.5±0.2	−10.2±0.04
Right and left ventricle			
RVEDAi, cm^2/m^2	12.5±0.4	13.9±0.1	14.2±0.2
RV FW strain, %	−24.8±1.1	−23.2±0.3	−23.3±0.6
LVEDVi, mL/m^2	62.0±2.3	75.0±0.07	79.9±1.2
LV GLS, %	−18.9±0.4	−18.1±0.1	−18.4±0.2
RWT	0.30±0.01	0.33±0.00	0.36±0.00
LV mass, g/m^2	71.6±3.4	89.4±1.0	101.8±1.8
E, cm/s	89.2±13.8	87.1±14.3	84.0±15.3
A, cm/s	47.3±8.9	42.0±8.9	40.7±3.4
e', cm/s	20.3±3.2	19.7±3.4	19.0±2.7
E/A, ratio	1.94±0.44	2.15±0.50	2.15±0.56
E/e', ratio	4.43±0.86	4.54±0.93	4.51±0.89

All measures are reported as mean ± standard error. Training age: years of training in an elite training program. Training time per week: hours of training per week.
BSA, body surface area; LVEDVi, Left ventricular end-diastolic volume indexed to BSA; LV GLS, left ventricular global longitudinal strain; LV mass, left ventricular mass; MaxLAVi, LA systolic volume indexed to BSA; MinLAVi, LA diastolic volume indexed to BSA; RVEDAi, RV end-diastolic area indexed to BSA; RV FW strain, RV free wall strain; RWT, relative wall thickness.

LA contractile function, as measured by LA contraction strain, was lower in athletes from high and intermediate CD-groups than athletes from the low CD-group. At the same time, there was no difference between high and intermediate CD-groups (table 2 and figure 3D). LA contraction strain adjusted for CD-group and age was lower in men (men: 10.3% vs women: 11.0) compared with women (difference 0.7 (CI 0.0% to 1.4%); p=0.049). Interaction between sex and CD-group and LA contraction strain was borderline significant (p=0.077). Furthermore, LA contraction strain was reduced with higher CD-group in men (high CD-group −9% (CI −10% to −9%); intermediate CD-group −10% (CI −11 to −10%); low CD-group 14% (CI −16% to −12%); p for difference between CD-groups <0.001) but not in women (high CD-group −11% (CI −13% to −10%); intermediate CD-group −11% (CI −12% to −10%); low CD-group −12% (CI −14% to −11%); p for difference between CD-groups=0.314).

Association between LA size and strain

Linear regression analysis showed that LA size and function were lightly interdependent, with decreasing LA reservoir strain in athletes associated to increasing LAVimax (r^2=0.009; p=0.058), while LA contraction strain significantly increased with lower LAVimin (r^2=0.041; p<0.001). Moreover, a statistically significant correlation was observed around the LA strain measures, indicating that increased LA reservoir strain was associated with higher levels of LA contraction strain (r^2=0.397; p<0.001).

Left ventricular diastolic function

Left ventricular filling pressure (as measured by E/e') showed no variation between the CD-groups (difference high vs low: 0.141 ratio CI −0.237 to 0.519 ratio; p=0.476; difference intermediate vs low: 0.219 ratio CI −0.138 to 0.576 ratio; p=0.232).

Table 2 Echocardiographic findings adjusted for sex and age

	Low cardiac demand n=31	Intermediate cardiac demand n=343	High cardiac demand n=108	Difference High vs low (95% CI)	Difference Intermediate vs low (95% CI)
Left atrium					
maxLAVi, mL/m^2	20.2±1.4	25.2±0.4	28.4±0.7	8.2 (5.3 to 11.1)	4.9 (2.2 to 7.7)
LA reservoir strain, strain, %	46.1±1.6	41.4±0.5	40.8±0.9	−5.2 (−8.8 to −1.5))	−4.8 (−8.1 to −1.5)
minLAVi, mL/m^2	8.2±0.9	10.0±0.3	12.0±0.5	3.7 (1.8 to 5.6)	1.6 (−0.1 to 3.4)
LA contraction strain, %	−12.9±0.7	−10.6±0.2	−10.0±0.4	2.8 (1.3 to 4.3)	2.3 (0.9 to 3.7)
Right and left ventricle					
RVEDAi, cm^2/m^2	12.7±0.4	13.4±0.1	14.2±0.2	1.6 (0.7 to 2.5)	1.1 (0.3 to 1.9)
RV FW strain, %	−24.3±1.1	−23.6±0.4	−22.8±0.6	1.5 (−1.0 to 4.0)	0.7 (−1.6 to 3.1)
LVEDVi, mL/m^2	62.5±2.1	73.5±0.6	78.5±1.1	16.0 (11.5 to 20.5)	11.0 (6.8 to 15.2)
LV GLS, %	−18.8±0.4	−18.5±0.1	−18.4±0.2	0.4 (−0.5 to 1.3)	0.3 (−0.5 to 1.1)
RWT	0.3±0.01	0.3±0.00	0.4±0.00	0.06 (0.04 to 0.07)	0.03 (0.01 to 0.05)
LV mass, g/m^2	70.9±3.0	88.8±0.9	98.4±1.6	27.4 (20.8 to 34.0)	16.9 (10.7 to 23.1)
E, cm/s	91.2±2.5	87.5±0.8	86.8±1.4	−4.4 (−10.0 to 1.2)	−3.7 (−8.9 to 1.6)
A, cm/s	47.3±1.7	43.2±0.5	41.1±0.1	−6.3 (−10.0 to −2.5)	−5.0 (−8.6 to −1.5)
e’, cm/s	20.9±0.6	19.5±0.2	19.5±0.3	−1.4 (−2.6 to −0.1)	−1.4 (−2.5 to −0.2)
E/A, ratio	1.99±0.09	2.14±0.03	2.20±0.05	0.21 (0.01 to 0.42)	0.06 (-0.06 to 0.42)
E/e’, ratio	4.37±0.17	4.59±0.05	4.51±0.09	0.14 (-0.24 to 0.52)	−0.22 (-0.14 to 0.58)

All echocardiographic values are presented as mean ± standard error and difference (95% CI).
BSA, body surface area; LVEDVi, left ventricular end-diastolic volume indexed to BSA; LV GLS, left ventricular global longitudinal strain; LV mass, Left ventricular mass; MaxLAVi, LA systolic volume indexed to BSA; MinLAVi, LA diastolic volume indexed to BSA; RVEDAi, RV end-diastolic area indexed to BSA; RV FW strain, RV free wall strain; RWT, relative wall thickness.

Prevalence of abnormal LA size or function

We observed 12.8% of all athletes demonstrating enlarged maxLAVi, and this was only present in athletes from the high (19%) and intermediate CD-group (10%; figure 3A). Sex did not affect the prevalence of enlarged maxLAVi in any group (high CD-group men 21% vs women 16%; p=0.587; intermediate CD-group men 10% vs women 9%; p=0.818). LA reservoir strain was reduced to the below reference limits in 32% of athletes (figure 3B). Low LA reservoir strain was more prevalent in men (men 31% vs women 16%; p<0.001).

The prevalence of minLAVi above reference limits was 33% in all athletes (figure 3B), with a higher prevalence in more demanding sports (high CD-group 51%; intermediate CD-group 28%; low CD-group 17%; p<0.001). More men than women had minLAVi increased beyond reference limits (men 33% vs women 21%; p=0.003), while LA contraction strain was reduced under normal reference limits in 60% of athletes (figure 3D). There were no differences in reduced LA contraction strain between the sexes (men 62% vs women 55%; p=0.162).

EVALUATION OF TRAINING AGE ON ATRIAL REMODELLING

Training age did not affect LA size and function (maxLAVi 0.14 mL/m^2 per training year SE: 0.13; p=0.275; minLAVi 0.05 mL/m^2 per training year SE: 0.08; p=0.515; LA reservoir strain −0.24% per training year SE: 0.16; p=0.121; LA contraction strain 0.07% per training year SE: 0.07; p=0.266), or ventricular size and function (RVEDAi 0.01 mL/training year SE: 0.04; p=0.717; LVEDVi 0.27 mL/training year SE: 0.22; p=0.214; left ventricular global longitudinal strain: 0.6% SE: 0.04; p=0.155; right ventricular free wall strain: 0.15 SE: 0.10; p=0.147).

Discussion

In this large cross-sectional study of elite athletes, we present results from the full spectrum of sports in an ethnic, homogenous young Scandinavian population of men and women. The main findings were: (1) LA size increased with a higher CD-group, while LA reservoir and contraction strain decreased; (2) male sex is associated with increased LA size and LA reservoir and contraction strain compared with women; (3) LA size and function are not affected by training age in the short term; (4) LA size and function were highly heterogenous within CD-groups.

The importance of CD on LA filling and contraction

LA filling measured by maxLAVi increased with a higher CD-group, and we observed a moderate prevalence of enlarged LA beyond reference limits in line with the previous studies on athletic remodelling.[24] Increased LV filling pressure may be a cause for LA dilation,[25] however we found no differences between CD-groups. MinLAVi was used to measure LA emptying and followed the same pattern as maxLAVi, with an increase in size associated with a higher CD-group. These results corroborate D’Ascenzi *et al*, who found structured exercise-increased

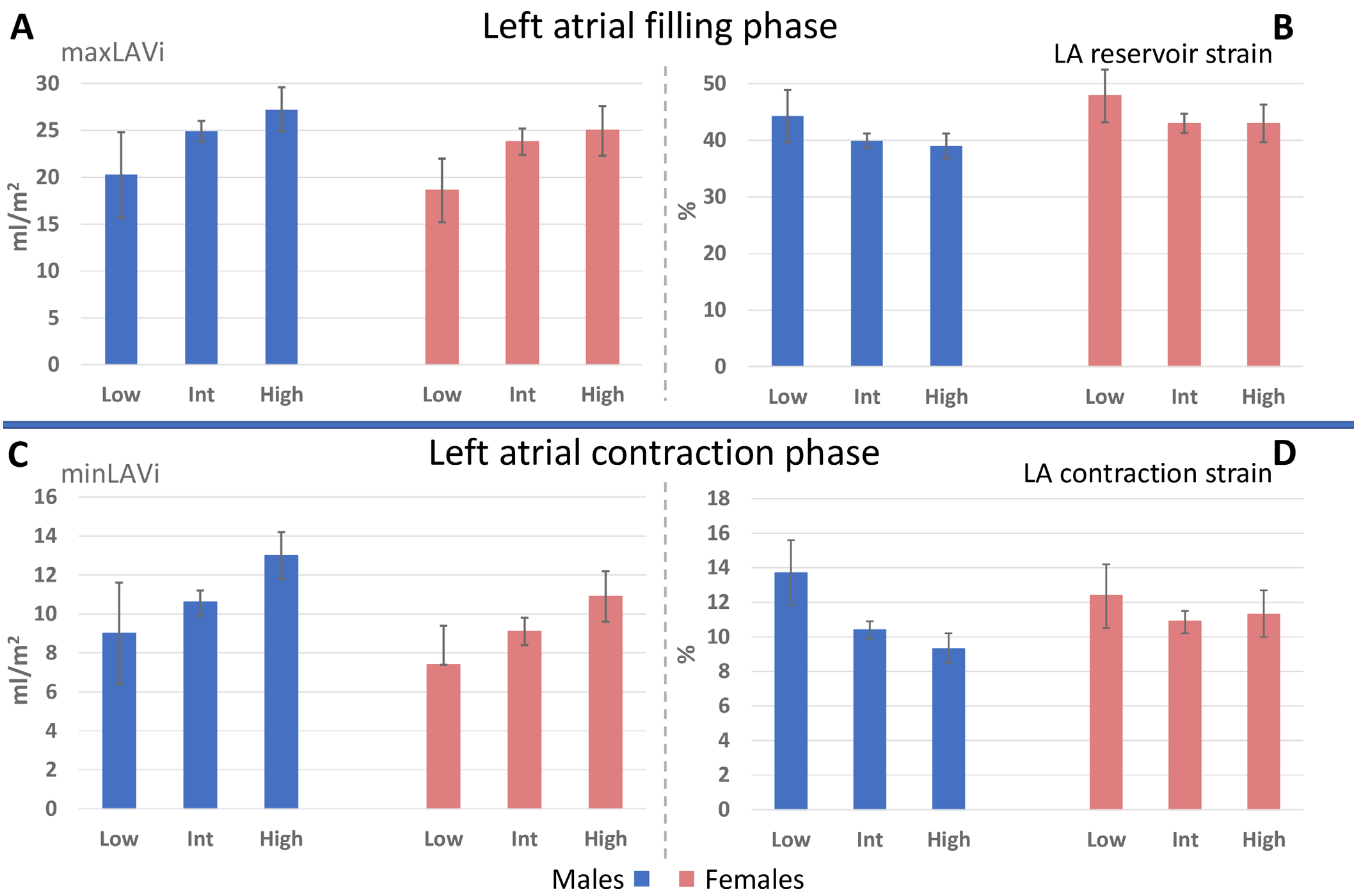

Figure 2 Age-adjusted least squares means of LA filling phase (maxLAVi and LA reservoir strain) and LA contraction phase (minLAVi, and LA contraction strain) stratified by cardiac demands group and sex. Error bars signify 95% CIs. Left atrial maximum volume indexed to body surface area (maxLAVi, A), left atrial reservoir strain (B), left atrial minimum volume indexed to body surface area (minLAVi, C) and left atrial contraction strain (D). Groups: low, intermediate and high cardiac demands group. LA, left atrial; maxLAVi, LA maximum volume.

minLAVi and maxLAVi throughout a season in 26 professional football players,[26] while the same authors in a different study of 150 elite athletes (comparable to our Intermediate CD-group) found minLAVi was 14% larger in athletes compared with controls,[24] similarly we found 11% increase in minLAVi between our intermediate and low CD-groups.

Increased LV volume is associated to reduced LV global longitudinal strain, though this association may be more complex in the LA. In our cohort of athletes from multiple sports, we found high maxLAVI was tentatively correlated to reduced LA reservoir strain, an association also found by Cheema *et al*,[27] though other studies have not found this association.[6 24] This disparity remains unexplained, though differences could be related to sex, ethnicity sporting disciplines or season heterogenicity.

Reduced LA strain is not uncommon in athletes, and LA contraction strain, in particular, is often found below the reference limits for the background population.[23] Our results align with previous studies on LA filling and contraction function in athletes[24] and suggest that well-trained athletes with high LA filling volumes effectively meet the demands of resting cardiac output, requiring less deformation at rest with a large contractile reserve.

The importance of sex on LA filling and contraction

There is currently no conclusive evidence associating extreme exercise and cardiac arrhythmia in female athletes. However, this perceived difference in male and female remodelling may be due to insufficient data on female athletic remodelling.[15 28] Men exhibit larger absolute chamber dimensions than women, although these differences are mostly offset by indexing for BSA.[16] In the present large cohort of ethnically homogenous men and women, maxLAVi and minLAVi were larger in men than women, but both sexes increased in size with increasing CD groups. Additionally, we saw no interaction between sex and CD-group, suggesting that atrial dilation in response to exercise is similar between men and women. LA contraction strain exhibited borderline interaction with sex and CD-group, indicating a possible sex-specific difference in LA contractile function. This was corroborated by reduced LA contraction strain in higher CD-groups in men, a pattern absent in women. Similarly, a recent study by Simard *et al* found LA strains responded differently to exercise in male and female athletes.[29] A prospective study of the differences in LA strain between male and female athletes is warranted to provide further insight into whether reduced LA function and, explicitly,

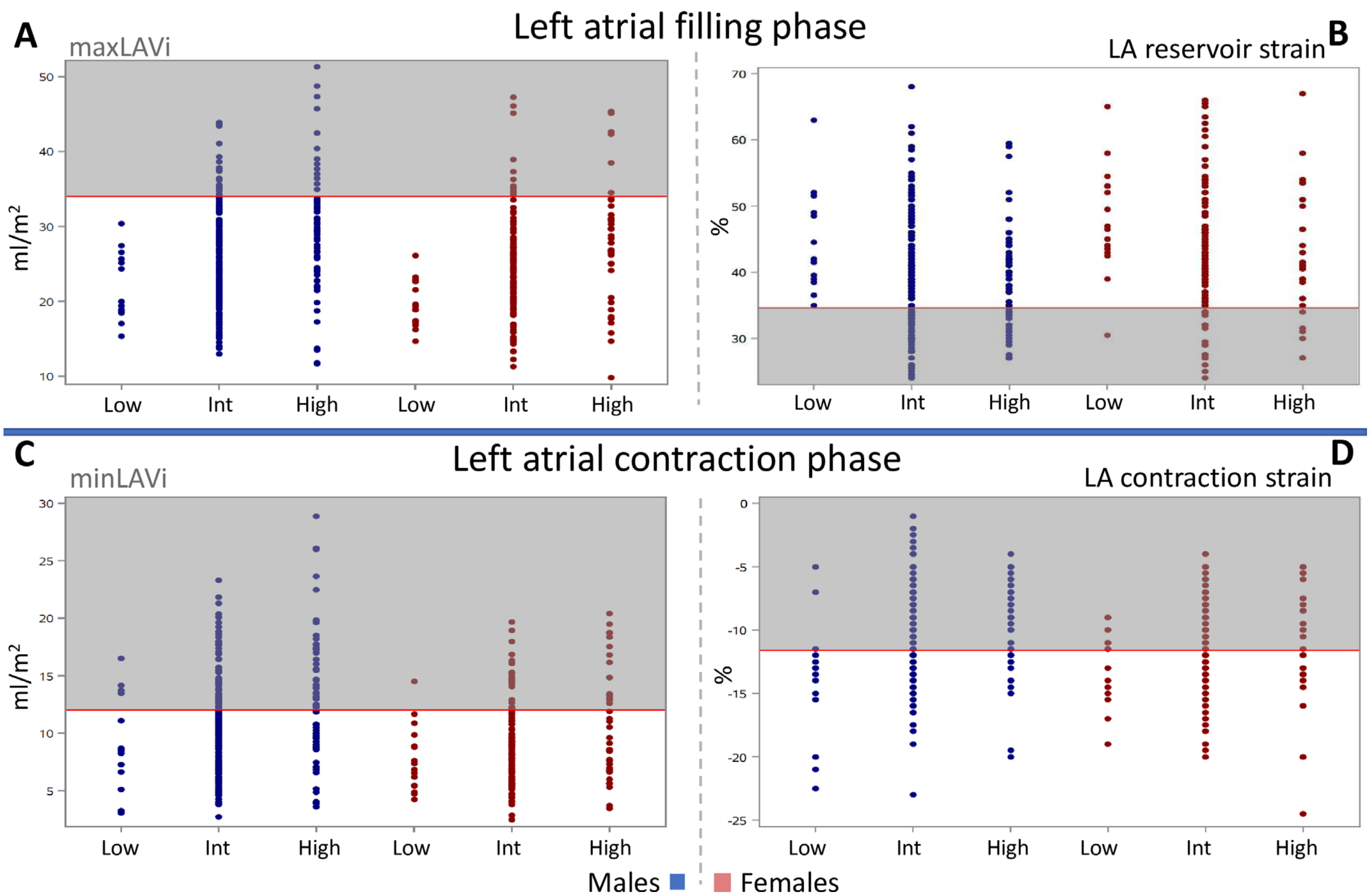

Figure 3 Age-adjusted individual values of left atrial size and function in the LA filling phase (maxLAVi and LA reservoir strain) and LA contraction phase (minLAVi, and LA contraction strain) stratified by cardiac demands group and sex. Grey area denotes values outside reference range. Left atrial maximum volume indexed to body surface area (maxLAVi, A), Left atrial reservoir strain (B), left atrial minimum volume indexed to body surface area (minLAVi, C) and left atrial contraction strain (D). Groups: low, intermediate and high cardiac demands group. LA, left atrial; maxLAVi, LA maximum volume.

LA contraction strain seen in men are prognostic for future LA structure and function pathology.

The importance of training age on LA filling and contraction

Age and training age variability were low in our cohort, reducing sensitivity to time-dependent variables. We found no time-dependent change in peak maxLAVi or minLAVi. In a prospective study of Olympic athletes, Pellicia *et al* found a small increase in maxLAVi of 1–2 mL over a mean follow-up time of 9 years,[30] which may be within the margin of error for this cross-sectional study. Training age did not affect either the LA reservoir strain or the LA contraction strain. These results align with those of Brugger *et al*, who found no effect of lifetime training hours on LA strains in 95 athletes despite marked increases in LA volumes with higher lifetime training.[31]

Prevalence of abnormal values of LA size and function

In elite athletes, distinguishing benign cardiac adaptations from pathological LA remodelling with increased risk of later-onset AF remains challenging. Prior studies have presented wide ranges of LA size measures.[23] In our cohort, each CD-group's individual LA size and function measures were highly heterogeneous, as presented in figure 3. A cause of this variation may be that LA size and, thereby, the function are highly dynamic in the relatively short term and dependent on loading conditions.[26 32] Additionally, some degree of variability persists within each CD-group as multiple sports are categorised in the same group, despite differences in duration and intensity of the required effort (eg, rowing vs triatlon).

A decline in LA function has been proposed as a potential precursor for later AF, given that atrial fibrosis strongly correlates with AF and is theorised to diminish atrial compliance and contraction.[8 33] However, resting measures of LA strains are expectedly lower in athletes, and we find that >30% of athletes had reduced LA reservoir strain and >60% reduced LA contraction strain beyond reference limits. We have no clinical indication of LA pathology in our cohort of young elite athletes, which indicates that even athlete-specific reference limits cannot be used as a marker of pathology in isolation. Our results further highlight that the interpretation of resting measures in athletes in the clinical setting must include repeated measures over time or stress testing, as athletes commonly exceed reference limits.

In conclusion, we found that in young elite athletes, LA size increased with increasing CD-group. LA strains decreased with higher CD-group in men but not significantly in women. Whether male and female athletes respond differently to the extreme CD of elite endurance training is uncertain. Our results suggest a possible sex-based distinction in the physiological remodelling of the LA, though not to a level of statistical significance. Interpreting abnormal resting LA strains in athletes, aimed at distinguishing between physiology and pathology, remains challenging.

Clinical implications

We supply much-needed knowledge on sex-based differences in exercise-induced LA remodelling from the largest single sample study of Scandinavian elite athletes from multiple sports. The long-term impact of exercise-induced enlarged atria in women is uncertain. Furthermore, we documented reduced LA contraction strain in male athletes with high cardiac demand compared with women, suggesting that female athletes are less prone to long-term exercise-induced atrial dysfunction. However, longitudinal investigations of LA contraction strain are needed to elucidate the differences in male and female risk of pathological remodelling of the LA and, thereby, the risk of future AF.

Limitations

Our cohort was very homogenous in ethnicity, age and training age, limiting the extrapolation of conclusions to other ethnicities or older populations. Athletes were grouped according to a modified Mitchell classification of sports. However, differences within each CD-group persist (eg, rowers compete over the Olympic distance of ~6 min in the high CD-group, while Ironman triathletes compete over durations up to 9 hours). As a cross-sectional study, results are less sensitive to changes in time-dependent variables. Hence, the effects of training age may be challenging to ascertain in this study. There was an uneven number of men and women in the cross-sectional sample, and the low demand group was small (<20 athletes from either sex), resulting in wide CIs reflecting a poor precision for the low group. This consequently reduces the likelihood of attaining significance in comparative statistical analysis. Elite athletes perform supervised training that differs significantly from non-elite athletes in intensity and volume. Therefore, extrapolating the conclusions to recreational athletes should be done with caution. Functional adaptations in athletes can be difficult to assess without stress testing, and this was not performed on this cohort.

Author affiliations

[1]Department of Cardiology, Copenhagen University Hospital, Bispebjerg-Frederiksberg, Copenhagen, Denmark
[2]Section for Biostatistics and Evidence-Based Research, The Parker Institute, Frederiksberg, Denmark
[3]Department of Cardiology, Copenhagen University Hospital, Amager-Hvidovre Hospital, Copenhagen, Denmark
[4]Research Unit of Rheumatology, University of Southern Denmark, Department of Clinical Research, Odense, Denmark
[5]Department of Clinical Medicine, University of Copenhagen, Copenhagen, Denmark
[6]Department of Physical and Occupational Therapy, Copenhagen University Hospital, Bispebjerg-Frederiksberg, Copenhagen, Denmark

Acknowledgements The authors wish to acknowledge the contribution of Team Danmark for their help in recruitment of participants and dissemination of results to relevant partners.

Contributors MA contributed to data curation, formal analysis, funding acquisition, visualisation and writing of the original draft. SGT contributed to data curation, formal analysis and project administration. RC contributed to data analysis and supervision. JJT contributed to data analysis and supervision. CHD contributed to funding acquisition and supervision. HR contributed to conceptualisation, investigation, funding acquisition, visualisation and supervision. All authors contributed to methodology, writing-review and editing. All authors read and approved the final version of the manuscript and agreed with the authors' presentation order. AI (ChatGPT) was employed sparingly and selectively to refine the written language of the manuscript through sentence-level rephrasing. AI (ChatGPT) was used solely for rephrasing few individual sentences with the aim of improving the written language of the manuscript. HR is the guarantor of the manuscript.

Funding This work was funded and supported by a grant from the national Danish elite sports organisation (Team Danmark; grant id: N/A), Helsefonden (22-B-0423). Section for Biostatistics and Evidence-Based Research, the Parker Institute, Bispebjerg and Frederiksberg Hospital is supported by a core grant from the Oak Foundation (OCAY-18-774-OFIL). The Hartmann foundation Grant id: A38322).

Disclaimer AI (ChatGPT) was used solely for rephrasing individual sentences and searching for proper synonymes when needed, with the aim of improving the written language of the manuscript.

Competing interests None declared.

Patient and public involvement Patients and/or the public were involved in the design, or conduct, or reporting, or dissemination plans of this research. Refer to the Methods section for further details.

Patient consent for publication Not applicable.

Ethics approval This study involves human participants and was approved by the Regional Regional Ethics Committee of the Capital Region of Denmark (H-21043707). Participants gave informed consent to participate in the study before taking part.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available upon reasonable request. The data supporting this study will be made available upon reasonable request to the corresponding author.

ORCID iDs

Mikkel Aaroee http://orcid.org/0009-0005-8810-3251
Christian Have Dall http://orcid.org/0000-0002-5075-0980
Hanne Rasmusen http://orcid.org/0000-0002-3617-4112

REFERENCES

1 Jakovljevic DG. Physical activity and cardiovascular aging: physiological and molecular insights. *Exp Gerontol* 2018;109:S0531-5565(17)30100-6:67–74:.
2 Singam NSV, Fine C, Fleg JL. Cardiac changes associated with vascular aging. *Clin Cardiol* 2020;43:92–8.
3 Gjerdalen GF, Hisdal J, Solberg EE, *et al*. The Scandinavian athlete's heart; echocardiographic characteristics of male professional football players. *Scand J Med Sci Sports* 2014;24:e372–80.

4 Sheikh N, Papadakis M, Carre F, *et al*. Cardiac adaptation to exercise in adolescent athletes of African Ethnicity: an emergent elite athletic population. *Br J Sports Med* 2013;47:585–92.
5 Sharma S, Merghani A, Mont L. Exercise and the heart: the good, the bad, and the ugly. *Eur Heart J* 2015;36:1445–53.
6 Park J-H, Kim KH, Rink L, *et al*. Left atrial enlargement and its association with left atrial strain in university athletes participated in 2015 Gwangju summer Universiade. *Eur Heart J Cardiovasc Imaging* 2020;21:865–72.
7 Pelliccia A, Culasso F, Di Paolo FM, *et al*. Physiologic left ventricular cavity dilatation in elite athletes. *Ann Intern Med* 1999;130:23–31.
8 Chen YC, Voskoboinik A, Gerche AL, *et al*. Prevention of pathological atrial remodeling and atrial fibrillation. *J Am Coll Cardiol* 2021;77:S0735-1097(21)01233-X:2846–64:.
9 Song G, Liu J, Ren W, *et al*. Reversible changes of left atrial function during pregnancy assessed by two-dimensional speckle tracking echocardiography. *PLoS One* 2015;10:e0125347.
10 Merghani A, Malhotra A, Sharma S. The U-shaped relationship between exercise and cardiac morbidity. *Trends Cardiovasc Med* 2016;26:S1050-1738(15)00171-1:232–40:.
11 Flannery MD, Kalman JM, Sanders P, *et al*. State of the art review: atrial fibrillation in athletes. *Heart Lung Circ* 2017;26:S1443-9506(17)30503-6:983–9:.
12 Petrungaro M, Fusco L, Cavarretta E, *et al*. Long-term sports practice and atrial fibrillation: an updated review of a complex relationship. *J Cardiovasc Dev Dis* 2023;10:218.
13 Elliott AD, Linz D, Mishima R, *et al*. Association between physical activity and risk of incident arrhythmias in 402 406 individuals: evidence from the UK Biobank cohort. *Eur Heart J* 2020;41:1479–86.
14 Kunutsor SK, Seidu S, Mäkikallio TH, *et al*. Physical activity and risk of atrial fibrillation in the general population: meta-analysis of 23 cohort studies involving about 2 million participants. *Eur J Epidemiol* 2021;36:259–74.
15 Myrstad M, Aarønæs M, Graff-Iversen S, *et al*. Does endurance exercise cause atrial fibrillation in women *Int J Cardiol* 2015;184:S0167-5273(15)00256-9:431–2:.
16 Rao SJ, Shah AB. Exercise and the female heart. *Clin Ther* 2022;44:S0149-2918(21)00492-6:41–9:.
17 Nattel S. Physical activity and atrial fibrillation risk: it's complicated; and sex is critical. *Eur Heart J* 2020;41:1487–9.
18 Tischer SG, Mattsson N, Storgaard M, *et al*. Results of voluntary cardiovascular examination of elite athletes in Denmark: proposal for Nordic collaboration. *Scand J Med Sci Sports* 2016;26:64–73.
19 Lang RM, Bierig M, Devereux RB, *et al*. Recommendations for chamber Quantification: a report from the American society of echocardiography's guidelines and standards committee and the chamber Quantification writing group, developed in conjunction with the European Association of echocardiography, a branch of the European society of cardiology. *J Am Soc Echocardiogr* 2005;18:1440–63.
20 Lang RM, Badano LP, Mor-Avi V, *et al*. Recommendations for cardiac chamber Quantification by echocardiography in adults: an update from the American society of echocardiography and the European Association of cardiovascular imaging. *Eur Heart J Cardiovasc Imaging* 2015;16:233–71.
21 Badano LP, Miglioranza MH, Mihăilă S, *et al*. Left atrial volumes and function by three-dimensional echocardiography: reference values, accuracy, reproducibility, and comparison with two-dimensional echocardiographic measurements. *Circ Cardiovasc Imaging* 2016;9:e004229.
22 Barbier P, Solomon SB, Schiller NB, *et al*. Left atrial relaxation and left ventricular systolic function determine left atrial reservoir function. *Circulation* 1999;100:427–36.
23 Cuspidi C, Sala C, Tadic M, *et al*. Left atrial volume in elite athletes: A meta-analysis of echocardiographic studies. *Scand J Med Sci Sports* 2019;29:922–32.
24 D'Ascenzi F, Pelliccia A, Natali BM, *et al*. Increased left atrial size is associated with reduced atrial stiffness and preserved reservoir function in athlete's heart. *Int J Cardiovasc Imaging* 2015;31:699–705.
25 Nistri S, Galderisi M, Ballo P, *et al*. Determinants of echocardiographic left atrial volume: implications for normalcy. *Eur J Echocardiogr* 2011;12:826–33.
26 D'Ascenzi F, Pelliccia A, Natali BM, *et al*. Training-induced dynamic changes in left atrial reservoir, Conduit, and active volumes in professional soccer players. *Eur J Appl Physiol* 2015;115:1715–23.
27 Cheema B, Kinno M, Gu D, *et al*. Left atrial size and strain in elite athletes: A cross-sectional study at the NBA draft combine. *Echocardiography* 2020;37:1030–6.
28 Iskandar A, Mujtaba MT, Thompson PD. Left Atrium size in elite athletes. *JACC Cardiovasc Imaging* 2015;8:S1936-878X(15)00297-1:753–62:.
29 Simard F, Sanz-de la Garza M, Vaquer-Seguí A, *et al*. Sex as a main determinant of bi-atrial acute and chronic adaptation to exercise. *Eur J Appl Physiol* 2022;122:2585–96.
30 Pelliccia A, Kinoshita N, Pisicchio C, *et al*. Long-term clinical consequences of intense, uninterrupted endurance training in Olympic athletes. *J Am Coll Cardiol* 2010;55:1619–25.
31 Brugger N, Krause R, Carlen F, *et al*. Effect of lifetime endurance training on left atrial mechanical function and on the risk of atrial fibrillation. *Int J Cardiol* 2014;170:419–25.
32 Sanchis L, Garza M, Bijnens B, *et al*. Gender influence on the adaptation of atrial performance to training. *Eur J Sport Sci* 2017;17:720–6.
33 Hirose T, Kawasaki M, Tanaka R, *et al*. Left atrial function assessed by speckle tracking echocardiography as a Predictor of new-onset non-valvular atrial fibrillation: results from a prospective study in 580 adults. *Eur Heart J Cardiovasc Imaging* 2012;13:243–50.