ESC European Society of Cardiology

European Heart Journal - Cardiovascular Imaging (2026) **27**, 490–501
https://doi.org/10.1093/ehjci/jeaf333

ORIGINAL PAPER

Left atrioventricular ratio (LA:LV): using left ventricular size as the reference for identifying maladaptive left atrial remodelling

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Received 10 April 2025; revised 7 July 2025; accepted 11 November 2025; online publish-ahead-of-print 27 November 2025

See the editorial comment for this article 'One size atrium does not fit all ventricles', by N.J. Kelly and L.E. Sade, https://doi.org/10.1093/ehjci/jeaf334.

Introduction Remodelling of the left atrium (LA) and left ventricle (LV) occurs in response to pathological and physiological stimuli, yet their inter-dependence is often overlooked in clinical practice. The left atrioventricular ratio (LA:LV)—the ratio of maximal LA end-systolic volume (LAESV) to LV end-diastolic volume (LVEDV)—may offer valuable context for distinguishing physiological from pathological cardiac remodelling.

Methods and results This study evaluated LA:LV, assessed via echocardiography, and cardiorespiratory fitness assessed as peak oxygen uptake (VO_2peak) in a multi-centre international cohort spanning the cardiorespiratory fitness spectrum. Exercise capacity in healthy participants was categorized by VO_2 peak quartiles, and cardiac structural differences were analysed. Among 2943 adults (1600 healthy, 1343 pathology), healthy individuals had a median LA:LV of 0.49 [0.38, 0.61], consistent with LVEDV being roughly twice the LAESV. Pathology revealed higher LA:LV ratios [0.53 (0.38–0.75), $P < 0.001$], with marked elevations amongst AF [0.60 (0.45–0.78)] and HFpEF [0.70 (0.51–0.88)]—a 30% increase vs. healthy adults. The highest indexed LA volumes occurred in the highest VO_2 peak quartile [Q4: 36 (28–46) mL/m^2], while the LA:LV ratio was highest in Q1 [0.53 (0.42–0.69)]. Among participants with elevated LAVi (≥34 mL/m^2), concordance with elevated LA:LV ratio (≥0.75) varied markedly by fitness level: ~60% in Q1-Q2 vs. only 7% in Q4, highlighting the importance of fitness context when interpreting LA enlargement.

Conclusion The LA:LV ratio effectively discriminates between adaptive and maladaptive atrial remodelling. LA:LV is typically ~0.5. Lower ratios correlate with higher functional capacity and physiological remodelling, whereas ratios ≥0.75 may indicate pathological remodelling and warrant consideration of atrial pathology.

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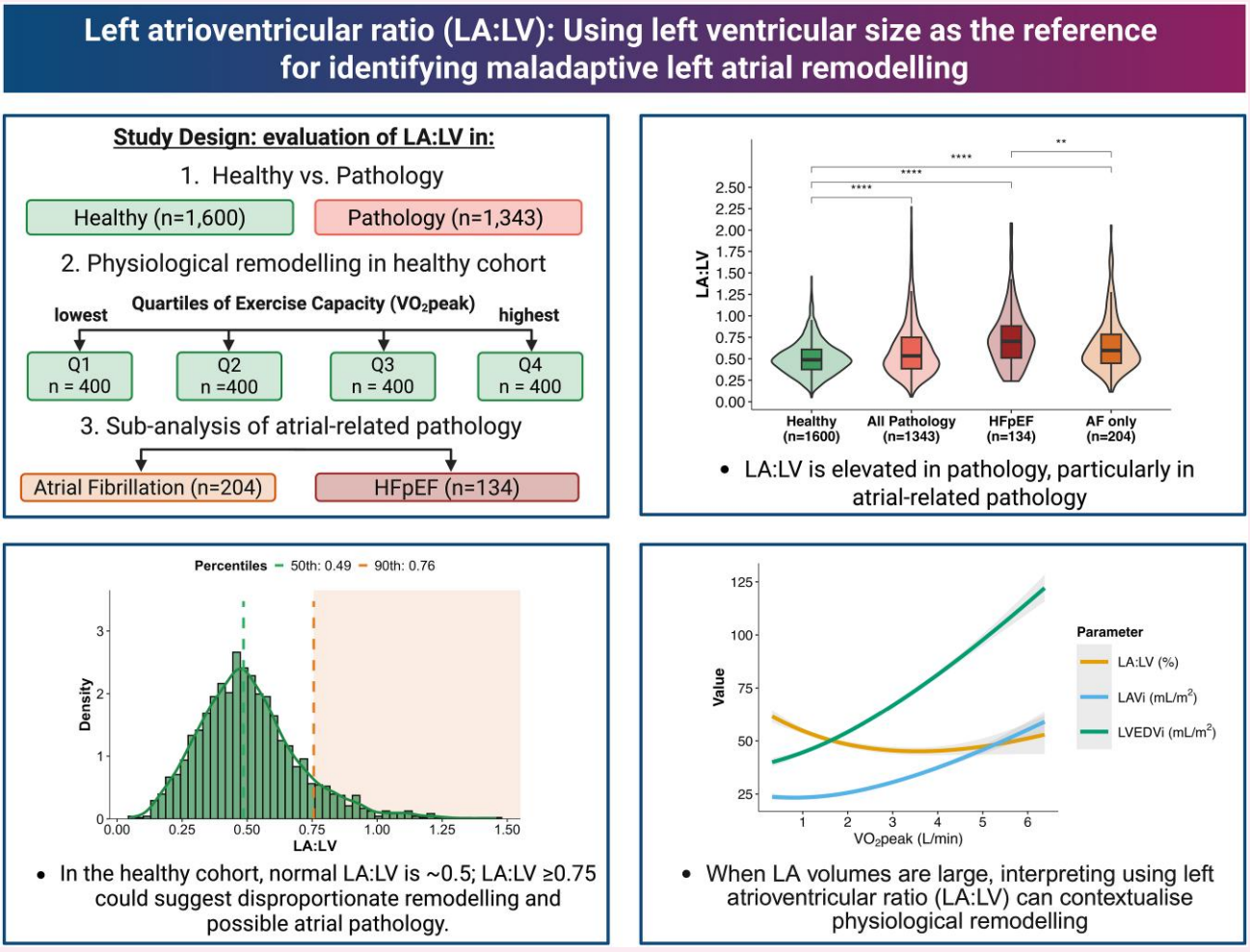
Graphical Abstract



Clinical utility of the left atrioventricular ratio. *Created in BioRender. La gerche, A. (2025)* https://BioRender.com/05z5mzg.

Keywords cardiac remodelling • cardiorespiratory fitness • echocardiography • functional capacity • left atrioventricular ratio • LA:LV

Introduction

Remodelling of the LA and left ventricle (LV) is a response to both physiological and pathological stimuli. While LA enlargement is a well-recognized marker of cardiovascular risk and disease progression,[1] it is also a hallmark of physiological adaptations, particularly in endurance athletes[2,3] and during pregnancy.[4] This duality presents a diagnostic challenge: how can clinicians differentiate between benign and maladaptive forms of left atrial enlargement?

Traditionally, the clinical focus has been on absolute LA volume as a predictor of adverse outcomes, including atrial fibrillation (AF), heart failure (HF), and stroke.[5–7] However, standalone measurements of LA size may be misleading, particularly in individuals with preserved ejection fraction (EF), where LV size and function play a more significant role in determining overall cardiac performance and exercise capacity.[8] Additionally, indexing measurements to body surface area (BSA) can be problematic, as it leads to underestimation of dilation in overweight individuals.[9] LV volume, particularly when indexed to body size, has been shown to correlate more strongly with exercise capacity than LA volume alone.[10,11] This raises an important question: should LA size be evaluated in isolation, or should it be considered in relation to LV size to better contextualize their proportionality and clinical significance?

The left atrioventricular ratio (LA:LV), calculated as LA end-systolic volume (LAESV) divided by LV end-diastolic volume (LVEDV), provides a simple yet physiologically grounded approach to this issue.[12] By using the maximum volumes of both chambers, occurring at different cardiac phases, the ratio captures the interplay between the LA, which serves as a reservoir and conduit for LV filling, and the LV, the heart's primary pumping chamber.[13,14] In physiological states, such as athletic training, LA and LV remodelling enlargement has been suggested to be proportionate.[15] Conversely, disproportionate LA enlargement could be indicative of maladaptive structural changes.[16]

This study aims to explore how exercise capacity impacts LA:LV remodelling in a population of healthy and diseased individuals. We

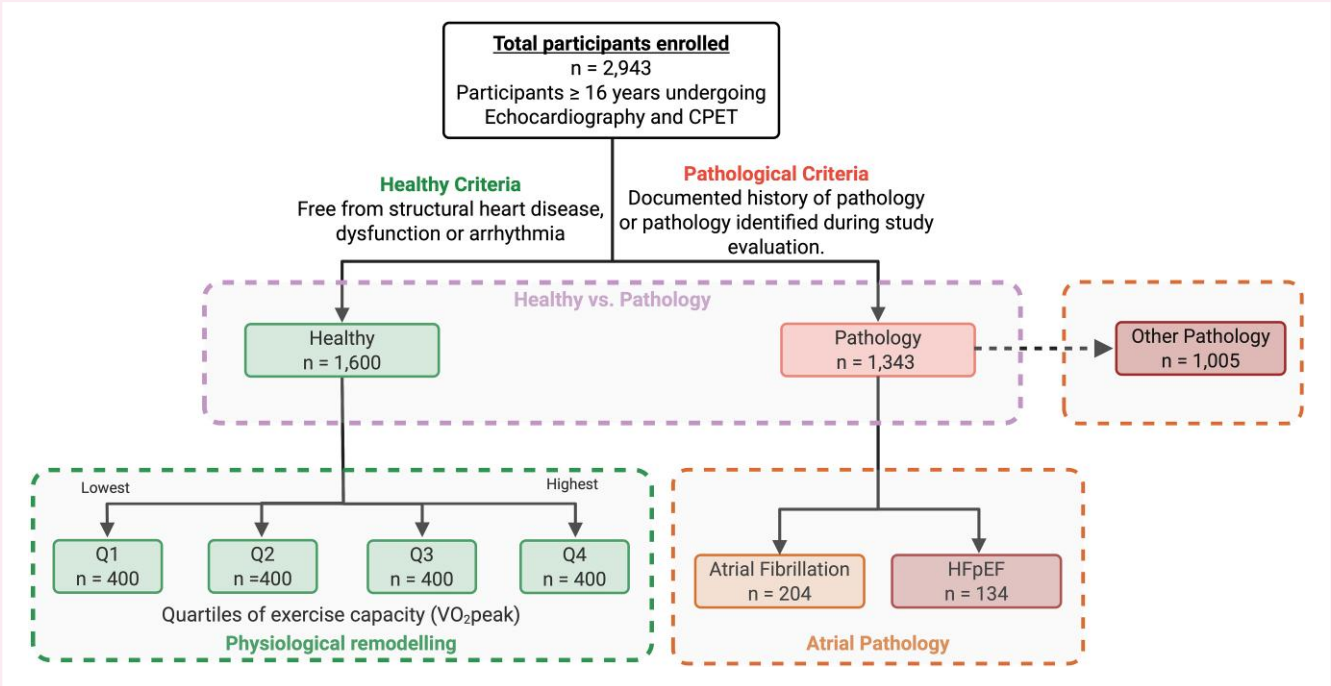


Figure 1 Study flow diagram. Study flow depicting cohort derivation from 2943 participants who underwent TTE and CPET between April 2016 and January 2024, stratified into healthy ($n = 1600$) and pathology ($n = 1343$) cohorts. Created in BioRender. La Gerche, A. (2025) https://BioRender.com/kq2vp9m.

hypothesize that a lower LA:LV ratio indicates adaptive remodelling and is associated with higher exercise capacity, whereas elevated ratios reflect maladaptive changes linked to reduced exercise tolerance and underlying cardiac pathology. By investigating the relationship between the LA:LV and VO_2 peak across a diverse international cohort, spanning asymptomatic athletes to patients with heart failure with preserved ejection fraction (HFpEF), this study seeks to characterize the LA:LV ratio as potential marker for distinguishing adaptive from maladaptive cardiac remodelling.

Methods

Study design and population

This prospective, international multi-centre observational study included 2943 participants aged ≥16 years who underwent transthoracic echocardiography (TTE) and cardiopulmonary exercise testing (CPET) between April 2016 and January 2024 (*Figure 1*). Participants were identified from two primary locations: (i) Melbourne, Australia ($n = 512$), including participants from Baker Heart and Diabetes Institute and St Vincent's Institute of Medical Research, Melbourne, Australia, comprising elite endurance athletes and healthy controls enrolled in prospective research protocols, including a dedicated cohort with documented AF; and (ii) Hasselt, Belgium ($n = 2431$), comprising consecutive patients referred to Jessa Hospital for clinical evaluation of dyspnoea. Patients with exercise intolerance and no former clinical diagnosis were diagnosed with HFpEF based on a H_2FPEF score ≥6 or a HFA-PEFF score ≥5. Clinical research studies were conducted in accordance with the ethical standards set forth by the Human Research Ethics Committees at the respective participating sites.

Study cohort derivation

Primary analysis: complete cohort ($n = 2943$)

The initial characterization of the LA:LV relationship across the spectrum of cardiovascular health and disease included all individuals aged ≥16 years with available TTE and CPET measurements. Participants were categorized into either a Pathology or Healthy cohort.

Pathology cohort ($n = 1343$)

Participants were systematically categorized as having cardiac pathology sequentially based on: (i) history of ischemic heart disease or myocardial infarction ($n = 614$); (ii) congenital heart disease including Fontan circulation ($n = 9$); (iii) moderate or severe valvular heart disease ($n = 86$); (iv) LV systolic dysfunction, defined as LVEF <50% ($n = 237$); (v) pulmonary hypertension, defined as pulmonary artery systolic pressure ≥50 mmHg or mean pulmonary artery pressure ≥35 mmHg at rest ($n = 59$); (vi) Positive HFpEF probability scores, including HFpEF with AF ($n = 134$); and (vii) AF only, either documented history or detected during exercise testing ($n = 204$). Sub-analysis within the Pathology cohort was performed to examine atrial-related pathology in HFpEF (group 6) and AF (group 7).

Healthy cohort ($n = 1600$)

To establish normative LA:LV relationships across the spectrum of exercise capacity while minimizing confounding from pathological remodelling, we applied stringent pathological categorization. The healthy cohort of 1600 ostensibly healthy participants, including 326 endurance athletes, represents individuals free from significant structural heart disease, systolic or diastolic dysfunction, and arrhythmia, thereby providing a reference population for cardiac adaptation across varying levels of exercise capacity. The cohort was stratified into quartiles based on VO_2 peak: Q1 ($n = 400$, lowest exercise capacity), Q2 ($n = 400$), Q3 ($n = 400$) and Q4 ($n = 400$, highest exercise capacity) to demonstrate healthy LA:LV remodelling.

Clinical information and anthropometry

Standardized protocols were implemented across all centres for measurements and data collection procedures. Body height and weight were recorded, and body mass index (BMI) was calculated, along with BSA

derived using the Du Bois method. Resting systolic (SBP) and diastolic blood pressure (DBP) were measured. Clinical information was collected by lifestyle questionnaire.

Echocardiography

A comprehensive TTE was performed (Vivid E9 or E95 ultrasound system, GE Healthcare, Horten, Norway) at rest, with participants lying at a 45° angle on a semi-supine tilt table ergometer for exercise echocardiography (Belgian cohort) or alternatively, in the lateral decubitus position. The acquisition of resting two-dimensional (2D), Doppler, and tissue Doppler images was conducted in accordance with the current guidelines.[17,18] The left atrial end-systolic volume (LAESV) and left ventricular end-diastolic volume (LVEDV) were calculated using the biplane method of disks summation technique. The LA and LV were both indexed to BSA, respectively (LAVi and LVEDVi). Conventional and tissue Doppler images were obtained from the four-chamber view to derive measurements of diastolic filling parameters, including the mitral peak early inflow velocity (E) and the mitral peak late inflow velocity (A). The mitral annular early diastolic (e′) longitudinal velocity was evaluated through pulsed-wave tissue Doppler, and the ratio of the early mitral inflow to early diastolic mitral annular velocity (E/e′) was calculated as an average of septal and lateral e′, which serves as an estimate of LV filling pressures. Analysis of TTE images was conducted at one of two core laboratory facilities, both utilizing the same software (EchoPAC^{TM}, GE Healthcare, Horten, Norway) and standardized methods.

Cardiopulmonary exercise testing

CPET was performed using an individualized continuous ramp protocol on either a semi-supine (Belgian cohort)[19] or upright (Australian cohort) bicycle ergometer (Lode, Groningen, The Netherlands). Gas analysis, including volume of oxygen uptake (VO_{2-}), expired carbon dioxide (VCO_2) and respiratory exchange ratio (RER) were measured throughout the test. Exercise capacity was assessed using peak oxygen uptake (VO_2peak) and defined as a 30-s rolling average of the six highest 5-s oxygen uptake values. Heart rate (HR) and rhythm were monitored continuously throughout exercise using a 12-lead electrocardiogram. Maximum exercise testing was defined as an RER >1.05 and/or maximal HR >90% predicted. Percentage of predicted exercise capacity ($VO_{2\%pred}$) was used to adjust for expected differences in age, sex, and body habitus. $VO_{2\%pred}$ was calculated as VO_2 peak indexed to normal standards for exercise capacity derived from the Wasserman/Hansen equation, a standard method for estimating exercise capacity based on age, sex, and body habitus.[20]

LA:LV and exercise capacity

The association between the LA:LV and VO_2 peak was evaluated using logistic regression to account for the non-linear distribution of the data. The study population was specifically designed to include the upper and lower limits of the physiological remodelling spectrum. To establish physiological risk zones for LA:LV, we identified thresholds for disproportionate remodelling at the 75th, 90th, 95th, and 97.5th percentiles, corresponding to values of 0.61, 0.76, 0.87, and 0.97, respectively (see Supplementary data online, *Figure S1*). These values were rounded to 0.5, ≥0.60, ≥0.75, ≥0.85 and ≥0.95, respectively, to improve interpretability and to align with clinically relevant thresholds.

Statistical analyses

Continuous variables were tested for normality using the Shapiro-Wilk test and visual inspection of Q-Q plots. Due to non-normal distribution, continuous variables are presented as median with interquartile range [25th percentile, 75th percentile], whereas categorical variables are summarized as frequencies and percentages. Descriptive statistics were calculated for the entire cohort. Between-group comparisons were performed using the Wilcoxon rank-sum test for continuous variables and Fisher's exact test for categorical variables. For comparisons across multiple groups (healthy, pathology, AF, HFpEF), pairwise Wilcoxon tests were used. The relationship between exercise capacity and LA:LV was assessed using logarithmic regression modelling [LA:LV ~ log (VO_2 peak)] based on visual inspection of the data distribution. Model fit was assessed using R^2 and Pearson's correlation coefficient. Sex-stratified analyses were performed to examine potential sex-specific relationships. Cohen's *d* was calculated to quantify effect sizes between groups. Statistical analysis was conducted using RStudio (R Foundation for Statistical Computing, Vienna, Austria), with statistical significance set at $P < 0.05$.

Results

Complete cohort

We analysed LA:LV ratios and exercise capacity in 2943 adults (1600 healthy, 1343 pathological). The pathology cohort demonstrated significantly higher LA:LV ratios than healthy adults [median: 0.53 (0.38–0.75) vs. 0.49 (0.38–0.61), $P < 0.001$], representing a 10% median increase (*Figure 2*). Among specific pathologies, both AF [$n = 204$; median: 0.60 (0.45–0.78)] and HFpEF [$n = 134$; median: 0.70 (0.51–0.88)] showed marked LA:LV elevation, a 30% increase compared with healthy adults (both $P < 0.001$; Supplementary data online, *Figure S2*). The inverse relationship between LA:LV and exercise capacity across the complete cohort is shown in Supplementary data online, *Figure S3*.

Healthy cohort and demographics

Table 1 summarizes baseline characteristics in the healthy population. The mean VO_2peak of this study population was 1.55 L/min [1.13, 2.36], equivalent to 20.1 mL/kg/min [14.8, 31.0]. The median age of the study population was 60 years [47, 70]. Age decreased between quartiles of VO_2peak, from 71 years [65, 77] in Q1, to 63 years [56, 71] in Q2, to 58 years [48, 65] in Q3, and reaching the lowest age of 38 years [22, 53] in Q4. The overall population was 50% female and 50% male. Sex distribution varied across exercise capacity quartiles, with females comprising 79% of Q1, 65% of Q2, 37% of Q3, and 21% of Q4. The median BMI was 25.7 kg/m^2 [23.2, 29.3], with a resting HR of 70 bpm [63, 80] and resting blood pressure measurements of 136 [123, 150] mmHg systolic and 79 [70, 87] mmHg diastolic. Concerning medications, 10% were on statin therapy, 9% were on angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARB), and 8% were on beta-blocker or calcium channel blocker therapy. Lifestyle risk factors included 4% active smokers, 19% with a history of smoking, 13% with diabetes, and 33% with hypertension.

Cardiac remodelling and exercise capacity

Figure 3 shows the distribution of LA:LV ratios across quartiles of exercise capacity. The data reveals a progressive decrease in disproportionate LA:LV ratios in higher exercise capacity quartiles, suggesting that better exercise capacity is associated with more favourable atrioventricular remodelling. LA and LV volumes, along with the LA:LV volumetric ratio, are further stratified by quartiles of exercise capacity in *Figure 4*. LAVi was highest in Q4 [36 mL/m^2 (28, 46)], followed by Q1 [24 mL/m^2 (18, 29)], Q3 [23 mL/m^2 (17, 30)] and Q2 [22 mL/m^2 (17, 29)]. The population's median LA:LV was 0.49 [0.38, 0.61], demonstrating that the maximal LV volume is typically double the size of the corresponding maximal LA value. This remained true throughout exercise capacity as changes in LA:LV were modest, with highest LA:LV in Q1 [0.53 (0.42, 0.69)], minor decreases through Q2 [0.50 (0.37, 0.62)], Q3 [0.45 (0.32, 0.59)], and a slight rise in Q4 [0.48 (0.40, 0.56)]. Notably, both absolute LA and LV volumes were greatest in Q4, reflecting the healthy cardiac remodelling associated with enhanced exercise capacity.

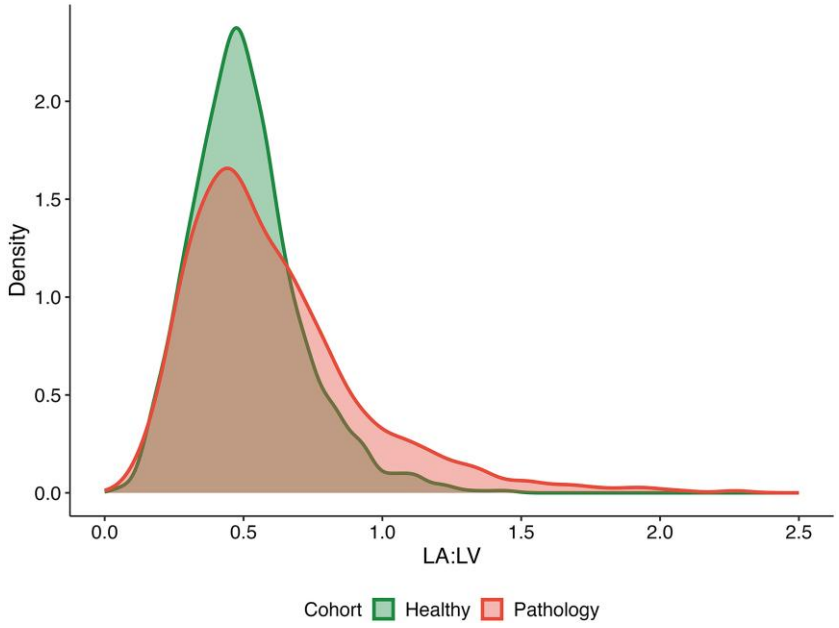


Figure 2 Distribution of left atrioventricular ratio in the complete cohort. Density plot showing the LA:LV distribution in the complete cohort ($n = 2943$), comprising healthy participants ($n = 1600$) and participants with cardiac pathology ($n = 1343$).

Table 1 Baseline characteristics by exercise capacity (VO_2 peak) quartiles in the healthy cohort ($n = 1600$)

	All	Q1	Q2	Q3	Q4
n	1600	400	400	400	400
Demographics					
Sex					
Male	796 (49.8)	83 (20.8)	141 (35.2)	254 (63.5)	318 (79.5)
Female	804 (50.2)	317 (79.2)	259 (64.8)	146 (36.5)	82 (20.5)
Age, y	60 [47, 70]	71 [65, 77]	63 [56, 71]	58 [48, 65]	38 [22, 53]
Anthropometric					
Body weight, kg	76 [67, 86]	71 [62, 80]	76 [67, 86]	81 [70, 91]	77 [68, 86]
BMI, kg/m^2	25.7 [23.2, 29.3]	26.8 [23.7, 30.0]	27.1 [23.9, 30.1]	26.8 [23.9, 30.3]	23.6 [21.7, 25.9]
Hemodynamic					
Resting HR, bpm	70 [63, 80]	70 [62, 79]	72 [65, 79]	70 [63, 81]	70 [61, 80]
BP Sys, mmHg	136 [123, 150]	143 [132, 155]	140 [127, 153]	137 [122, 149]	126 [117, 137]
BP Dia, mmHg	79 [70, 87]	80 [73, 88]	82 [74, 90]	81 [73, 89]	71 [63,80]
Medications					
Statin (%)	166 (10.4)	66 (16.5)	42 (10.5)	42 (10.5)	16 (4.0)
ACE/ARB (%)	150 (9.4)	56 (14.0)	39 (9.8)	36 (9.0)	19 (4.8)
B-blockers/CCB (%)	128 (8.0)	58 (14.5)	31 (7.8)	34 (8.5)	5 (1.2)
Risk Factors					
Current Smoker (%)	61 (3.8)	20 (5.0)	27 (6.8)	14 (3.5)	0 (0.0)
Ex smoker (%)	298 (18.6)	84 (21.0)	89 (22.2)	87 (21.8)	38 (9.5)
Diabetes (%)	208 (13.0)	82 (20.5)	68 (17.0)	53 (13.2)	5 (1.2)
Hypertension (%)	520 (32.5)	201 (50.2)	170 (42.5)	121 (30.2)	28 (7.0)
Exercise Capacity					
VO_2 peak, L/min	1.55 [1.13, 2.36]	0.91 [0.77, 1.03]	1.30 [1.21, 1.44]	1.86 [1.68, 2.09]	3.40 [2.80, 4.21]

Continued

Table 1 Continued

	All	Q1	Q2	Q3	Q4
VO_2 peak, mL/kg/min	20.1 [14.8, 31.0]	12.4 [10.5, 14.6]	17.4 [15.2, 19.9]	23.4 [19.9, 27.9]	45.8 [35.9, 57.8]
Echocardiographic					
Left Ventricular Structure					
IVSd, cm	1.0 [0.8, 1.1]	1.0 [0.9, 1.2]	1.0 [0.9, 1.1]	1.0 [0.8, 1.1]	0.9 [0.8, 1.0]
LVPWd, cm	0.9 [0.8, 1.0]	0.9 [0.8, 1.0]	0.9 [0.8, 1.0]	0.9 [0.8, 1.0]	0.9 [0.8, 1.0]
LVM, g	148 [118, 181]	133 [109, 164]	132 [108, 159]	152 [118, 181]	171 [147, 203]
LVMi, g/m^2	78 [64, 92]	75 [62, 92]	72 [59, 85]	77 [62, 90]	88 [74, 101]
LVIDd, cm	4.6 [4.2, 5.1]	4.3 [4.0, 4.7]	4.4 [4.0, 4.7]	4.7 [4.3, 5.0]	5.2 [4.8, 5.6]
RWT	0.38 [0.33, 0.44]	0.41 [0.35, 0.47]	0.40 [0.34, 0.46]	0.38 [0.32, 0.45]	0.35 [0.31, 0.39]
Left Atrial Structure					
LAESV, mL	47 [34, 64]	41 [31, 53]	40 [32, 53]	45 [33, 58]	70 [54, 89]
LAVi, mL/m^2	25 [18, 33]	24 [18, 29]	22 [17, 29]	23 [17, 30]	36 [28, 46]
LA:LV	0.49 [0.38, 0.61]	0.53 [0.42, 0.69]	0.50 [0.37, 0.62]	0.45 [0.32, 0.59]	0.48 [0.40, 0.56]
Left Ventricular Volumes and Function					
LVEDV, mL	96 [74, 127]	75 [63, 90]	82 [69, 100]	101 [84, 119]	145 [121, 177]
LVEDVi, mL/m^2	51 [41, 65]	42 [35, 52]	45 [38, 54]	52 [44, 61]	75 [62, 90]
LVEF, %	61 [56, 66]	61 [57, 68]	63 [56, 69]	62 [57, 67]	58 [55, 63]
LV GLS	−19 [−20, −17]	−16 [−18, −14]	−18 [−21, −14]	−19 [−20, −18]	−19 [−21, −18]
Diastolic Function					
Mitral E-wave velocity, cm/s	62 [51, 75]	61 [52, 73]	61 [52, 73]	59 [49, 70]	69 [55, 81]
Mitral A-wave velocity, cm/s	61 [48, 76]	74 [63, 88]	68 [58, 80]	60 [48, 72]	44 [35, 54]
E/A	0.98 [0.78, 1.35]	0.80 [0.70, 1.00]	0.90 [0.72, 1.07]	0.93 [0.76, 1.26]	1.60 [1.20, 2.21]
E/e′	7.4 [6.1, 9.7]	9.8 [8.1, 12.1]	8.7 [7.4, 10.8]	7.7 [6.3, 9.4]	6.3 [5.2, 7.2]

Data is presented as the number (*n*) and percentage (%) or as median with interquartile range [25th percentile, 75th percentile].
Abbreviations: ACE, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BMI, body mass index; BP, blood pressure; CCB, calcium channel blockers; Dia, diastolic; E/A, ratio of early to late mitral inflow velocity; E/e′, ratio of early mitral inflow velocity to early mitral annular velocity; HR, heart rate; IVSd, interventricular septal wall thickness at end-diastole; LA:LV, left atrioventricular ratio; LAESV, left atrial end-systolic volume; LAVi, left atrial volume indexed to BSA; LV GLS, left ventricular global longitudinal strain; LVEDV, left ventricular end-diastolic volume; LVEDVi, left ventricular end-diastolic volume indexed to BSA; LVEF, left ventricular ejection fraction; LVIDd, left ventricular internal diameter at end-diastole; LVM, left ventricular mass; LVMi, left ventricular mass indexed to BSA; LVPWd, left ventricular posterior wall thickness at end-diastole; RWT, relative wall thickness; Sys, systolic; VO_2 peak, peak oxygen consumption.

Using the LAVi threshold of 34 mL/m^2, 54.5% ($n = 218$) of Q4 participants exceeded this cutoff, compared with 16.0% ($n = 64$) in Q3, 9.5% ($n = 38$) in Q2, and 13.8% ($n = 55$) in Q1. Conversely, when applying the LA:LV threshold of 0.75, a reverse pattern emerged: 17.8% ($n = 71$) in Q1, 13.2% ($n = 53$) in Q2, 7.5% ($n = 30$) in Q3, and 4.2% ($n = 17$) in Q4 met these criteria. Most importantly, among participants with LAVi ≥34 mL/m^2, the concordance with elevated LA:LV (≥0.75) varied markedly by exercise capacity. Only 7% (16/218) of Q4 participants with elevated LAVi also demonstrated elevated LA:LV, compared with 23% (15/64) in Q3, 55% (21/38) in Q2, and 62% (34/55) in Q1.

The relationship between exercise capacity and LV volumes, LA volumes and LA:LV (as a percentage) are presented altogether in *Figure 5*. A curvilinear relationship was observed between LA:LV and VO_2 peak with individuals with the lowest VO_2 peak demonstrating higher LA:LV, then a very weakly negative relationship between LA:LV and VO_2 peak. *Figure 6* shows a scatterplot of the population. After adjusting for age, sex, and anthropometric differences using the Wasserman-Hansen equation (see Supplementary data online, *Figure S4*), the curvilinear relationship between LA:LV and exercise capacity was attenuated, showing a weak negative linear relationship between LA:LV and $VO_{2\%pred}$.

Structural vs. exercise remodelling and exercise capacity

To further investigate the clinical utility of LA:LV, we examined the relationship of LA:LV with GLS in 477 patients where both measurements were available (see Supplementary data online, *Tables S1–3*). Both LA:LV ($r = -0.255$, $P < 0.001$) and LV global longitudinal strain ($r = -0.241$, $P < 0.001$) demonstrated similar inverse correlations with VO_2 peak (see Supplementary data online, *Figure S5*). Notably, LA:LV and LV GLS were not correlated with each other ($r = 0.030$, $P = 0.52$), suggesting these parameters capture different aspects of cardiac remodelling. In multivariable regression analysis, both parameters remained independent predictors of exercise capacity (LA:LV: $\beta = -1.85$, $P < 0.001$; LV GLS: $\beta = -0.11$, $P < 0.001$), with the combined model explaining 11.9% of VO_2 peak variance compared with 6.5% and 5.8% for each parameter alone (see Supplementary data online, *Table S2*). The absence of multicollinearity (VIF = 1.001 for both parameters) supports their complementary nature of LA:LV.

Sex differences in cardiac remodelling

When stratified by sex, the correlation between VO_2 peak and LA:LV was found to be slightly higher in females ($r = -0.08$; $P = 0.027$)

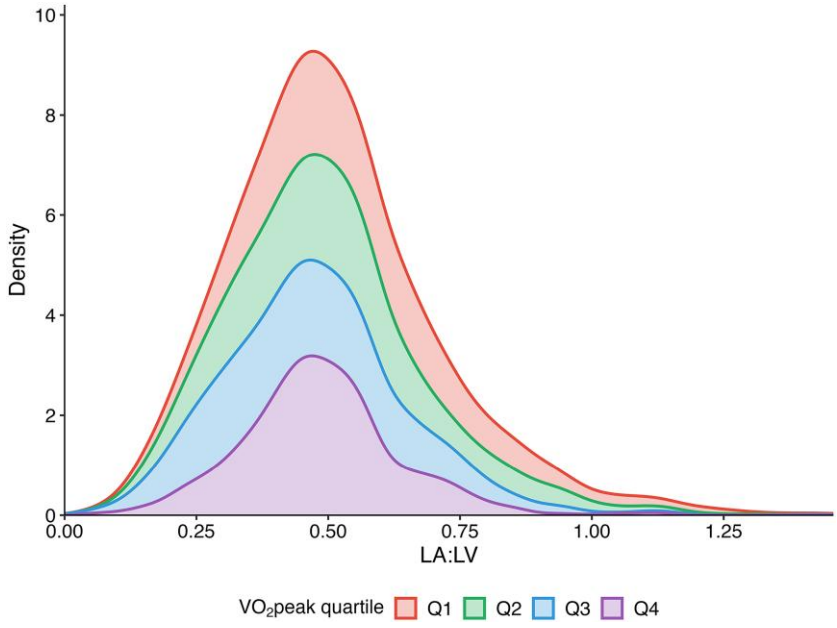


Figure 3 Distribution of left atrioventricular ratio across exercise capacity quartiles in the healthy cohort. Stacked density plot showing LA:LV distribution across quartiles of exercise capacity (VO_2 peak) in the healthy cohort ($n = 1600$). Q1 = lowest exercise capacity, Q4 = highest exercise capacity.

compared with males ($r = -0.03$; $P = 0.427$). However, part of this could be attributed to males demonstrating significantly greater exercise capacity, with the highest VO_2 peak in males being 44% higher than the highest value observed in females. Furthermore, the female cohort demonstrated a higher prevalence of severely reduced exercise capacity, resulting in overrepresentation within the lower VO_2 peak quartiles where the relationship with LA:LV exhibits a steeper, non-linear increase.

Discussion

Differentiating healthy and pathological remodelling

In this large, multi-centre international cohort, we observed a high prevalence of LA enlargements and examined the utility of LA:LV as a tool to differentiate between physiological and pathological atrial remodelling (Graphical Abstract). We found that LA:LV was increased in individuals with cardiac pathology, especially in conditions where disproportionate atrial remodelling might be expected, such as AF or HFpEF. Our findings also support the value of the LA:LV as a supplementary marker to contextualize LA enlargement in subjects without known cardiac pathology. In this setting, individuals with the highest exercise capacity had the largest LA volumes but normal LA:LV. Conversely, an elevated LA:LV was predominantly found among individuals with reduced exercise capacity, which could indicate maladaptive remodelling. These results highlight the importance of considering LA:LV proportionality, rather than relying solely on LA size, when assessing diastolic function and cardiovascular risk. We suggest that an LA:LV of ~ 0.5 suggests physiological normalcy and found that an LA:LV cut point of 0.75 could be used as a cut-off to indicate disproportionate remodelling.

LA:LV ratio as a contextual marker of cardiac remodelling

The strength of the LA:LV as a measurement lies in its ability to provide physiological context for cardiovascular risk stratification. Traditionally, an enlarged LA has been viewed as a surrogate marker of chronic volume overload and elevated filling pressures, with established associations to adverse outcomes such as AF, HF, and stroke. However, as observed in our study, this interpretation does not always apply in cases of ostensibly healthy remodelling, such as in endurance athletes or in pregnancy, where LA enlargement is a balanced adaptation to meet increased circulatory demands. In these cases, large LA volumes are accompanied by proportionate LV remodelling, reflected by low LA:LV. This proportionality indicates adaptive rather than maladaptive remodelling, reinforcing the notion that LA size alone does not determine clinical significance.

LA:LV in the athlete's heart

Approximately 40% of highly trained endurance athletes have a severely dilated LA.[21] This falls under the spectrum of the 'athlete's heart' phenomenon, in which symmetrical enlargement of all cardiac chambers is well described. When 'indexed' to LV size, a low LA:LV reflects this physiological symmetry, highlighting enhanced flow rates and stroke volume. However, at the upper extremes of exercise capacity, there was a slightly elevated and more heterogenous relationship with LA:LV, indicated by wider variability. Although the Healthy cohort was devised to remove overt pathology such as AF, the subtle deviation from the expected trend could suggest maladaptive remodelling in those with exceptionally high exercise capacity. These observations align with the extreme exercise hypothesis, which suggests that the upper extremes of exercise capacity may be associated with pathological outcomes, including AF.[22–27] A U-shaped curve has been described between exercise capacity and AF, such that AF prevalence is most common among those with the lowest and highest exercise

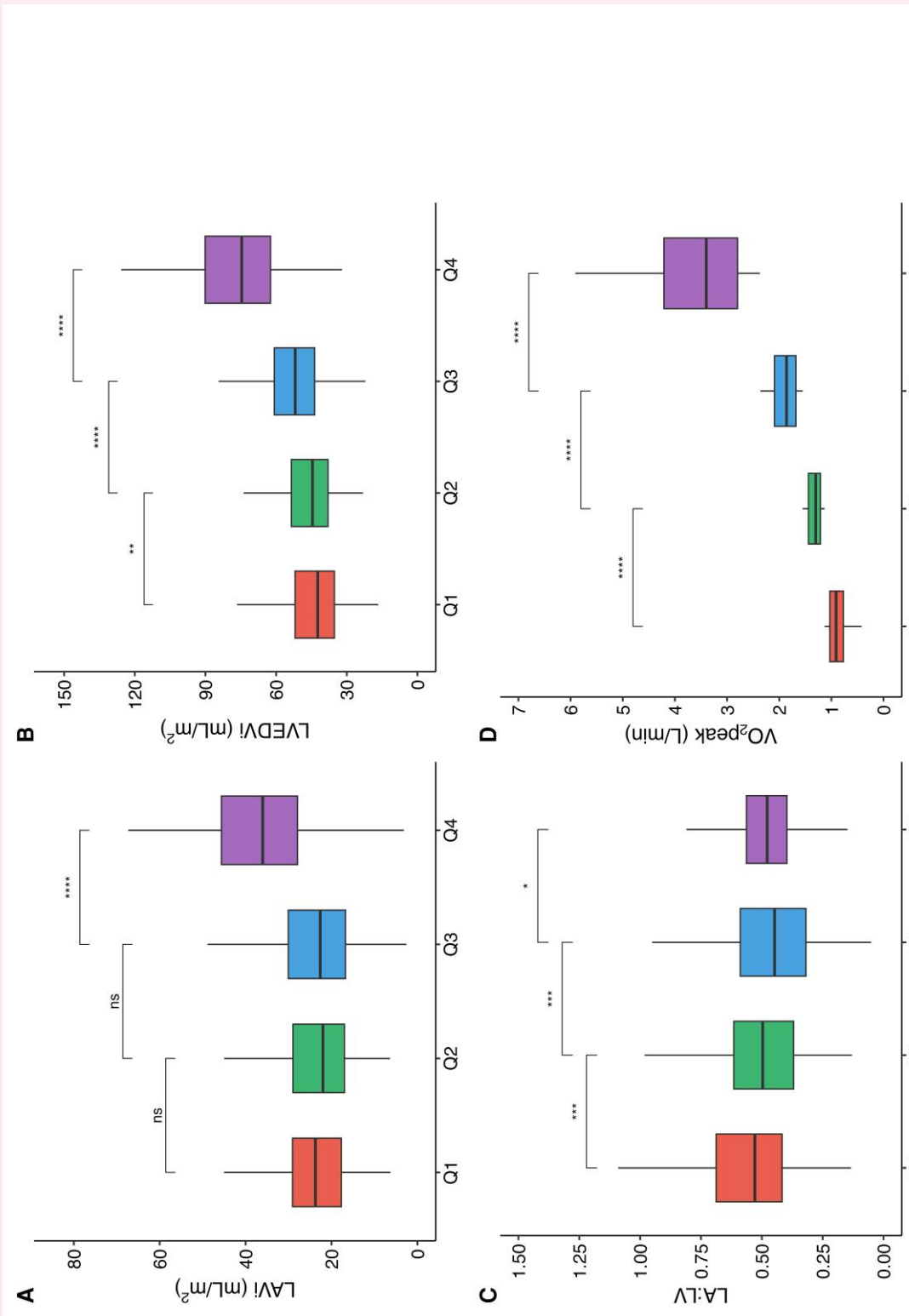


Figure 4 Cardiac volumes and left atrioventricular ratio across exercise capacity quartiles in the healthy cohort. Left atrial volume index (LAVi), left ventricular end-diastolic volume index (LVEDVi), and left atrioventricular ratio (LA:LV) across exercise capacity quartiles (Q1 = lowest, Q4 = highest) in the healthy cohort ($n = 1600$). Adjacent quartile comparisons: ns = $P > 0.05$; *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$; ****$P < 0.0001$.

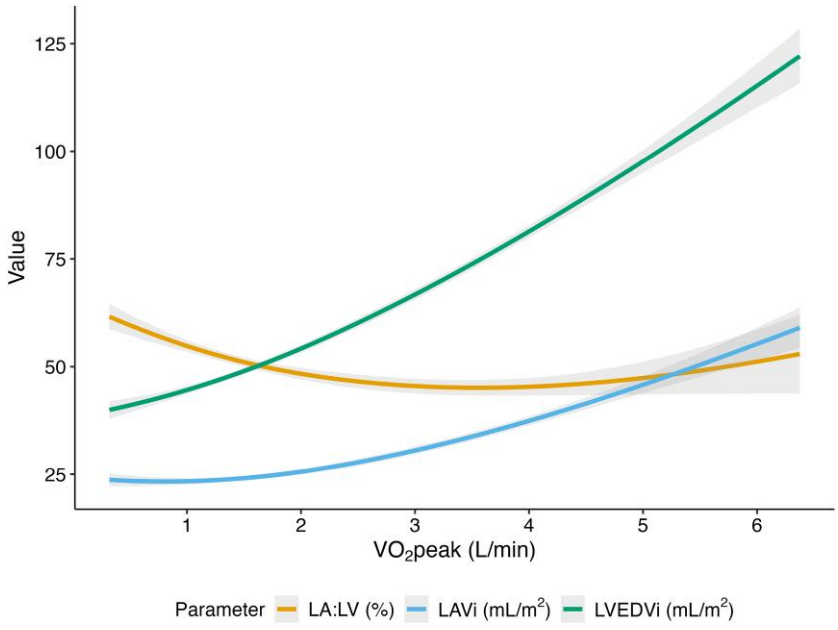


Figure 5 Relationship between LA, left ventricle and exercise capacity in the healthy cohort. Loess regression analysis between left atrial volume index (LAVi), left ventricular end-diastolic volume index (LVEDVi), left atrioventricular ratio (LA:LV), and exercise capacity (VO_2 peak) in the healthy cohort ($n = 1600$).

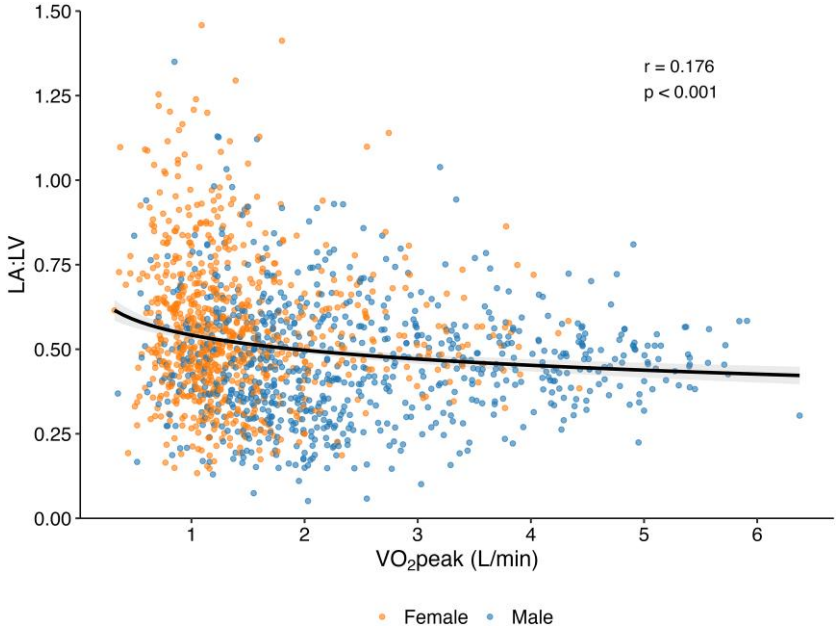


Figure 6 Association between left atrioventricular ratio and exercise capacity in the healthy cohort. Log-transformed scatterplot demonstrating the inverse relationship between LA:LV and exercise capacity (VO_2 peak) in the healthy cohort ($n = 1600$). Colours represent LA:LV percentiles.

capacity.[26,28] However, considering the commonality of LA enlargement in endurance athletes, largely without clinical consequences, it appears that LA volume is not the standalone driving mechanism behind an athlete's AF risk.[29] Historically, it has been suggested that extreme endurance exercise may confer a cardioprotective effect against AF in females.[26] However, more recent evidence challenges this notion, suggesting that while female athletes may experience a lower relative risk of AF compared with their male counterparts, the risk is

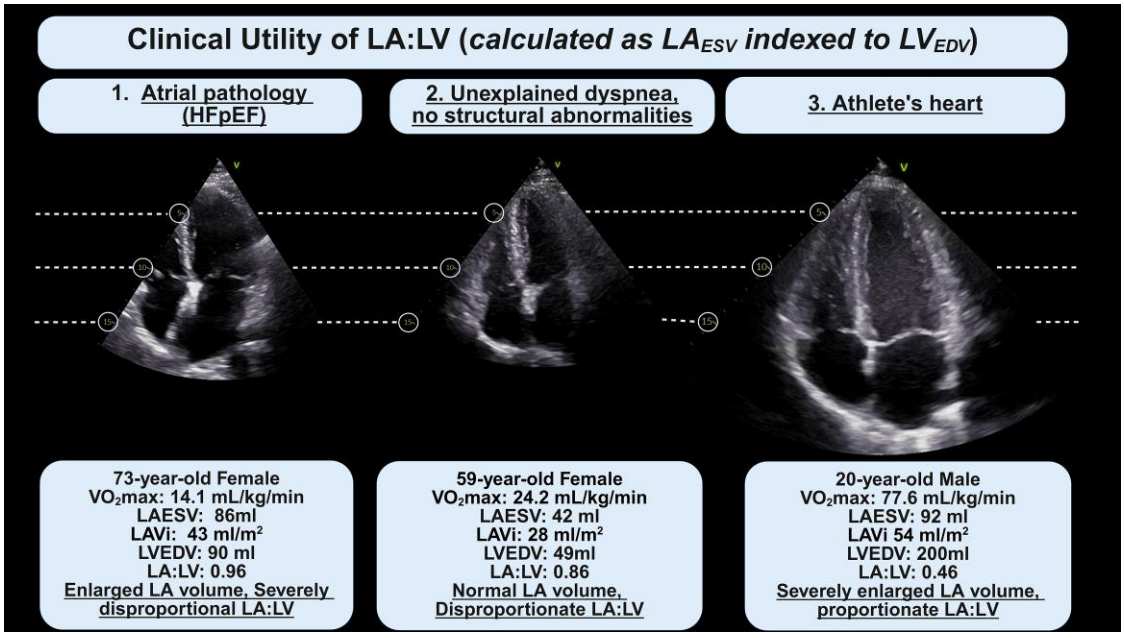


Figure 7 Clinical application of the left atrioventricular ratio in differentiating healthy cardiac adaptations and pathology. Representative echocardiographic 4-chamber views demonstrating three clinical scenarios where the LA:LV provides diagnostic value in distinguishing healthy adaptations from pathology.

nonetheless elevated compared with the general population.[30,31] Thus, our findings suggest that the low LA:LV of athletes indicates the adaptive nature of LA remodelling and increased LA:LV in both sexes could be suggestive of maladaptive changes.

Abnormal LA:LV despite 'normal' LA and LV

Extensive literature links the association between chamber enlargement, both in the LA and LV, with cardiovascular events, including incident HF.[5–7] However, recent studies suggest that a small LV may contribute to diminished cardiac reserve and unexplained dyspnoea in some patients.[32,33] This oversimplification of pathophysiology, where large or small chamber volumes are viewed as inherently pathological, neglects the mechanistic context of LA and LV volume relationship. The importance of chamber proportionality is supported by evidence that hydraulic forces during diastole, generated by cross-sectional area differences between the LA and LV, facilitate LV filling.[34,35] This provides a compelling physiological basis for why the relationship between LA and LV sizes, whether quantified as area differences or volume ratios, may be more clinically relevant than their absolute sizes. In the absence of confounding pathology (i.e. mitral valve disease or AF being notable exceptions), LA and LV volumes are moderately collinear. However, the LV is strongly associated with VO_2peak, whereas the LA has a moderate association. Therefore, an individual with normal LA and LV volumes as compared to normal reference values, could still present with an elevated LA:LV, which could indicate compensatory LA hypertrophy, LV atrophy, or both.

LA:LV and exercise capacity

Our study found that after corrections for age, sex and anthropometry, LA:LV is not a strong predictor of VO_2 peak. This is expected due to the collinearity between LA:LV, age, sex and exercise capacity, highlighting the functionality of LA:LV as a measurement. In the absence of valve pathology or AF, there is a tendency for symmetrical remodelling, where LA:LV remains within a narrow range. However, the variation in exercise capacity was substantial, with a 10-fold or greater difference among the cohort. Although younger, fitter individuals were more likely to have a low LA:LV and, conversely, the older individual with lower exercise capacity is expected to have a higher LA:LV, the association is insufficiently strong for LA:LV to be used as a stand-alone predictor of exercise capacity. This relationship remained consistent across genders, illustrating the utility of the LA:LV as a simple, sex-agnostic assessment of cardiac physiology.

LA:LV vs. Alternative Left Atrial Parameters

We calculated the LA:LV using LAESV indexed to LVEDV, an approach recently validated in healthy individuals.[12] This approach captures maximal chamber volumes at different phases of the cardiac cycle, offering a comprehensive assessment of chamber enlargement with prognostic utility for exercise capacity. In contrast, left atrial coupling index (LACI) has been used in some prior studies, with LA end-diastolic volume (LAEDV) indexed to LVEDV,[36,37] measured at the same cardiac cycle timepoint of mitral valve closure. However, other work on left atrial volumetric/mechanical coupling index uses the same LACI terminology but defines it as LAVi indexed to tissue Doppler velocity at atrial contraction (LAVi/TDI-a′).[16] This distinction emphasizes the need for standardized measurements and common terminology that balance theoretical precision with clinical accessibility. While it may seem logical to use volumes measured at the same phase of the cardiac cycle, LAESV is more readily available in clinical practice, making LA:LV a more practical tool for everyday use. Minimal LA volume measures also tend to have greater variability, which may introduce imprecision in derived ratios.[38] Additionally, both LA and LV strain have gained recognition as markers of cardiac function, offering insight into the pressure-volume

relationship throughout the cardiac cycle. While LA strain demonstrates predictive value for exercise capacity in clinical populations,[39] it correlates poorly with exercise capacity in healthy individuals.[21] The same findings are observed in LV strain, with elite athletes presenting with mildly lower strain values than untrained controls.[40] The combination of structural volume ratios alongside strain could have clinical utility and should be explored.

Clinical implications and potential applications

The simplicity of LA:LV makes it suitable for incorporation into echocardiographic reporting templates and clinical decision-making algorithms, streamlining cardiovascular risk assessment. The LA:LV offers significant clinical utility in the following settings (*Figure 7*):

(1) **Athletic Heart vs. Pathology:** In athletes, LA enlargement is a common healthy adaptation to prolonged endurance training. Our findings demonstrate that despite having larger LA volumes, athletes typically exhibit low LA:LV, supporting the notion of physiological remodeling. Elevated LA:LV in the absence of high exercise capacity may prompt further investigation into underlying pathology.

(2) **Heart Failure with Preserved Ejection Fraction or Atrial Fibrillation:** Both HFpEF and AF are frequently associated with disproportionate LA enlargement due to diastolic dysfunction and elevated filling pressures. An elevated LA:LV in these populations could aid in risk stratification, as well as disease monitoring and management.

(3) **Unexplained Dyspnea and Borderline LA Enlargement:** In cases where LA size is mildly elevated but clinical significance is uncertain, LA:LV can provide additional context, helping clinicians determine whether the enlargement is compensatory or indicative of early dysfunction.

(4) **Pathological LA remodeling:** Underlying pathology is associated with pathological LA remodeling, providing further utility for LA:LV in detecting early signs of pathology and guiding clinical decision-making.

Limitations

Our study sample was specifically designed to capture a broad spectrum of exercise capacity and extremes of chamber remodelling. The study's observational nature limits causal inference. The analysis, based on cross-sectional data, revealed variability in LA:LV across all VO_2 peak quartiles. Long-term studies are needed to assess the consequences of a higher LA:LV to determine whether it represents maladaptive remodelling and increased HF risk. While the inclusion of both healthy and pathological populations strengthen the findings, potential selection bias exists, particularly with the overrepresentation of athletes. Additionally, the differences between centres for posture and phenotype in both TTE and CPET introduced collinearity, preventing adjustment for these variables in our models and potentially affecting absolute measurements. Although our study highlights the diagnostic potential of the LA:LV, its prognostic utility, especially in predicting AF, HF hospitalization, or mortality, remains to be established.

Conclusions

The LA:LV is a simple, accessible, and physiologically grounded tool for distinguishing between adaptive from maladaptive cardiac remodelling. By contextualizing LA enlargement relative to LV size, it provides valuable insights into cardiac function and exercise capacity that standalone measurements may overlook. In clinical practice, the LA:LV ratio could enhance risk stratification, particularly in populations where LA enlargement is ambiguous or confounded by physiological adaptation. Easily obtainable from routine echocardiography, the LA:LV has broad applicability in both athletic and clinical cardiology. This study identifies a normal LA:LV as ~0.5 and LA:LV values ≥0.75 indicate disproportional remodelling and warrant consideration of atrial pathology. Future studies should explore its prognostic value and potential as a therapeutic or monitoring tool in cardiovascular disease management.

Supplementary data

Supplementary data are available at *European Heart Journal - Cardiovascular Imaging* online.

Author contributions

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Funding

This work was supported by a project grants from the National Health and Medical Research Council of Australia (NHMRC; APP1130353). L.S. and K.J. supported by an Australian Government Research Training Program Scholarship. A.M. is supported by a NHMRC postgraduate scholarship (IGNT2030942). P.D. is supported by a Royal Australian College of Physicians Research Entry Scholarship (2023RES00039), a National Health and Medical Research Council Postgraduate Scholarship (I2031119) and a National Heart Foundation of Australia PhD Scholarship (I107659). A.L.G. is supported by a National Health and Medical Research Council of Australia Investigator Grant (Level 1, APP2027105).

Conflict of interest: None declared.

Data availability

The data underlying this article could be shared on reasonable request.

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