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ASSOCIATION BETWEEN LEFT ATRIAL STRAIN AND LEFT ATRIAL VOLUME IN OVERWEIGHT AND OBESE ADULTS: A CROSS-SECTIONAL SPECKLE-TRACKING ECHOCARDIOGRAPHIC STUDY

[1]Dr Avinash Perumal Vijayarangam, [2*]Dr Sharada. K, [3]Vasundhara. G

[1]Post Graduate, Department of General medicine, Saveetha Medical College and Saveetha Institute of Medical and Technical Sciences, Saveetha University, Saveetha Nagar, Thandalam, Chennai Bengaluru, Nh 48, Chennai, Tamilnadu 602105, India, ORCID ID: 0009-0005-3664-6393, email ID- avinashviji96@gmail.com
[2*]Professor, Department of Cardiology, Saveetha Medical College and Saveetha Institute of Medical and Technical Sciences, Saveetha University, Saveetha Nagar, Thandalam, Chennai Bengaluru, Nh 48, Chennai, Tamilnadu 602105, India, email ID- sharadadrive@gmail.com
[3]Department of allied health sciences- cardiovascular technology, Saveetha Medical College and Saveetha Institute of Medical and Technical Sciences, Saveetha University, Saveetha Nagar, Thandalam, Chennai Bengaluru, Nh 48, Chennai, Tamilnadu 602105, India gopibhavni@gmail.com
*Corresponding Author:Dr Sharada. K, Email ID: sharadadrive@gmail.com, avinashviji96@gmail.com

ABSTRACT

Background: Maximum left atrial (LA) volume indexes structural remodelling in obesity, whereas LA reservoir strain (LASr) measured by speckle-tracking echocardiography indexes atrial function. Whether the two markers move together across the spectrum of elevated body mass index (BMI) is unsettled.

Objectives: To compare biplane LA volume and phasic LA strain across BMI strata, and to test whether strain and volume are associated with each other and with BMI independently of age, sex and comorbidity.

Materials and Methods: Seventy-five adults with BMI ≥25 kg/m² and preserved left ventricular ejection fraction underwent transthoracic echocardiography at a tertiary centre. Maximum LA volume (LAVmax) and reservoir, conduit and contractile strain were obtained from apical four-chamber, two-chamber and biplane measurements. Participants were grouped as BMI 25.0–30.0 (n = 15), >30.0–35.0 (n = 20) and >35.0 kg/m² (n = 40), and compared using analysis of variance or the Kruskal–Wallis test, Spearman correlation and multivariable linear regression.

Results: Mean age was 56.7 ± 9.4 years; 49 participants (65.3%) were men. Biplane LASr fell progressively across BMI groups (median 20.0%, 16.0% and 13.0%; Kruskal–Wallis $p < 0.001$; p for trend < 0.001), whereas biplane LAVmax did not differ (39.5, 38.5 and 40.3 mL; $p = 0.355$). BMI correlated inversely with LASr ($\rho = -0.496$, 95% confidence interval [CI] −0.658 to −0.291; $p < 0.001$) but not with LAVmax ($\rho = 0.179$; $p = 0.125$). LAVmax and LASr were only weakly related ($\rho = -0.225$; $p = 0.053$), and this attenuated after adjustment for BMI (partial $r = -0.168$; $p = 0.150$). On multivariable regression BMI was the only independent predictor of LASr (B = −0.504% per kg/m², 95% CI −0.766 to −0.242; $p < 0.001$; adjusted $R^2 = 0.187$); no variable predicted LAVmax (model $p = 0.610$). Impaired LASr (<22%) affected 60 participants (80.0%), LAVmax >32 mL affected 63 (84.0%).

Conclusion: In overweight and obese adults with preserved ejection fraction, LA reservoir strain deteriorated as BMI rose, independently of age, sex and comorbidity, whereas LA volume showed no BMI gradient and was not significantly associated with strain. Atrial function tracks adiposity more closely than chamber size does; the two should not be treated as interchangeable.

KEYWORDS: Body mass index, echocardiography, left atrial strain, left atrial volume, obesity, speckle-tracking echocardiography

INTRODUCTION

Excess adiposity has become one of the defining chronic conditions of contemporary clinical practice. The World Heart Federation and the World Obesity Federation have described the obesity epidemic as a global public health crisis that contributes to more than 2.8 million deaths every year, acting on the cardiovascular system through haemodynamic, metabolic, inflammatory and neurohumoral pathways that are only partly captured by conventional risk scoring.[1] The problem is not confined to high-income settings. The nationally representative ICMR-INDIAB survey estimated the weighted prevalence of generalised obesity in Indian adults at 28.6% and of abdominal obesity at 39.5%, with hypertension present in 35.5% and diabetes in 11.4%; the burden is heaviest in urban populations and in the southern states.[2] Among the cardiac consequences of adiposity, atrial arrhythmia is one of the earliest and most consequential: in the Framingham cohort each additional unit of body mass index (BMI) raised the adjusted risk of new-onset atrial fibrillation by roughly 4% to 5%.[3]

The left atrium sits at the interface between ventricular filling pressure and pulmonary venous return, and it responds to chronic volume and pressure loading by dilating. Left atrial (LA) enlargement is therefore treated as a barometer of cumulative diastolic burden and is an established predictor of atrial fibrillation, stroke, incident heart failure and

cardiovascular death.[6,7] In the population-based MONICA/KORA study, which followed 1212 adults for ten years, obesity emerged as the single most powerful determinant of left atrial enlargement, outweighing hypertension.[5] The mechanistic picture has since been refined. Obese patients with heart failure and preserved ejection fraction display greater plasma volume expansion, more biventricular remodelling, increased epicardial fat and greater pericardial restraint than their non-obese counterparts, a constellation now recognised as a distinct obese phenotype.[4] Adiposity, in other words, does not simply accompany atrial disease; it appears to generate it.

Chamber size, however, is a late and relatively coarse marker. Two-dimensional speckle-tracking echocardiography permits direct measurement of atrial deformation through the three phases of the atrial cycle — reservoir, conduit and contractile — and reservoir strain in particular has emerged as a sensitive, load-dependent index of atrial myocardial function and left ventricular filling pressure.[8] A meta-analysis of 40 studies places normal reservoir strain at approximately 39%, with contractile strain near 17%.[9] Crucially, strain abnormalities can be demonstrated in hypertensive and diabetic patients whose atrial dimensions remain entirely normal,[10] which suggests that functional impairment may precede the structural change that volumetric indices detect. In the community-based Asklepios study of 1531 middle-aged adults, reservoir strain declined stepwise from 35.3% in those with normal weight to 30.9% in those with obesity, and conduit strain fell in parallel, differences that persisted after adjustment for age, sex and other confounders.[11]

What remains unsettled is whether the structural and the functional marker behave concordantly once the population is restricted to people who are already overweight or obese. Most of the supporting literature contrasts obese participants with normal-weight controls, so the wide dynamic range of BMI carries much of the statistical signal; far fewer studies have asked how strain and volume co-vary within an obese cohort, and fewer still have done so in Indian patients, in whom cardiometabolic risk clusters at a lower absolute BMI than in western populations. A further methodological gap concerns the apical window from which deformation is measured. Reservoir strain derived from the four-chamber view, the two-chamber view and the biplane average are frequently reported interchangeably, yet the atrium is not geometrically symmetrical about the two planes, and the extent of disagreement between them has rarely been quantified in an obese cohort in whom acoustic access is poor. The distinction matters clinically, because if strain and volume diverge then a normal LA volume cannot be taken as reassurance that the atrium is functioning normally, and a patient in whom weight reduction would be most valuable may be discharged without further evaluation. The present study was therefore designed with three aims: to quantify biplane LA maximum volume and phasic LA strain across three strata of elevated BMI; to test the association between structural and functional markers, and of each with BMI, after accounting for age, sex, diabetes, hypertension and coronary artery disease; and to compare the values obtained from the two apical windows against the biplane average.

MATERIALS AND METHODS

Study design and setting

This was a single-centre, hospital-based, cross-sectional observational study conducted in the Department of General Medicine of a tertiary care teaching hospital in Chennai, Tamil Nadu, India, over a six-month period. Consecutive adults with an elevated body mass index who were referred for transthoracic echocardiography from inpatient and outpatient services were considered for enrolment.

Participants and eligibility

Adults aged 18 to 90 years with a body mass index of 25 kg/m² or above and preserved left ventricular systolic function were eligible. Patients were excluded if the acoustic window was inadequate for reliable endocardial border tracking, if significant valvular heart disease was present, if they were pregnant, or if they were known to have hypothyroidism. Seventy-nine patients were screened; four were excluded because the case record was incomplete or internally inconsistent, leaving 75 participants for analysis (Figure 1). Every participant gave written informed consent.

Sample size

No formal a priori sample size calculation was performed; the sample comprised all eligible patients presenting during the enrolment window. A post hoc computation indicated that 75 participants afford 80% power at a two-sided alpha of 0.05 to detect a correlation coefficient of magnitude 0.32 or greater.

Data collection and variables

Demographic details, anthropometry, pulse rate and comorbid conditions were recorded on a structured proforma designed for the study. Hypertension, diabetes mellitus and coronary artery disease were taken as present if documented in the medical record or if the patient was receiving specific treatment. Body mass index was calculated as weight in kilograms divided by the square of height in metres, and participants were stratified into three groups: 25.0 to 30.0 kg/m², above 30.0 to 35.0 kg/m², and above 35.0 kg/m². Body surface area was derived using the Mosteller formula. All entries were independently checked by two investigators; discrepancies were referred to a senior cardiologist. Records were additionally screened for physiologically implausible values and for internal inconsistency before analysis.

Echocardiography

Transthoracic echocardiography was performed with the patient in the left lateral decubitus position. Linear left ventricular dimensions, ejection fraction, fractional shortening, end-diastolic and end-systolic volumes and left ventricular mass were obtained according to the recommendations of the American Society of Echocardiography and the European Association of Cardiovascular Imaging.[12] Maximum left atrial volume (LAVmax) was measured at end-systole, immediately before

mitral valve opening, from dedicated non-foreshortened apical four-chamber and two-chamber views, and the biplane value was derived from both planes.[12]

Left atrial deformation imaging

Left atrial deformation was assessed by two-dimensional speckle-tracking echocardiography from the apical four-chamber and two-chamber views, with the region of interest traced along the atrial endocardium and adjusted to exclude the appendage and the pulmonary veins. Zero strain was set at ventricular end-diastole, using the R wave of the QRS complex as the fiducial point, in keeping with the EACVI/ASE consensus on standardisation of atrial deformation imaging.[13] Reservoir strain (LASr), conduit strain (LAScd) and contractile strain (LASct) were recorded separately for each apical view and as the biplane average. Conduit and contractile strain are reported as negative values, consistent with the sign convention of the analysis software. Left ventricular diastolic function was assessed qualitatively as part of the routine study.[14] An LASr below 22% was taken as impaired on the basis of published lower limits of normal,[9] and an LAVmax above 32 mL was taken as indicating left atrial dilatation.

Statistical analysis

Analyses were performed with IBM SPSS Statistics version 25 (IBM Corporation, Armonk, New York, USA), with confirmatory computation in Python 3 (SciPy 1.17 and statsmodels 0.14). The distribution of each continuous variable was examined with the Shapiro–Wilk test overall and within each BMI group, and homogeneity of variance with the Levene test. Normally distributed variables are summarised as mean ± standard deviation and non-normally distributed variables as median with interquartile range; categorical variables are given as counts and percentages. Three-group comparisons used one-way analysis of variance with Tukey honestly significant difference post hoc testing where the parametric assumptions were satisfied, and the Kruskal–Wallis test with Dunn post hoc testing and Bonferroni adjustment where they were not. Effect sizes are reported as eta-squared or epsilon-squared. Linear trend across the ordered BMI groups was assessed by ordinary least squares regression on the group rank, and for binary outcomes by the Cochran–Armitage test. Categorical variables were compared with the Pearson chi-square test, or with the Fisher–Freeman–Halton exact test estimated by Monte Carlo simulation (20 000 replicates) where an expected cell count fell below five. Correlations were quantified with the Pearson coefficient when both variables were normally distributed and with the Spearman rank coefficient otherwise; 95% confidence intervals were derived by the Fisher z transformation. Partial correlation was used to adjust the strain–volume relationship for BMI and age. Agreement between the apical four-chamber, apical two-chamber and biplane measurements of each parameter was tested by the Friedman test, with repeated-measures analysis of variance as a parametric sensitivity analysis and pairwise Wilcoxon signed-rank tests with Bonferroni adjustment. Two multivariable linear regression models were fitted, the first with biplane LASr and the second with biplane LAVmax as the dependent variable, each with BMI, age, sex, diabetes, hypertension and coronary artery disease as independent variables. Unstandardised coefficients with 95% confidence intervals, standardised coefficients, adjusted R^2 and variance inflation factors are reported. Because the residuals of the strain model departed from normality, heteroscedasticity-consistent (HC3) standard errors and bias-corrected bootstrap confidence intervals from 5000 resamples were computed as sensitivity analyses. A two-sided p value below 0.05 was considered statistically significant. Smoking and alcohol use, although listed on the proforma, were not captured in the analysable dataset and could not be modelled.

Ethical considerations

The study protocol was approved by the Institutional Ethics Committee of Saveetha Medical College and Hospital, Saveetha Institute of Medical and Technical Sciences. The study was conducted in accordance with the Declaration of Helsinki, and written informed consent was obtained from every participant.

RESULTS

Data validation

Of the 79 patients screened, four were excluded for incomplete or ambiguous case records, and 75 entered the analysis (Figure 1). Data completeness was high. Ejection fraction was not recorded for one participant. Two strain values were physiologically implausible, being positive where a negative deformation is obligatory — one biplane conduit strain and one apical four-chamber contractile strain — and both were set to missing rather than imputed, so that analyses of those two variables rest on 74 observations. No other outlier exceeded the range that is plausible for this population, and no participant was excluded on the basis of an extreme value.

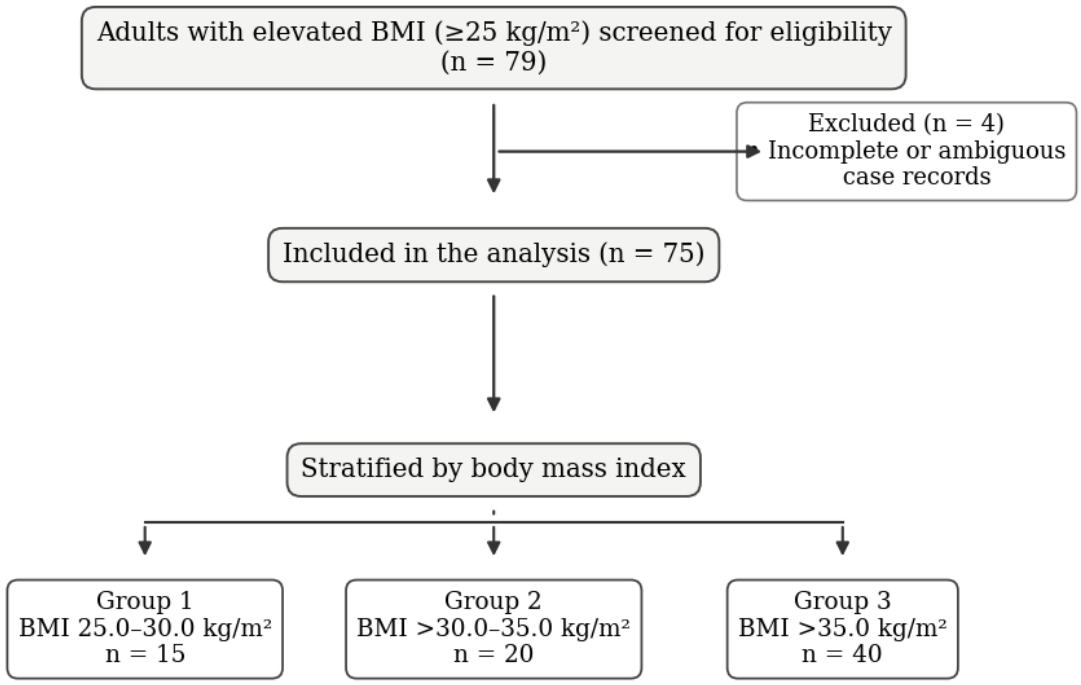

Figure 1: Flow of participants through the study.

Baseline characteristics

The mean age of the cohort was 56.7 ± 9.4 years (range 34 to 79) and 49 participants (65.3%) were men. Median BMI was 35.3 kg/m² (interquartile range 31.3 to 39.3), with 15 participants (20.0%) in the 25.0 to 30.0 kg/m² stratum, 20 (26.7%) in the >30.0 to 35.0 kg/m² stratum and 40 (53.3%) above 35.0 kg/m². Coronary artery disease was the commonest comorbidity, documented in 37 participants (49.3%), followed by diabetes mellitus in 33 (44.0%) and hypertension in 20 (26.7%). Left ventricular ejection fraction was preserved throughout, with a median of 60% (58 to 60.8). Baseline characteristics by BMI group are set out in Table 1. Participants in the lowest BMI stratum were on average nine years younger than the other two groups (49.2 ± 9.2 versus 59.4 ± 7.0 and 58.2 ± 9.3 years; p = 0.002), and, as expected, weight, body surface area and BMI itself rose across the strata. The distribution of sex, hypertension, diabetes and coronary artery disease did not differ between groups (all p > 0.34). Neither ejection fraction (p = 0.937), left atrial anteroposterior diameter (p = 0.948), end-systolic volume (p = 0.710) nor left ventricular mass (p = 0.067) differed significantly across BMI strata.

Table 1: Baseline demographic, clinical and echocardiographic characteristics of the study population, overall and by body mass index group (n = 75)

Variable	All (n = 75)	BMI 25.0–30.0 (n = 15)	BMI >30.0–35.0 (n = 20)	BMI >35.0 (n = 40)	p value
Age (years)*	56.7 ± 9.4	49.2 ± 9.2	59.4 ± 7.0	58.2 ± 9.3	0.002†
Male sex, n (%)	49 (65.3)	8 (53.3)	12 (60.0)	29 (72.5)	0.348‡
Pulse rate (beats/min)*	84.3 ± 8.1	88.0 ± 7.3	85.3 ± 9.2	82.4 ± 7.4	0.060†
Height (cm)§	155 (150–159)	158 (154.5–163)	157 (152–159)	150 (147–157)	<0.001¶
Weight (kg)§	79 (73–88)	65 (58.5–70)	79.5 (78–83)	88 (79–90)	<0.001¶
Body mass index (kg/m²)§	35.3 (31.3–39.3)	28.2 (26.2–29.0)	32.9 (31.5–33.8)	39.2 (36.4–40.3)	<0.001¶
Body surface area (m²)§	1.84 (1.74–1.94)	1.68 (1.61–1.77)	1.86 (1.82–1.91)	1.91 (1.79–1.98)	<0.001¶
Hypertension, n (%)	20 (26.7)	5 (33.3)	5 (25.0)	10 (25.0)	0.882#

Variable	All (n = 75)	BMI 25.0–30.0 (n = 15)	BMI >30.0–35.0 (n = 20)	BMI >35.0 (n = 40)	p value
Diabetes mellitus, n (%)	33 (44.0)	5 (33.3)	9 (45.0)	19 (47.5)	0.638‡
Coronary artery disease, n (%)	37 (49.3)	9 (60.0)	11 (55.0)	17 (42.5)	0.430‡
Ejection fraction (%)§**	60 (58–60.8)	60 (58.3–60)	59.5 (58–61.3)	60 (58–60)	0.937¶
LA anteroposterior diameter (cm)§	4.40 (4.20–4.65)	4.40 (4.25–4.60)	4.35 (4.17–4.70)	4.40 (4.20–4.62)	0.948†
LV end-diastolic volume (mL)§	90 (70–100)	99 (89–125)	77 (62.5–104)	93 (76–99)	0.096¶
LV end-systolic volume (mL)§	30 (21–36)	32 (23–37.5)	24 (19.8–38.5)	32 (23–35)	0.710¶
LV mass (g)*	229.6 ± 62.6	261.7 ± 64.6	229.0 ± 67.4	217.8 ± 56.4	0.067†

BMI: Body mass index; LA: Left atrium; LV: Left ventricle. *Mean ± standard deviation. §Median (interquartile range). †One-way analysis of variance. ¶Kruskal–Wallis test. ‡Pearson chi-square test. #Fisher–Freeman–Halton exact test (Monte Carlo, 20 000 replicates). **n = 74.

Left atrial volume and strain across body mass index groups

Biplane LA reservoir strain declined progressively as BMI rose (Table 2, Figure 2a). Median LASr was 20.0% (17.0 to 24.0) in the 25.0 to 30.0 kg/m² group, 16.0% (14.0 to 19.3) in the >30.0 to 35.0 kg/m² group and 13.0% (12.0 to 16.3) in the >35.0 kg/m² group (Kruskal–Wallis H = 16.07, p < 0.001; epsilon-squared 0.195; p for linear trend < 0.001). Dunn post hoc testing with Bonferroni adjustment localised the difference to the comparison between the lowest and the highest BMI stratum (p < 0.001); the adjacent comparisons did not reach significance (25.0–30.0 versus >30.0–35.0 kg/m², p = 0.249; >30.0–35.0 versus >35.0 kg/m², p = 0.100).

Biplane LA maximum volume, in contrast, showed no significant gradient (Table 2, Figure 2b). Median LAVmax was 39.5 mL (30.3 to 49.0), 38.5 mL (36.3 to 47.5) and 40.3 mL (36.3 to 52.3) in the three groups respectively (Kruskal–Wallis H = 2.07, p = 0.355; p for trend = 0.155). The same was true when volume was indexed to body surface area (medians 22.5, 20.7 and 21.2 mL/m²; p = 0.978). Neither biplane conduit strain (p = 0.585) nor biplane contractile strain (p = 0.135) differed significantly across BMI strata, although contractile strain was numerically less negative in the leanest group. Single-plane measurements were similarly unrevealing: apical two-chamber reservoir strain, conduit strain, contractile strain and volume all failed to separate the groups, and the apparent difference in apical four-chamber reservoir strain (p = 0.021) was non-monotonic, driven by an isolated dip in the intermediate stratum, and is unlikely to be meaningful.

Table 2: Left atrial volume and phasic left atrial strain across body mass index groups

Parameter	BMI 25.0–30.0 (n = 15)	BMI >30.0–35.0 (n = 20)	BMI >35.0 (n = 40)	Test statistic	p value	p for trend
Biplane LASr (%)	20.0 (17.0–24.0)	16.0 (14.0–19.3)	13.0 (12.0–16.3)	H = 16.07	<0.001	<0.001
Biplane LAScd (%)†	−11.0 (−16.5 to −8.5)	−11.5 (−15.0 to −5.0)	−9.0 (−12.5 to −6.0)	H = 1.07	0.585	0.288
Biplane LASct (%)	−12.0 (−14.0 to −8.0)	−14.5 (−17.5 to −11.8)	−13.0 (−16.0 to −11.8)	H = 4.00	0.135	0.091
Biplane LAVmax (mL)	39.5 (30.3–49.0)	38.5 (36.3–47.5)	40.3 (36.3–52.3)	H = 2.07	0.355	0.155
Biplane LAVmax index (mL/m²)	22.5 (17.4–29.3)	20.7 (19.6–26.4)	21.2 (19.1–28.6)	H = 0.05	0.978	0.982
A4C LASr (%)	19.0 (17.0–21.0)	17.0 (13.5–19.0)	19.5 (18.0–21.0)	H = 7.72	0.021	0.208

Parameter	BMI 25.0–30.0 (n = 15)	BMI >30.0–35.0 (n = 20)	BMI >35.0 (n = 40)	Test statistic	p value	p for trend
A4C LAScd (%)	−9.0 (−14.0 to −8.0)	−11.5 (−15.5 to −6.0)	−10.0 (−13.5 to −6.0)	H = 0.26	0.878	0.852
A4C LASct (%)*‡	−8.9 ± 5.3	−12.6 ± 6.4	−13.0 ± 6.0	F = 2.59	0.082	0.045
A4C LAVmax (mL)*	42.5 ± 9.4	44.8 ± 9.5	41.4 ± 8.3	F = 1.01	0.368	0.457
A2C LASr (%)	26.0 (13.0–30.0)	26.0 (20.0–27.3)	23.0 (20.0–27.3)	H = 0.15	0.928	0.561
A2C LAScd (%)	−11.0 (−19.0 to −6.0)	−8.5 (−11.0 to −4.5)	−8.0 (−12.0 to −6.0)	H = 3.34	0.188	0.093
A2C LASct (%)	−14.0 (−16.0 to −3.5)	−15.5 (−18.3 to −13.0)	−14.5 (−19.3 to −13.0)	H = 4.03	0.133	0.028
A2C LAVmax (mL)*	36.1 ± 15.6	35.3 ± 14.2	37.0 ± 13.1	F = 0.10	0.903	0.770

Data are median (interquartile range) unless marked *, in which case mean ± standard deviation. A2C: Apical two-chamber; A4C: Apical four-chamber; BMI: Body mass index; LAScd: Left atrial conduit strain; LASct: Left atrial contractile strain; LASr: Left atrial reservoir strain; LAVmax: Maximum left atrial volume. H: Kruskal–Wallis statistic; F: One-way analysis of variance statistic. †n = 74 (one implausible positive value excluded). ‡n = 74 (one implausible positive value excluded); group n = 14, 20, 40. Post hoc for biplane LASr (Dunn, Bonferroni-adjusted): 25.0–30.0 vs >35.0 kg/m², p < 0.001; 25.0–30.0 vs >30.0–35.0 kg/m², p = 0.249; >30.0–35.0 vs >35.0 kg/m², p = 0.100. Epsilon-squared for biplane LASr = 0.195.

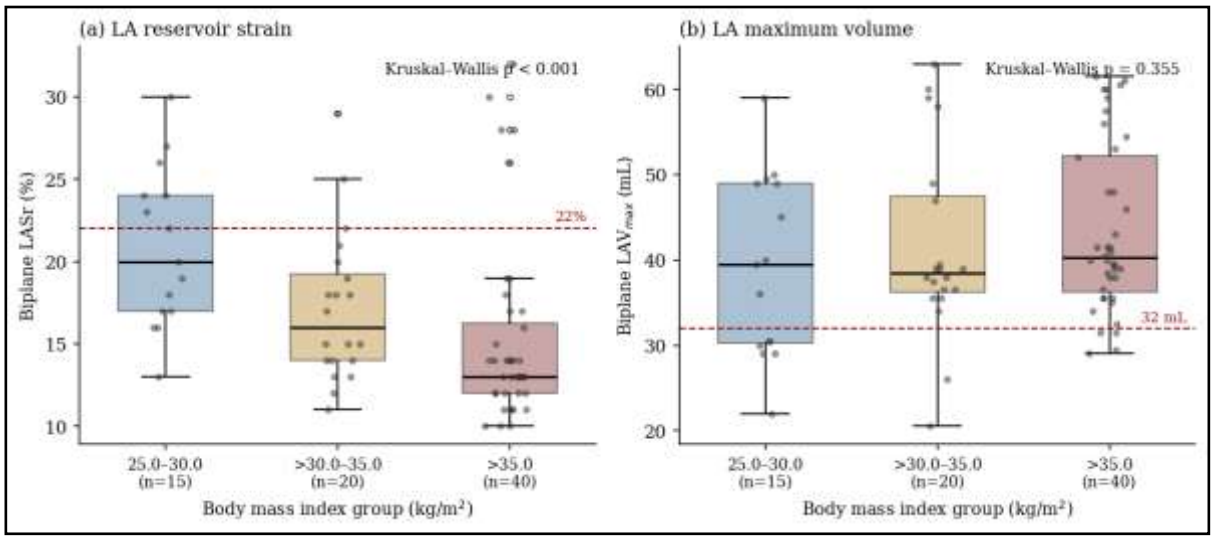

Figure 2: Distribution of (a) biplane left atrial reservoir strain and (b) biplane maximum left atrial volume across body mass index groups. Boxes show the median and interquartile range; whiskers extend to 1.5 times the interquartile range; individual observations are overlaid. Dashed lines mark the abnormality thresholds of 22% for reservoir strain and 32 mL for maximum volume.

Correlation and partial correlation

Correlation analysis reinforced this dissociation (Table 3, Figure 3). BMI was inversely and moderately correlated with biplane LASr (Spearman ρ = −0.496, 95% CI −0.658 to −0.291; p < 0.001) but showed no significant relationship with biplane LAVmax (ρ = 0.179, 95% CI −0.064 to 0.402; p = 0.125), with volume indexed to body surface area (ρ = 0.042; p = 0.719) or with LA anteroposterior diameter (ρ = 0.046; p = 0.696). Neither conduit nor contractile strain was related to BMI (p = 0.567 and p = 0.570). The relationship between LAVmax and LASr was weak, inverse and did not attain statistical significance (ρ = −0.225, 95% CI −0.441 to 0.016; p = 0.053); it weakened further once BMI was held constant (partial r = −0.168, 95% CI −0.382 to 0.063; p = 0.150) and after additional adjustment for age (partial r = −0.166; p =

0.155). The inverse association between BMI and LASr, by contrast, survived adjustment for age (partial r = −0.409, 95% CI −0.583 to −0.199; p < 0.001). Biplane LASr was weakly and positively correlated with left ventricular mass (ρ = 0.260; p = 0.024); no other correlation reached significance.

Table 3: Correlation of body mass index and left atrial volume with left atrial phasic strain and other echocardiographic variables (n = 75)

Variable pair	Coefficient (ρ)	95% CI	p value
BMI vs biplane LASr	−0.496	−0.658 to −0.291	<0.001
BMI vs biplane LAVmax	0.179	−0.064 to 0.402	0.125
BMI vs biplane LAVmax index	0.042	−0.200 to 0.279	0.719
BMI vs biplane LAScd†	0.068	−0.177 to 0.304	0.567
BMI vs biplane LASct	−0.067	−0.302 to 0.176	0.570
BMI vs LA anteroposterior diameter	0.046	−0.196 to 0.283	0.696
BMI vs A4C LASr	0.190	−0.053 to 0.411	0.103
BMI vs A2C LASr	0.027	−0.215 to 0.265	0.821
Biplane LAVmax vs biplane LASr	−0.225	−0.441 to 0.016	0.053
Biplane LAVmax vs biplane LAScd†	−0.062	−0.299 to 0.182	0.598
Biplane LAVmax vs biplane LASct	0.007	−0.234 to 0.246	0.956
Biplane LAVmax vs LA anteroposterior diameter	−0.029	−0.268 to 0.212	0.802
Age vs biplane LASr	0.055	−0.187 to 0.292	0.637
Age vs biplane LAVmax	0.047	−0.195 to 0.284	0.688
Biplane LASr vs LV mass	0.260	0.021 to 0.471	0.024
Biplane LASr vs ejection fraction	0.188	−0.056 to 0.411	0.108
Biplane LAVmax vs LV mass	0.192	−0.051 to 0.413	0.099
Partial: biplane LAVmax vs LASr, adjusted for BMI	−0.168	−0.382 to 0.063	0.150
Partial: biplane LAVmax vs LASr, adjusted for BMI and age	−0.166	−0.381 to 0.067	0.155
Partial: BMI vs biplane LASr, adjusted for age	−0.409	−0.583 to −0.199	<0.001

Spearman rank correlation coefficients with Fisher z-transformed 95% confidence intervals; the three partial correlations are Pearson coefficients computed on regression residuals. A2C: Apical two-chamber; A4C: Apical four-chamber; BMI: Body mass index; CI: Confidence interval; LA: Left atrium; LAScd: Left atrial conduit strain; LASct: Left atrial contractile strain; LASr: Left atrial reservoir strain; LAVmax: Maximum left atrial volume; LV: Left ventricle. †n = 74.

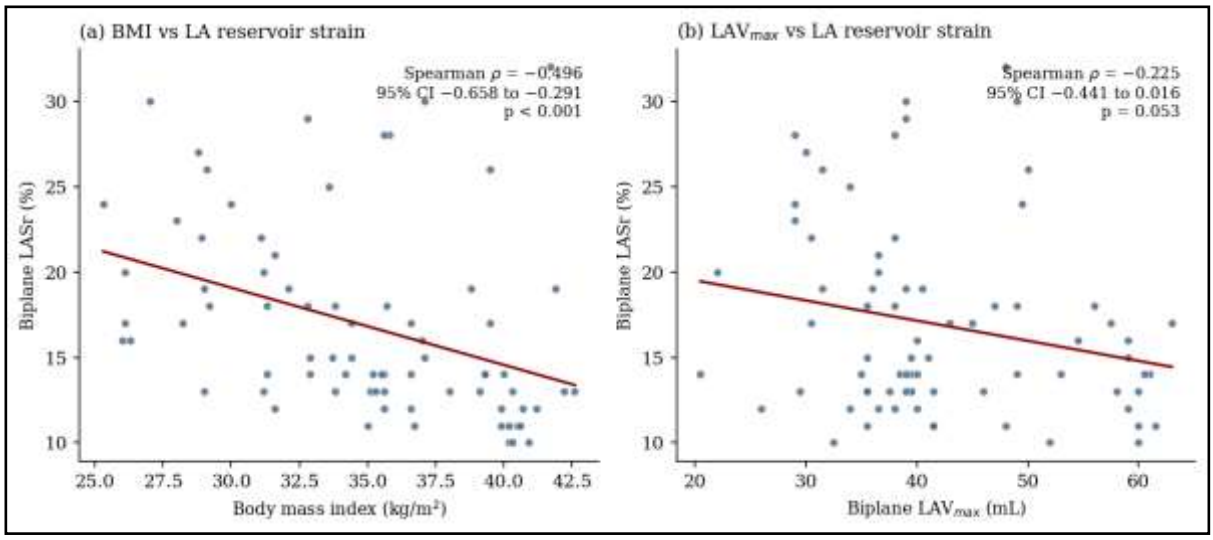

Figure 3: Scatterplots of (a) body mass index against biplane left atrial reservoir strain and (b) biplane maximum left atrial volume against biplane left atrial reservoir strain, with least-squares regression lines.

View-wise comparison

Values obtained from the two apical windows were not interchangeable (Table 4). Reservoir strain was systematically higher in the apical two-chamber view than in the apical four-chamber view (median 23.0% versus 19.0%) and lowest for the biplane average (15.0%); the Friedman test was highly significant ($\chi^2 = 42.09$, $p < 0.001$), as was repeated-measures analysis of variance ($F [2, 148] = 20.29$, $p < 0.001$). Pairwise testing confirmed that the two single-plane values differed from one another ($p < 0.001$) and that the two-chamber value differed from the biplane value ($p < 0.001$), whereas the four-chamber and biplane values did not ($p = 0.102$). Contractile strain likewise varied by view (Friedman $\chi^2 = 25.41$, $p < 0.001$), as did LAVmax ($\chi^2 = 8.48$, $p = 0.014$), the two-chamber volume being some 8 mL lower than the four-chamber volume ($p = 0.006$). Conduit strain did not differ significantly across views on the Friedman test ($p = 0.356$), although the parametric sensitivity analysis reached significance ($p = 0.007$), a discrepancy consistent with the marked skew of that variable.

Table 4: Comparison of left atrial strain and volume measured in the apical four-chamber view, the apical two-chamber view and as the biplane average

Parameter	A4C	A2C	Biplane	Friedman χ^2	p value
LASr (%), n = 75	19.0 (17.0–21.0)	23.0 (20.0–28.0)	15.0 (13.0–19.0)	42.09	<0.001
LAScd (%), n = 74	−10.0 (−14.8 to −6.3)	−8.0 (−13.0 to −6.0)	−10.0 (−13.0 to −6.0)	2.07	0.356
LASct (%), n = 74	−13.0 (−15.0 to −8.0)	−15.0 (−18.0 to −13.0)	−13.0 (−16.0 to −11.0)	25.41	<0.001
LAVmax (mL), n = 75	42.0 (37.0–49.0)	34.0 (26.0–45.0)	39.5 (35.5–49.0)	8.48	0.014

Data are median (interquartile range). Bonferroni-adjusted Wilcoxon signed-rank post hoc results — LASr: A4C vs A2C $p < 0.001$, A2C vs biplane $p < 0.001$, A4C vs biplane $p = 0.102$; LASct: A4C vs A2C $p = 0.012$, A4C vs biplane $p = 0.017$, A2C vs biplane $p = 0.055$; LAVmax: A4C vs A2C $p = 0.006$, A2C vs biplane $p = 0.002$, A4C vs biplane $p = 0.989$. Repeated-measures analysis of variance gave $F (2, 148) = 20.29$, $p < 0.001$ for LASr; $F (2, 146) = 5.18$, $p = 0.007$ for LAScd; $F (2, 146) = 4.75$, $p = 0.010$ for LASct; and $F (2, 148) = 9.80$, $p < 0.001$ for LAVmax. A2C: Apical two-chamber; A4C: Apical four-chamber; LAScd: Left atrial conduit strain; LASct: Left atrial contractile strain; LASr: Left atrial reservoir strain; LAVmax: Maximum left atrial volume.

Subgroup analyses

Subgroup analyses are presented in Table 5. Neither biplane LASr nor biplane LAVmax differed significantly by sex, diabetes, hypertension or coronary artery disease (all $p \geq 0.080$). The largest of these non-significant differences was between men and women, men having a lower reservoir strain (16.0 ± 4.9% versus 18.6 ± 6.5%; Cohen $d = −0.47$; $p = 0.080$). Conduit and contractile strain were likewise unrelated to any comorbidity.

Table 5: Subgroup comparison of biplane left atrial reservoir strain and maximum volume by sex and comorbidity

Subgroup	n	Biplane LASr (%)	p value	Biplane LAVmax (mL)	p value
Male	49	16.0 ± 4.9	0.080	43.2 ± 10.7	0.370
Female	26	18.6 ± 6.5		40.2 ± 9.8	
Diabetes present	33	17.0 ± 5.6	0.748	43.6 ± 11.3	0.180
Diabetes absent	42	16.8 ± 5.7		41.0 ± 9.6	
Hypertension present	20	17.3 ± 5.8	0.773	42.2 ± 10.5	0.986
Hypertension absent	55	16.8 ± 5.6		42.2 ± 10.5	
Coronary artery disease present	37	16.2 ± 4.6	0.663	40.7 ± 10.1	0.205
Coronary artery disease absent	38	17.6 ± 6.4		43.6 ± 10.6	

Data are mean ± standard deviation; p values from the Mann–Whitney U test (both variables non-normally distributed). Cohen d for the sex difference in LASr was −0.47. LASr: Left atrial reservoir strain; LAVmax: Maximum left atrial volume.

Multivariable regression

In the multivariable model for biplane LA reservoir strain (Table 6), the six predictors together explained a modest but significant fraction of the variance ($F [6, 68] = 3.84$, $p = 0.002$; $R^2 = 0.253$; adjusted $R^2 = 0.187$). BMI was the dominant and only robust independent predictor: each 1 kg/m² increment was associated with a 0.504 percentage-point fall in reservoir strain (95% CI −0.766 to −0.242; standardised $\beta = -0.424$; $p < 0.001$). Male sex was associated with a 2.73 percentage-point lower strain (95% CI −5.272 to −0.187; $p = 0.036$), but this coefficient was not stable under heteroscedasticity-consistent standard errors ($p = 0.093$) or bootstrap resampling (95% CI −5.665 to 0.237) and should be regarded as provisional. Age, diabetes, hypertension and coronary artery disease contributed nothing (all $p \geq 0.080$). Multicollinearity was absent (maximum variance inflation factor 1.23; all tolerances above 0.81). The BMI coefficient was unchanged by the sensitivity analyses (HC3 95% CI −0.744 to −0.264; bootstrap 95% CI −0.741 to −0.277) and by the further addition of LAVmax to the model, in which volume itself remained non-significant (B = −0.083 per mL, 95% CI −0.198 to 0.032; $p = 0.153$).

The corresponding model for biplane LA maximum volume explained essentially none of the variance and was not significant overall ($F [6, 68] = 0.75$, $p = 0.610$; adjusted $R^2 = -0.021$). No independent variable approached significance, BMI included (B = 0.320 mL per kg/m², 95% CI −0.227 to 0.866; $p = 0.247$) (Table 7). In unadjusted models the same asymmetry was evident: BMI alone accounted for 14.5% of the variance in reservoir strain (B = −0.452; $p < 0.001$) but only 3.1% of the variance in LA volume (B = 0.390; $p = 0.131$).

Table 6: Multivariable linear regression with biplane left atrial reservoir strain as the dependent variable (n = 75)

Predictor	B	95% CI	Standardised β	p value	VIF	Tolerance
Body mass index (per 1 kg/m²)	−0.504	−0.766 to −0.242	−0.424	<0.001	1.11	0.901
Age (per year)	0.109	−0.023 to 0.242	0.185	0.103	1.14	0.877
Male sex	−2.729	−5.272 to −0.187	−0.234	0.036	1.09	0.917
Diabetes mellitus	0.237	−2.359 to 2.834	0.021	0.856	1.23	0.813
Hypertension	0.605	−2.229 to 3.438	0.048	0.672	1.16	0.862
Coronary artery disease	−2.234	−4.747 to 0.278	−0.201	0.080	1.17	0.855
Constant	30.864	19.731 to 41.998	—	<0.001	—	—

Model F (6, 68) = 3.84, p = 0.002; R^2 = 0.253; adjusted R^2 = 0.187. Breusch–Pagan p = 0.070; Durbin–Watson 1.34. Because the residuals departed from normality (Shapiro–Wilk p < 0.001), sensitivity analyses were performed: with HC3 robust standard errors the BMI coefficient was −0.504 (95% CI −0.744 to −0.264; p < 0.001) and male sex was no longer significant (p = 0.093); bootstrap 95% confidence intervals (5000 resamples) were −0.741 to −0.277 for BMI and −5.665 to 0.237 for male sex. Adding biplane LAVmax to the model left BMI unchanged (B = −0.477; p < 0.001) while LAVmax itself was non-significant (B = −0.083 per mL, 95% CI −0.198 to 0.032; p = 0.153; adjusted R^2 = 0.200). B: Unstandardised coefficient; CI: Confidence interval; VIF: Variance inflation factor.

Table 7: Multivariable linear regression with biplane maximum left atrial volume as the dependent variable (n = 75)

Predictor	B	95% CI	Standardised β	p value	VIF	Tolerance
Body mass index (per 1 kg/m²)	0.320	−0.227 to 0.866	0.144	0.247	1.11	0.901
Age (per year)	−0.051	−0.327 to 0.225	−0.046	0.712	1.14	0.877
Male sex	2.416	−2.891 to 7.724	0.111	0.367	1.09	0.917
Diabetes mellitus	1.904	−3.516 to 7.324	0.091	0.486	1.23	0.813
Hypertension	−0.979	−6.893 to 4.935	−0.042	0.742	1.16	0.862
Coronary artery disease	−1.555	−6.799 to 3.689	−0.075	0.556	1.17	0.855
Constant	32.543	9.302 to 55.783	—	0.007	—	—

Model F (6, 68) = 0.75, p = 0.610; R^2 = 0.062; adjusted R^2 = −0.021. Breusch–Pagan p = 0.445; Durbin–Watson 1.36. HC3 robust standard errors did not alter any inference (BMI p = 0.270). In the unadjusted model, BMI explained 3.1% of the variance in LAVmax (B = 0.390, 95% CI −0.118 to 0.899; p = 0.131). B: Unstandardised coefficient; CI: Confidence interval; VIF: Variance inflation factor.

Prevalence of abnormal atrial indices

Applying conventional thresholds, impaired reservoir strain (<22%) was present in 60 participants (80.0%) and left atrial dilatation (LAVmax >32 mL) in 63 (84.0%); 54 participants (72.0%) met both criteria, 6 (8.0%) had impaired strain with a normal volume, 9 (12.0%) had a dilated atrium with preserved strain, and 6 (8.0%) had neither abnormality. The prevalence of impaired strain rose across BMI strata, from 8 of 15 (53.3%) to 17 of 20 (85.0%) to 35 of 40 (87.5%) (chi-square p = 0.015; Fisher–Freeman–Halton exact p = 0.019; Cochran–Armitage z = 2.56, p for trend = 0.010), as did the prevalence of left atrial dilatation, from 9 of 15 (60.0%) to 18 of 20 (90.0%) to 36 of 40 (90.0%) (exact p = 0.022; p for trend = 0.017) (Figure 4). A more stringent strain cut-off of 18% classified 48 participants (64.0%) as abnormal.

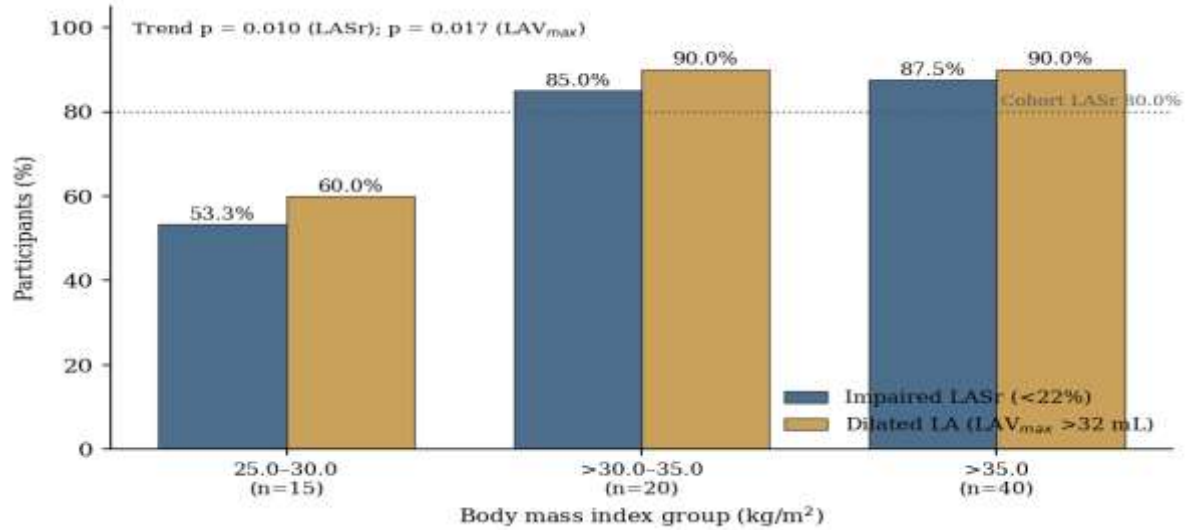

Figure 4: Prevalence of impaired left atrial reservoir strain (<22%) and of left atrial dilatation (maximum volume >32 mL) by body mass index group.

DISCUSSION

This cross-sectional study of 75 overweight and obese adults with preserved ejection fraction produced a single, clean and somewhat unexpected result: left atrial reservoir strain fell steadily as body mass index rose, whereas left atrial maximum volume did not. The strain gradient was steep, monotonic and independent of age, sex, diabetes, hypertension and coronary artery disease, with each unit of BMI costing roughly half a percentage point of reservoir strain. The volume gradient was absent by every test applied — across groups, in correlation, in unadjusted regression and in adjusted regression, and whether volume was expressed in absolute terms or indexed to body surface area. Correspondingly, the association between the two atrial markers themselves was weak, inverse and formally non-significant, and it attenuated further once BMI was taken into account. On these data the null hypothesis of no significant association between left atrial strain and left atrial volume cannot be rejected.

The strain finding sits comfortably within the existing literature. In the Asklepios study of 1531 community-dwelling adults aged 35 to 55, reservoir strain declined from 35.3% in participants of normal weight to 33.1% in the overweight and 30.9% in the obese, and conduit strain fell from 21.6% to 16.7%, differences that persisted after multivariable adjustment.[11] Our gradient is steeper in relative terms, which is unsurprising: the Asklepios cohort was a decade younger, largely free of coronary disease and included a normal-weight reference group, whereas half of our participants carried a diagnosis of coronary artery disease and 44% had diabetes. The absolute values also differ substantially. Normal reservoir strain approximates 39% in meta-analysis[9] and global peak atrial longitudinal strain was 42.2 ± 6.1% in the original reference series of healthy volunteers,[15] against a median of 15% here. Some of that gap is real — our cohort was old, obese and comorbid — but a substantial part almost certainly reflects vendor and software differences in atrial deformation analysis, which are well recognised and are the reason the EACVI/ASE task force insisted on explicit reporting of the fiducial point and the tracking algorithm.[13] Absolute strain values from single-centre studies should therefore be compared with caution, whereas within-study gradients such as ours are considerably more robust.

The volume finding requires more careful handling, because it appears at first sight to contradict a large and consistent body of evidence. In the MONICA/KORA cohort obesity was the strongest predictor of incident left atrial enlargement over ten years, with an odds ratio of 2.4.[5] Left atrial size increases with BMI even in children,[17] obesity and overweight are associated with impaired left ventricular diastolic function in elderly community cohorts,[18] and surgically induced weight loss prevents the progressive rise in left atrial volume that is otherwise observed over three to four years.[16] A recent cardiac magnetic resonance study likewise found BMI to be an independent correlate of left atrial volume.[20] The reconciliation lies in the range of exposure. Every one of those studies contrasted obese participants with lean or normal-weight comparators; the relationship between BMI and left atrial volume is largely carried by that contrast. Our cohort begins at a BMI of 25.3 kg/m², so the leanest participant here would have been classified as overweight in each of those studies. Restriction of range attenuates correlation, and it did so here. There was, moreover, a ceiling effect: 84% of participants already had an LAVmax above 32 mL and 60% of even the lowest BMI stratum met that criterion, leaving little room for volume to discriminate. The prevalence data make the same point from the opposite direction — left atrial dilatation did increase across BMI strata as a dichotomous outcome (p for trend 0.017) even though the continuous measurement did not.

Indexing deserves separate comment. Standard practice is to normalise left atrial volume to body surface area,[12] yet body surface area itself rises with adiposity, so indexing systematically deflates the apparent atrial size of exactly the patients in whom enlargement matters most. We reported absolute volume as the primary measure for this reason and examined the indexed value as a secondary analysis; neither showed a BMI gradient, so the choice of denominator does not explain our result. It does, however, explain why studies that index to body surface area often report weaker obesity–volume associations than studies that index to height.

Why should function deteriorate while size does not? The most parsimonious explanation is that the two markers report on different stages of the same process. Reservoir strain integrates atrial compliance, atrial contractile reserve and left ventricular longitudinal shortening, and is sensitive to myocardial changes that precede chamber dilatation. Mondillo and colleagues demonstrated precisely this in hypertensive and diabetic patients whose atrial volumes were entirely normal but whose strain was already abnormal,[10] and the state-of-the-art review by Thomas and co-workers positions reservoir strain as an earlier and more discriminating marker of diastolic dysfunction than volume.[8] In obesity the mechanisms are now reasonably well delineated. Epicardial and peri-atrial fat infiltrates the atrial myocardium and generates conduction slowing, voltage reduction and fractionation on electroanatomical mapping, changes proportional to the volume of adjacent fat and independent of chamber size.[19] Obese patients with heart failure and preserved ejection fraction show greater pericardial restraint and plasma volume expansion, both of which load the atrium and stiffen it.[4] Fibrosis, inflammation and insulin resistance complete the picture. A stiff, fibrotic, fat-infiltrated atrium may deform poorly long before it dilates measurably, and once dilatation is established — as it was in most of our patients — volume ceases to track further functional decline. Left atrial volume remains a powerful prognostic marker of cumulative diastolic burden,[6,7] but it is a marker of accumulated damage, not of current function.

A secondary but practically important observation concerns the apical window. Reservoir strain was consistently higher in the two-chamber view than in the four-chamber view (median 23.0% versus 19.0%; $p < 0.001$), and left atrial volume was some 8 mL lower in the two-chamber view ($p = 0.006$). The direction of the strain difference exactly reproduces the observation of Cameli and colleagues, who found two-chamber peak atrial longitudinal strain of 44.3 ± 6.0% against 40.1 ± 7.9% in the four-chamber view in healthy volunteers.[15] Because these single-plane values are not interchangeable, and because the biplane average behaved differently from either component in our data, studies that report atrial strain without specifying the view cannot be pooled meaningfully. This is exactly the concern that motivated the standardisation consensus,[13] and our data support its recommendation that the view and the averaging method be stated explicitly.

Neither diabetes, hypertension nor coronary artery disease was independently associated with reservoir strain or with atrial volume in this cohort, and no subgroup difference reached significance. This should not be read as evidence of absence. The comorbidity prevalences were high but the subgroups were small, and the confidence intervals around each coefficient are wide enough to admit clinically meaningful effects. What the models do establish is that the BMI–strain relationship is not an artefact of confounding by these conditions: the coefficient barely moved on adjustment, and multicollinearity was negligible. The suggestion that men had lower reservoir strain than women is consistent with published sex differences but was not stable under robust or bootstrap inference here, and we regard it as hypothesis-generating.

The strengths of this work lie in its analytical discipline rather than in its scale. Every participant contributed a complete set of nine deformation measurements and three volumes, obtained from both apical windows, so that the view-wise comparison rests on paired data rather than on between-group inference. Distributional assumptions were tested rather than assumed, and non-parametric methods were used wherever they were violated, which matters because reservoir strain and atrial volume were both markedly skewed in this cohort. Physiologically impossible values were removed rather than retained or silently imputed. The regression coefficients were subjected to heteroscedasticity-consistent and bootstrap inference, which is what allowed us to distinguish the robust BMI effect from the fragile sex effect; a conventional analysis reporting only ordinary least squares p values would have declared both significant. Non-significant results are reported in full, including the primary volume comparison, and the trend tests and effect sizes are given alongside the p values so that the reader can judge the magnitude of what was and was not found. These choices matter in a literature where atrial strain values vary by vendor and where selective reporting of the significant comparison is a real risk.

The clinical implication is straightforward. In an overweight or obese patient with a preserved ejection fraction, a left atrial volume within normal limits does not exclude atrial dysfunction: six of our participants (8.0%) had impaired reservoir strain despite a normal atrial volume, and, since 80% of the cohort had a reservoir strain below 22%, subclinical atrial impairment was close to being the rule rather than the exception. Given that obesity predicts atrial fibrillation[3] and that reservoir strain predicts incident heart failure and atrial arrhythmia, adding strain to the routine study in this population is cheap, quick and likely to identify patients in whom weight reduction — which reverses atrial enlargement after bariatric surgery[16] — would have the greatest yield. Whether strain improvement follows weight loss in this specific population, and whether it translates into fewer arrhythmic events, will require longitudinal study.

Limitations

Several constraints qualify these findings. The design is cross-sectional and single-centre, so temporality and causation cannot be inferred and referral bias is possible. Enrolment was restricted to patients with a BMI of at least 25 kg/m², and the absence of a normal-weight comparator both restricts the range of the exposure and probably explains the null volume result; a ceiling effect compounded this, since 84% of participants already had an enlarged atrium. Diastolic Doppler indices, natriuretic peptides and epicardial fat thickness were not available, so patients could not be formally classified for heart failure with preserved ejection fraction. Strain was acquired on a single vendor platform without inter- or intra-observer reproducibility testing. Smoking and alcohol use were listed on the proforma but not captured in the analysable dataset and so could not be modelled. Recorded BMI diverged by more than 1 kg/m² from the value calculable from height and weight in 14 records, which raises the possibility of transcription error in the anthropometric fields. Finally, with 75 participants the study had only 49% power to detect a correlation of the magnitude observed between volume and strain, so a small true association cannot be excluded.

CONCLUSION

Among overweight and obese adults with preserved left ventricular ejection fraction, left atrial reservoir strain declined progressively and independently with rising body mass index, whereas left atrial maximum volume showed no such gradient and was not significantly associated with strain. Each additional unit of BMI cost approximately half a percentage point of reservoir strain after adjustment for age, sex and cardiometabolic comorbidity, while no measured variable predicted atrial volume. Subclinical atrial dysfunction was near-ubiquitous, affecting four in five participants. In this population, atrial function and atrial size convey different information and should not be treated as substitutes for one another; speckle-tracking assessment of reservoir strain adds clinically relevant information that volumetric measurement alone does not provide, and merits inclusion in the routine echocardiographic evaluation of the obese patient.

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CONFLICTS OF INTEREST

There are no conflicts of interest.

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