Obesity-induced left atrial dysfunction across age groups and cardiovascular conditions: a comprehensive review of diagnostic advances and clinical implications

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Abstract: Obesity is a global health concern, affecting nearly half of the world's population and significantly increasing cardiovascular disease (CVD) risk. Among various cardiac impacts, left atrial (LA) dysfunction is a notable complication, especially given its association with atrial fibrillation (AF), heart failure with preserved ejection fraction (HFpEF), and diastolic dysfunction. This comprehensive review synthesizes findings from studies across age groups, from children to adults, evaluating obesity-induced alterations in LA structure and function. Using advanced diagnostic tools like speckle tracking echocardiography, we examine the impact of obesity on LA function, the potential reversibility of LA dysfunction, and implications for CVD progression. Studies show that elevated body mass index (BMI) correlates with LA enlargement, increased stiffness, and impaired reservoir and conduit strain, with specific changes observed across different cardiovascular conditions. In children, obesity-related insulin resistance is associated with increased LA stiffness, marking early indicators of metabolic and cardiac dysfunction. In adults, higher BMI independently reduces LA strain, compromising function and raising the likelihood of AF recurrence post-cardioversion or percutaneous interventions. Additionally, clinical manifestations such as exercise intolerance in obese HFpEF patients highlight LA stiffness as a predictor of poorer quality of life and reduced physical capacity. Importantly, weight loss interventions, including bariatric surgery, show promise in reversing LA dysfunction, suggesting a potential for reducing obesity-related cardiac risks. Our review underscores the value of incorporating LA strain metrics in routine cardiac assessments to identify subclinical changes early and guide preventive strategies in obese patients. Further research into therapeutic approaches targeting LA function is essential for reducing HFpEF incidence and improving cardiovascular outcomes in obesity.

Keywords: Obesity; body mass index (BMI); left atrial dysfunction (LA dysfunction); cardiovascular disease (CVD); heart failure with preserved ejection fraction (HFpEF)

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Introduction

Globally, obesity presents a significant health concern. The Global Burden of Disease (GBD) Obesity Collaborators estimated that 39% to 49% of the world's population (2.8–3.5 billion people) are overweight or obese (1). In 2015, high body mass index (BMI) accounted for 4.0 million deaths more than two-thirds of which were caused by cardiovascular disease (CVD) (1). It was also found that both BMI-related deaths (41%) and BMI-related disability-adjusted life-years (34%) were caused by CVD among individuals with obesity (1). Obesity correlates significantly with cardiovascular disorders such as atrial fibrillation (AF), heart failure with preserved ejection fraction (HFpEF), and diastolic dysfunction (2). The ongoing Framingham Heart Study, initiated in 1948, has shown that obesity increases the likelihood of developing new-onset AF by 50% (3). Notably, for every 5-unit rise in BMI, the risk of AF increases by 30% (3).

Indeed, obesity significantly affects the function of the heart's left atrium. The application of techniques such as speckle tracking echocardiography (STE) has yielded key findings on the impact of obesity on left atrial (LA) function in various populations. This review synthesizes findings from studies examining the association between obesity and LA function in individuals of all age groups, including children, adults, and those with heart pathologies such as AF and HFpEF (*Table 1*).

Methodology

This review aimed to synthesize current evidence on the association between obesity and LA structural and functional remodeling across pediatric and adult populations, with a focus on the pathophysiological mechanisms, clinical relevance, and potential reversibility through lifestyle and surgical interventions.

Search strategy

A comprehensive literature search was conducted using PubMed, Embase, and Google Scholar databases from January 2000 to December 2023. The search strategy included a combination of Medical Subject Headings (MeSH) and keywords such as: "obesity", "adiposity", "left atrial function", "left atrial strain", "atrial remodeling", "cardiac MRI", "echocardiography", "speckle tracking", "bariatric surgery", "pediatrics", "adolescents", "heart failure", and "atrial fibrillation". Reference lists of selected studies and relevant reviews were manually searched to identify additional eligible articles.

Inclusion and exclusion criteria

Studies were included if they met the following criteria:

- Evaluated LA size, LA function, or left atrial strain (LAS) in relation to obesity or weight change.
- Employed cardiac imaging techniques such as transthoracic echocardiography, STE, or cardiac magnetic resonance imaging (MRI).
- Included human subjects (pediatric or adult populations).
- Published in English in peer-reviewed journals.

Exclusion criteria included:

- Case reports, editorials, and narrative commentaries without original data.
- Animal studies or *in vitro* experiments.
- Studies without measurable LA outcomes.

Data extraction and synthesis

Relevant data were extracted from the included studies and organized thematically according to the following categories:

(I) Pathophysiological mechanisms linking obesity to atrial remodeling (including hemodynamic, metabolic, and neurohormonal pathways).
(II) Evidence in pediatric and adolescent populations.
(III) Adult population studies evaluating LAS, stiffness, and morphology in the context of BMI and central adiposity.
(IV) Reversibility of LA dysfunction following bariatric surgery or structured weight loss programs.
(V) Clinical implications for cardiovascular risk stratification and prevention of AF.

Table 1 Key studies on the impact of obesity on left atrial function

Study	Population	Key findings	Conclusion
Pucci *et al.*, 2024 (4)	Adults with non-valvular AF	Higher BMI linked to lower PALS	Obesity exacerbates LA dysfunction in AF
Mishima *et al.*, 2022 (5)	AF patients	CRF associated with better LA function	CRF may counteract obesity's impact on LA function
Cichoń *et al.*, 2019 (6)	Obese AF patients post-PVI	Lower LAS before and after PVI	LA dysfunction persists after PVI in obese patients
Larios *et al.*, 2024 (7)	Adolescents with severe obesity	Reduced LA and LV strain	Early myocardial impairment in obese adolescents
Aristizábal-Duque *et al.*, 2023 (8)	Rural children and adolescents	Reduced ventricular strain, no significant LA effect	Ventricular dysfunction precedes LA impairment in childhood obesity
Steele *et al.*, 2020 (9)	Adolescents with T2DM	Reduced LAS despite normal LA volumes	Obesity and T2DM lead to early diastolic dysfunction
Aga *et al.*, 2024 (10)	Obese adults	LAV/h^2 better identifies LA dysfunction than LAV/BSA	LAV/h^2 should be preferred for obese patients
Aga *et al*., 2024 (11)	Obese adults post-bariatric surgery	LA reservoir strain improved post-surgery	Weight loss may reverse LA dysfunction
Romero Dorta *et al.*, 2023 (12)	Women with elevated BMI	Progressive decline in LAS and diastolic function over time	Obesity worsens cardiac function over the long term
Singleton *et al.*, 2022 (13)	Older obese HFpEF patients	Higher LA stiffness linked to reduced exercise tolerance	LA stiffness is a therapeutic target in HFpEF
Alonso Gómez *et al.*, 2022 (14)	Obese patients with metabolic syndrome	LAS improves diastolic dysfunction classification	LAS enhances diagnostic accuracy
Chirinos *et al.*, 2019 (15)	Middle-aged adults (35–55 years)	Higher BMI linked to reduced LA reservoir and conduit strain; compensatory increase in booster function	Early subclinical LA dysfunction in obesity, potential heart failure risk
Mahfouz *et al.*, 2015 (16)	Obese children	Higher LA stiffness associated with insulin resistance	LA stiffness is a potential early marker for metabolic dysfunction in obese children

AF, atrial fibrillation; BMI, body mass index; CRF, cardiorespiratory fitness; HFpEF, heart failure with preserved ejection fraction; LA, left atrium; LAS, left atrial strain; LAV/h^2, left atrial volume indexed to height^2; LAV/BSA, left atrial volume indexed to body surface area; LV, left ventricle; PALS, peak atrial longitudinal strain; PVI, percutaneous pulmonary vein isolation; T2DM, type 2 diabetes mellitus.

Effects of obesity on the LA function in individuals with AF

Research has shown that LA enlargement often precedes AF (17), with BMI emerging as a strong predictor of LA size (2). Obesity-related factors such as elevated plasma volume, ventricular diastolic dysfunction, and increased neurohormonal activity may contribute to LA enlargement and electrical instability (18-20). Additionally, recent evidence links adiposity to direct myocardial alterations, possibly due to oxidative stress or lipoapoptosis (21,22) (*Figure 1*).

Extracardiac influences, including autonomic dysfunction and sleep apnea, may also elevate atrial arrhythmogenicity in obese individuals. For example, Kanagala *et al.* associated obstructive sleep apnea with increased AF recurrence post-cardioversion, hypothesizing that factors like hypoxemia, elevated afterload, or pulmonary vasoconstriction could be involved (23,24).

Further research underscores a connection between obesity and reduced LA function in AF patients. In a 2024 study, Pucci *et al.* examined 395 adult patients with non-valvular AF, grouping them by BMI (4). Echocardiography results showed that those in the highest BMI tertile

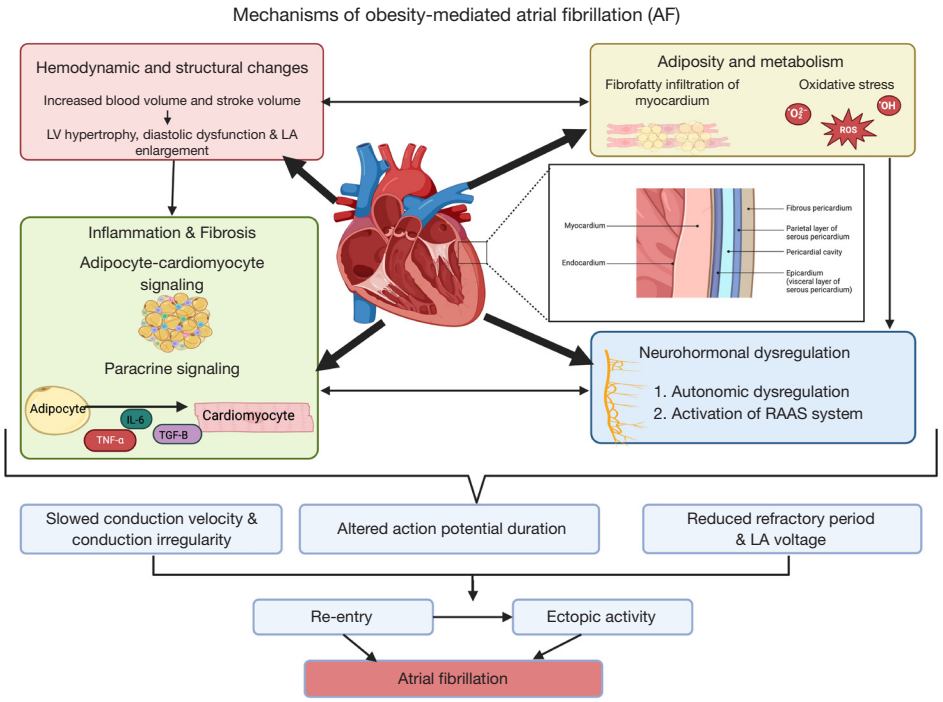

Figure 1 The mechanism of obesity-mediated atrial fibrillation. Created with BioRender.com. LA, left atrium; LV, left ventricle; RAAS, renin-angiotensin-aldosterone system; ROS, reactive oxygen species.

had significantly lower peak atrial longitudinal strain (PALS) compared to individuals with lower BMIs (P<0.002), and multivariate analysis confirmed that BMI was independently linked to reduced PALS, even after adjusting for other risk factors (β=–0.127, P<0.02) (4). These results suggest an association between obesity and impaired LA function in AF patients independently of other cardiovascular risks (4).

The DECAAF study provides valuable context and validation for our findings by demonstrating that atrial fibrosis, assessed through delayed enhancement MRI, is independently associated with AF ablation outcomes (1). DECAAF's identification of hypertension as the only baseline factor significantly associated with atrial fibrosis aligns with our observation that obesity, often comorbid with hypertension, contributes to structural and functional atrial remodeling (1). Importantly, DECAAF highlighted the weak correlation between AF clinical phenotype and degree of fibrosis which suggests that structural atrial abnormalities in obese patients may exist even in early or paroxysmal stages of AF (1).

In related work, Mishima *et al.* (2022) explored the roles of cardiorespiratory fitness (CRF), obesity, and LA function in 154 AF patients (5). This study observed that higher CRF was positively associated with improved LA emptying fraction (LAEF) and better LA reservoir, booster, and conduit strain, suggesting enhanced LA mechanical function and compliance in AF patients. Although obese patients displayed higher indexed LA volumes, obesity was not significantly associated with LA function or stiffness (5), highlighting CRF's potential benefit on LA function regardless of obesity.

Similarly, Cichoń *et al.* (2019) investigated LA function in obese patients undergoing percutaneous pulmonary vein isolation (PVI) for AF (6). Obese individuals exhibited lower LA global longitudinal strain (GLS) both before and after PVI compared to non-obese counterparts (6). indicating that obesity may hinder LA functional recovery even post-intervention, thereby impacting PVI outcomes and emphasizing the importance of obesity management in improving LA function in AF patients (6).

Magnetocardiography (MCG) and delayed enhancement MRI in DECAAF were used to detect atrial dysfunction and fibrosis, respectively. These modalities improve early identification of paroxysmal atrial fibrillation (PAF) and prediction of ablation outcomes, offering better patient stratification than conventional methods like electrocardiogram (ECG) or echocardiography alone (1,2).

Relationship between left atrial stiffness (LASt) and insulin resistance in children with higher body weight

Children with higher body weight and insulin resistance often exhibit increased LASt, likely due to metabolic dysregulation, chronic inflammation, and altered myocardial and vascular function (16). Insulin resistance, in particular, has been associated with chronic low-grade inflammation and increased fibrosis across various tissues, including the heart, contributing to structural and functional changes in the left atrium (16). This metabolic condition may impair myocardial metabolism and function, thereby reducing LA compliance (16). Although the precise mechanisms linking insulin resistance and LASt remain under exploration, contributing factors likely include extracellular matrix protein deposition, oxidative stress, and altered cardiac function.

In 2015, Mahfouz and colleagues investigated the relationship between obesity, LASt, and insulin resistance in a sample of children (16). The study involved 80 obese children (average BMI 28.2±3.1 kg/m^2) and 60 non-obese age-matched controls, assessing both groups for LAS and LASt using tissue Doppler and 2D speckle imaging (16). Findings revealed that obese children had significantly lower LAS (11.3±2.2 *vs.* 38.2±11.6, P<0.001) and higher LASt (1.12±0.23 *vs.* 0.21±0.11, P<0.001) compared to the control group (16). Moreover, a LASt value over 1.0 was found to predict insulin resistance with 92% sensitivity and 86% specificity (P<0.0001), indicating that elevated LASt may serve as a potential early marker of metabolic complications in pediatric obesity (16).

Despite overall preserved left ventricular (LV) function, there was a marked difference in LAS and LASt between obese and non-obese children, with insulin resistance showing a significant association with LASt in the obese group (16). The study underscored that higher LASt and reduced LAS in overweight children could indicate early cardiac changes related to metabolic risk factors. Mahfouz *et al.* (2015) highlighted the importance of utilizing measurements like LAS and LASt as indicators of potential cardiac issues in children, advocating for early detection and intervention in pediatric obesity (16).

Evaluation of LA function in adolescents and young adults affected by obesity

Obesity can lead to early changes in the size and function of the left atrium in young adults, which may be reversible with lifestyle improvements. However, in middle-aged adults, obesity can cause more serious and potentially permanent alterations in LA size and function, heightening the risk of heart rhythm issues and other cardiovascular complications. Although lifestyle modifications can still help middle-aged individuals, their effectiveness may be less pronounced compared to younger adults (7).

Larios *et al.* (2024) studied obesity's impact on the heart's LA chamber in adolescents, finding that obese teens showed strain in both the left atrium and left ventricle (LV), suggesting early signs of heart muscle stress (7). Another 2023 study by Aristizábal-Duque and colleagues examined children and adolescents in certain regions, finding that high BMI was associated with reduced strain in the right and remaining ventricles. However, LAS appeared unaffected, implying that early impacts may primarily affect ventricular function rather than the left atrium in childhood (8).

Additionally, adolescents and young adults with obesity and type 2 diabetes (T2DM) face a higher risk of HFpEF, though early LV diastolic dysfunction in these groups is not yet fully understood. Steele *et al.* (2020) investigated early LA and LV function in 331 individuals across normal-weight, obese, and T2DM groups. Using advanced echocardiographic techniques, they found that both obese and T2DM participants had significantly lower atrial reservoir, conduit, and booster strain compared to normal-weight individuals, despite similar LA volumes. Analysis confirmed these strain changes were linked to diastolic dysfunction markers (9). These results underscore the value of early LAS assessment in detecting LV diastolic dysfunction in young obese and T2DM individuals, highlighting the need for early cardiovascular screening (9). In summary, obesity affects LA function negatively in both young and middle-aged adults, though the severity and likelihood of improvement vary by age group.

Effects of excessive weight on cardiovascular function in middle-aged adults

Chirinos *et al.* (2019) studied the impact of obesity on LA function in 1,531 middle-aged adults (ages 35–55 years) from the Asklepios project, using STE (15). Participants were grouped by BMI: <25 kg/m^2, 25–29.9 kg/m^2, and ≥30 kg/m^2. The study found that LA reservoir and conduit function, measured by strain and strain rate, decreased significantly with higher BMI, indicating early signs of LA impairment in obese individuals (15). Obesity was

linked to reduced passive LA emptying and conduit strain, suggesting lower LA compliance, though an increase in LA booster pump function appeared to help maintain cardiac output as compensation (15). However, in cases of extensive LA dysfunction or AF, this compensatory function might weaken, potentially leading to heart failure in obese patients (15). These findings highlight obesity's early effects on LA function, contributing to CVD risk (15).

Hypertrophy of the left atrium and impaired diastolic performance in obese adults

Obesity can lead to increased pressure in the left atrium due to greater blood volume and pressure, which affects the filling process of the left ventricle. Chronic obesity and conditions like hypertension can also cause stiffening of the LA wall, reducing its ability to fill the ventricle properly during diastole. This may impair LA function, impacting its ability to contract effectively and potentially leading to heart failure symptoms such as shortness of breath, fatigue, and reduced exercise tolerance. If untreated, these issues can progress to more severe heart failure and raise cardiovascular event risk.

A study by Aga *et al.* found that obesity commonly leads to both LA enlargement and dysfunction (10). They highlighted that normalizing LA volume by height squared (LAV/h^2) was more accurate for identifying LA dysfunction than using body surface area (BSA) in obese individuals, suggesting LAV/h^2 as the preferred measurement method for this group (10).

Romero Dorta *et al.* (2023) conducted a seven-year study on the impact of obesity on LA function in women. The study found that overweight or obese women experienced a decline in LA reservoir and conduit strain compared to women with normal weight, underscoring the negative effect of obesity on LA function (12).

Clinical manifestations of atrial dysfunction and HFpEF

Singleton *et al.* (2022) studied how heart dysfunction affects elderly obese patients with HFpEF, finding that higher LASt correlated with reduced exercise capacity, lower peak oxygen consumption (VO_2), and poorer quality of life (13). Exercise intolerance, a key symptom of HFpEF, is linked to high morbidity and mortality. This study compared older obese HFpEF patients to healthy controls, measuring LA function, peak VO_2, 6-minute walk distance, and quality of life. Using MRI cine imaging, they found that HFpEF patients had lower LA reservoir and conduit strain, higher LASt, and lower scores in exercise and quality of life measures compared to controls. LASt emerged as an independent predictor of reduced exercise ability and quality of life, suggesting that targeting LA dysfunction could benefit obese HFpEF patients (13).

In a separate study, Alonso Gómez *et al.* examined LAS's role in evaluating diastolic function in obese patients with metabolic syndrome (14). Current guidelines often classify many of these patients as indeterminate for diastolic dysfunction. Alonso Gómez's team tested whether adding left atrial strain (LALS) to diagnostic criteria could clarify this classification. They assessed 229 patients with echocardiography, analyzing LV and LA strain, plus peak oxygen uptake (VO_2max). By replacing the left atrial volume index (LAVI) threshold with a LA reservoir strain ≤20%, the indeterminate cases dropped from 36% to 23%, while normal classifications rose (14). This adjustment suggests that incorporating LALS could enhance diagnostic accuracy for diastolic dysfunction in overweight and obese patients with metabolic syndrome (14).

Restoration of LA function by weight loss

Aga *et al.* (2024) found that weight loss might improve LA dysfunction related to obesity. In their study, obese patients who had bariatric surgery showed increased LA reservoir strain one-year post-surgery (11). Echocardiograms were conducted on 77 obese participants and 46 matched controls, with the obese group, re-evaluated one year after surgery. Before surgery, the obese patients had significantly lower LA function in reservoir, conduit, and contractile strain than controls. After one year, their LA reservoir strain gradually improved, indicating that LA dysfunction may be reversible (11). Multivariable analysis showed BMI independently reduced all LAS values, suggesting obesity could lead to HFpEF and that bariatric surgery may help address these strain abnormalities (11).

Clinical implication

The growing body of evidence linking obesity to adverse LA remodeling has significant clinical ramifications for cardiovascular prevention, diagnosis, and management. LA dysfunction, particularly impairments in LAS and reservoir function, has emerged as an early and sensitive marker of subclinical myocardial impairment. In both pediatric and

adult populations, obesity-driven LA changes precede overt heart failure or AF, highlighting the atrium's vulnerability to the systemic effects of excess adiposity.

Given that LA dysfunction is associated with an increased risk of AF, HFpEF, and overall cardiovascular morbidity, routine assessment of LA function—particularly using speckle-tracking echocardiography or cardiac MRI—may offer valuable prognostic insight in individuals with obesity. Moreover, the identification of LAS abnormalities in asymptomatic obese individuals may prompt earlier and more aggressive lifestyle and pharmacological interventions aimed at mitigating long-term cardiovascular risk.

Importantly, the observed partial or complete reversibility of LA dysfunction following weight loss—especially after bariatric surgery—supports the notion that LA remodeling is not entirely irreversible. This underscores the potential for cardiac functional recovery through metabolic and hemodynamic improvements. Clinicians should therefore consider LA function not only as a biomarker of disease burden but also as a modifiable therapeutic target in the broader strategy to reduce obesity-related cardiovascular complications.

Incorporating routine LA assessment into the cardiovascular evaluation of individuals with obesity may enhance early detection of subclinical cardiac disease and inform risk stratification and management. Future guidelines may benefit from integrating LAS into cardiovascular screening protocols for at-risk populations.

Limitations

This study has several limitations and contradictions that warrant consideration. The included literature demonstrates variability in study designs, imaging modalities, and population characteristics, limiting comparability across findings. Many studies are cross-sectional, restricting causal inferences between obesity and LA dysfunction. Additionally, the frequent presence of comorbidities such as hypertension and diabetes in obese individuals may confound associations, even when statistical adjustments are made. The lack of standardized methods for assessing LAS and function also contributes to inconsistencies. Contradictory findings exist regarding the reversibility of LA dysfunction following weight loss, and not all studies have found a clear correlation between BMI and LA abnormalities, suggesting a potential role for other metabolic or fat-distribution factors. Furthermore, age and sex may influence outcomes, but current evidence remains inconclusive.

Conclusions

Our review highlights the significant impact of obesity on LA structure and function, illustrating how excess weight can lead to early and potentially reversible LA dysfunction, as well as contribute to increased cardiovascular risk, particularly HFpEF. Findings from recent studies underscore that LAS measurements provide valuable insights into early subclinical changes in cardiac function due to obesity, offering a promising target for early detection and intervention. Furthermore, weight loss, especially through bariatric surgery, appears to improve LA dysfunction, suggesting a possible reversal of adverse structural and functional cardiac changes in obese individuals. Given these findings, integrating LAS metrics into routine cardiac assessments could enhance the identification of at-risk patients, particularly those with obesity and related metabolic conditions. Future research should continue to explore therapeutic strategies targeting LA function in obese populations to reduce the risk of HFpEF and improve overall cardiovascular health.

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