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Regression of Left Ventricular Mass After Bariatric Surgery

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Abstract Notwithstanding the presence of hypertension, obstructive sleep apnea, or both, obesity is associated with increased left ventricular (LV) mass. The effects of bariatric surgery on LV mass have been sparsely investigated by M-mode and two-dimensional (2D) echocardiography. Overall, Roux-en-Y gastric bypass, adjustable gastric banding, and sleeve gastrectomy reduce LV mass. However, the reduction in LV mass is extremely variable. Besides duration and severity of obesity, presence of hypertension, obstructive sleep apnea or both, and type of surgical procedures, the inaccuracy of M-mode and 2D echocardiography for assessment of LV mass contributes to the variable effects of bariatric surgery on LV mass. Three-dimensional (3D) echocardiography may obviate the limitations of M-mode 2D echocardiography for assessment of LV mass and allow an accurate appraisal of the effects of bariatric surgery on LV mass.

Keywords Left ventricular mass · Bariatric surgery · Left ventricular remodeling

Introduction

Obesity-associated left ventricular hypertrophy (LVH) has been extensively reviewed [1, 2, 3•, 4]. At variance with the conventional wisdom that correlates obesity with eccentric LVH, concentric LVH is now thought to be more prevalent than eccentric LVH in obese patients without hypertension or obstructive sleep apnea [5–7]. The effects of bariatric surgery on left ventricular mass (LVM) have been mostly reported after Roux-end-Y gastric bypass (RYGB) or adjustable gastric banding (AGB) surgery [8–13]. The most consistent effect of RYGB and AGB on the left ventricle is a reduction in LVM [12–14]. Reduction of LVM is clinically relevant, as it is associated with a lower cardiovascular risk independent of blood pressure (BP) changes [15–17]. Laparoscopic sleeve gastrectomy (LSG) is currently the most commonly performed surgical procedure for weight loss [18, 19]. It is easier to perform than RYGB and results in greater loss of body weight than laparoscopic AGB (LAGB) [20]. Because LSG has only recently emerged as the preferred bariatric surgical procedure, few reports are available regarding its effects on LV structure and function [21–27].

This review focuses on (1) the phenotypes and mechanisms of LV remodeling in obesity, (2) the effects of LSG and other bariatric surgical procedures on LVM in obesity, and (3) the limitations of two-dimensional (2D) echocardiography and increasing interest in real-time automated three-dimensional (3D) echocardiography for measurement of LVM as well as phenotypic classification of LVH in obesity.

LV Remodeling in Obesity

Pressure, volume, or pressure and volume cardiac overload alter LV structure and function, a process that is commonly referred to as LV remodeling [28]. Two-dimensional or 3D echocardiography allows serial measurements of LVM and geometry to assess progression of LV remodeling [29]. In contrast to CT angiography, 2D or 3D echocardiography does

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not expose patients to radiation, is less time consuming, and costs lower than cardiac magnetic resonance imaging (MRI) study. Echocardiographically, LVM is derived from linear measurements of LV end diastolic dimension (LVEDD) and LV posterior and septal wall thickness at end diastole (PWT, SWT) according to the formula: LVM (gram) = 0.8 × 1.04 × [(LVEDD + PWT + SWT) ^{3} − LVEDD^3] [30]. Traditionally, LVM is normalized to body surface area (BSA) to derive the LVM index (LVMI). LVH is defined as an LVMI >115 g/m^2 in men and >95 g/m^2 in women. Further characterization into concentric or eccentric LVH is based on an index of LV concentricity known as relative wall thickness (RWT) which is the ratio of 2 × PWT over LVEDD. In the presence of LVH, an RWT >0.42 suggests concentric hypertrophy, while RWT ≤0.42 suggests eccentric hypertrophy. In obese patients, the prevalence of LVH depends on whether LVM is indexed to BSA, or to height [1]. As BSA depends on body weight, indexing LVM to BSA attenuates the effects of obesity on LVM. Thus, LVM is preferably indexed to height$^{2.7}$ or to height$^{1.7}$ in obese patients. The exponent that best identifies LVM as a cardiovascular risk marker in obese patients is 1.7 [31].

Nearly three decades ago, measurements of RWT and LVM by 2D-guided M-mode echocardiography led to description of four patterns of LV remodeling in patients with hypertension (HTN) as detailed in Table 1 [32•]. By providing direct measurements of LVM and volume, cardiac MRI has allowed subclassification of eccentric LVH into indeterminate and dilated phenotypes and of concentric LVH into non-dilated and phenotypes (Table 2) [33••]. Thus, a 4-tiered classification of LVH has been proposed, wherein both concentric and eccentric LVH are further sub-divided based on the presence or absence of LV dilation [34].

The clinical implications of the 4-tiered classification of LVH were evaluated in the Campana Salute Network (CSN) registry of 8848 hypertensive patients [35]. Forty-four percent of patients were women and 42% were obese. The LV phenotypes (derived from measurements of LVM and volume by 2D echocardiography) and their prevalence are detailed in Table 3. Patients with non-dilated concentric LVH and with non-dilated and dilated eccentric LVH had a two- to nine-fold higher risk of cardiac events than patients with normal LV. Patients with dilated concentric LVH were free of significant cardiovascular risk. The 4-tiered classification of LVH identified hypertensive patients with a low risk of all cause or cardiovascular mortality in the Losartan Intervention for Endpoint reduction (LIFE) study [36]. Patients with eccentric non-dilated LVH were at lower risk of all cause or cardiovascular mortality, whereas patients with eccentric dilated LVH and concentric non-dilated and dilated LVH were at higher risk [36]. Lastly, the predictive value of the 4-tiered classification of LVH was confirmed in 2377 adults aged ≥65 years from the Cardiovascular Health Study over a 10-year follow up period [37]. In summary, addition of 2D-guided M-mode echocardiography derived LVM and volume improved cardiovascular risk prediction over that derived from standard scores.

When LVM is indexed to height$^{2.7}$ or height$^{1.7}$, an increased prevalence of LVH in obesity becomes apparent. The prevalence of LVH steadily increases with BMI ranging from 13% in normotensive obese individuals to 69% in morbidly obese individuals with BMI ≥50 kg/m^2 [11, 38]. Severe obesity as defined by a BMI ≥35 kg/m^2 was initially linked to LV dilation [38, 39]. The hemodynamic load and neurohormonal activation that accompany severe obesity were thought to promote LV dilation [38]. Subsequently, the LVM-to-volume ratio was correlated with BMI, waist circumference, and fat mass (FM) in 5098 participants in the Multi-Ethnic Study of Atherosclerosis (MESA), with a stronger association in men than in women [40•, 41]. The increase in LVM-to-volume ratio was due to a disproportionate rise in LVM compared to LV volume. Accordingly, a new paradigm that associates obesity to concentric LV remodeling was proposed [6, 42, 43]. The new paradigm was confirmed in 4343 elderly participants without overt cardiovascular diseases in the ARIC (Atherosclerosis Risk in Communities) study [44]. In the biracial cohort of this study, obesity was associated with an increase in LVM and a relatively smaller increase in LV diameter. Cardiac remodeling resulted in impaired LV function in women but not in men [44]. Further, BMI was reported to be the only significant independent predictor of LVM indexed to height in 400 male firefighters [45]. Lastly, obesity independently increased the risk of LVH in 4290 hypertensive participants in the CSN registry [46].

The relative contribution of total body weight (TBW), fat free mass (FFM), and fat mass (FM) to LVM and LV function was recently investigated in 1189 participants in the Study of Health in Pomerania (SHIP) with a follow up period of 5 years

Table 1 Phenotypes of LV remodeling by 2D-guided M-mode echocardiography

	Normal LV	Concentric remodeling	Concentric LVH	Eccentric LVH
LVM	Normal	Normal	↑	↑
RWT	Normal	↑	↑	Normal
Prevalence	52%	8%	13%	27%

Adapted from reference #32. *LV* left ventricular, *LVM* left ventricular mass, *RWT* relative wall thickness. RWT >0.42 concentric hypertrophy and RWT ≤0.42 eccentric hypertrophy

Table 2 Phenotypes of LV remodeling by cardiac MRI

	Concentric non-dilated LVH	Concentric dilated LVH	Eccentric indeterminate LVH	Eccentric dilated LVH
Increased LVM/height	+	+	+	+
Increased concentricity[a]	+	−	−	+
Increased LVEDV/BSA	−	+	−	+

Adapted from reference #33. *BSA* body surface area, *LV* left ventricular, *LVEDV* LV end diastolic volume, *LVM* left ventricular mass

[a] Concentricity = LV mass/LVEDV

[47]. Changes in LVM were shown to depend exclusively on changes in TBW and FM. An increase in FM was associated with LV concentric remodeling, while increases in TBW and FFM were associated with LV eccentric remodeling. Thus, body composition, and not total body weight, appears to determine the pattern of LV remodeling in obese patients.

Mechanisms of LV Remodeling in Obesity

Whether concentric or eccentric LV remodeling/LVH is the more prevalent phenotype in obesity remains an unresolved issue [3•]. The effects of duration of obesity and distribution of adipose tissue on LVM are difficult to estimate due to confounders, including HTN, obstructive sleep apnea, and oxidative stress that are frequently present in obese individuals [5, 48]. Overall, the mechanisms underlying LVH in obesity can be attributed to hemodynamic and non-hemodynamic factors (Fig. 1).

In addition to obesity-related renin-angiotensin-aldosterone and sympathetic nervous system activation, the high sodium load that accompanies high caloric intake tends to increase central blood volume, stroke volume, and cardiac output, and thereby promotes eccentric LVH [49]. Co-morbidities like HTN and obstructive sleep apnea add pressure overload to volume overload in obese patients, thereby promoting the concentric LVH phenotype [50]. While the concentric LVH phenotype does occur in obese patients free of co-morbidities [51, 52••], it is much more prevalent when obesity is associated with HTN. The effects of obesity and HTN on LVM are additive and interactive. Thus, in order to achieve effective LVH regression, therapy should target both weight loss and BP reduction in obese patients with HTN [1, 53•]. Similarly, all of the components of the metabolic syndrome (HTN, dyslipidemia, glucose intolerance, and obesity) contribute to increased LVM and need to be addressed in order to achieve substantial LVH regression [54].

Regional fat distribution is a key determinant of the LV remodeling phenotype in obesity [55]. Concentric LVH is linked to the amount of visceral adipose tissue (VAT) [41, 52••, 53•, 55]. Cardiac MRI assessment showed that concentric LV remodeling and VAT area are significantly related after adjustment for age, sex, ethnic background, HTN, diabetes, and lean body mass [52••]. Persistent elevation of systolic BP and reduced BP variability are associated with VAT and thereby tend to promote concentric LVH [56]. Lastly, both LVM and the amount of epicardial adipose tissue (EAT) increase with BMI [57–59]. However, the correlation between EAT and LVM is closer than that between LVM and BMI [57–59]. The precise mechanism delineating the contribution of EAT to LVH remains to be investigated.

Endocrine and paracrine release of inflammatory cytokines, angiotensin II, leptin, and adipokines by VAT also fosters development of concentric LVH in obese individuals [52••]. Macrophages infiltrate VAT and secrete large amounts of pro-inflammatory cytokines that, in turn, affect LV function and adverse remodeling [60, 61]. After adjusting for BMI and waist circumference, VAT remains significantly associated with C-reactive protein, interleukin-6, isoprostanes (marker of oxidative stress), and monocyte chemotractant protein-1 [62]. Further, the association between hyperglycemia/insulin resistance and LVH is primarily mediated by elevated BMI [63].

LV Mass After Laparoscopic Sleeve Gastrectomy

LSG has only gained wide acceptance as preferred surgical procedure for weight loss in the past few years. As a result, the effect of LSG on LV has not yet been extensively evaluated. The published literature provides an assessment of LVM post LSG in only 132 patients. Five single centers obtained 2D echocardiography and clinical parameters at baseline and at 6 to 12 months after LSG in groups of 8–51 patients [21–25]. All patients were BMI ≥35 kg/m^2 at baseline, and mean LV ejection fraction (LVEF) was 60% (range 55–63). BMI decreased from 46.6 kg/m^2 at baseline to 33.4 kg/m^2 at 6–12 months after LSG. LVM, derived by the Penn Convention [64], decreased from 218.4 to 181.4 g, an average reduction of 15% (7–21%). In four of the five studies, LSG

Table 3 Prevalence of LVH phenotypes in the Campania Salute Network (CSN) registry

Normal geometry	Concentric remodeling	Eccentric non-dilated LVH	Eccentric dilated LVH	Concentric non-dilated LVH	Concentric dilated LVH
66%	4.9%	20.4%	3.7%	5.1%	0.15%

Adapted from reference #35. *LVH* left ventricular hypertrophy

did not significantly alter LVEF, with increases in LVEF ranging from 1.3 to 3.3% [21–23, 25]. A 10% LVEF increase was reported in the only study that specifically enrolled obese patients with poorly controlled diabetes [24]. In this study, average HbA1c improved from 9.2% at baseline to 6.7% at 12 months following LSG. Only two of these five studies reported LV end diastolic volume (LVEDV) before and after LSG [22, 23]. Following LSG, LVEDV decreased from 126 to 119 mL and from 147 to 143 mL, respectively [22, 23]. All studies reported improvement in LV diastolic function after LSG, but none accounted for changes in cardiac loading conditions after LSG. The heterogeneity of 2D echocardiographic LV measurements and patient populations in these five studies precludes any attempt at meta-analysis.

LVM was evaluated by 3D echocardiography at baseline and after bariatric surgery in 52 patients, of whom 18 underwent LSG and 34 underwent laparoscopic RYGB (LRYGB) [26]. BMI decreased by 12 and 10 kg/m^2 after LSG and LRYGB, respectively. LSG decreased LVM from 115 to 99 g and LVEDV from 170 to 123 mL, while increasing LVEF from 63 to 68%. LSG and LRYGB had similar effects on LVM, LVEF, and LVEDV.

The effects of LSG on LV remodeling were recently assessed in 53 morbidly obese patients by 3D echocardiography [27]. Mean age and BMI were 37 years and 49 kg/m^2, respectively. Women comprised 62% of the patient population. LV measurements were obtained at baseline and 6 months after LSG. LVM decreased from 55 to 44 g/height$^{2.7}$, while LVEF increased from 60 to 68%. LVEDV decreased from 146 to 122 mL. LVEDV and LVM at baseline and LVM after LSG were significantly greater in men than women.

In summary, few studies have evaluated the effects of LSG on LVM. Preliminary experience indicates that 3D echocardiography provide more detailed assessment of LV structure and function than 2D echocardiography. The improvement in LV performance after bariatric surgery in patients who were evaluated with 3D echocardiography supports the view that the obesity-related decrease in LV systolic performance is reversible.

LV Mass After Gastric Bypass and Adjustable Banding

In 2013, Cuspidi et al. and Grapsa et al. reviewed the quantitative data on LV structure and/or function after bariatric surgery [4, 13]. Cuspidi et al. selected 23 reports for systematic review and meta-analysis [13]. The surgical procedures were RYGB, AGB, or vertical banded gastroplasty (VBG) in 21 of the 23 reports. Absolute LVM was available in 13 reports, and LVM indexed to height$^{2.7}$ in 8 reports. The decrease in absolute LVM averaged 33.6 g ranging from 13 to 63 g. While they

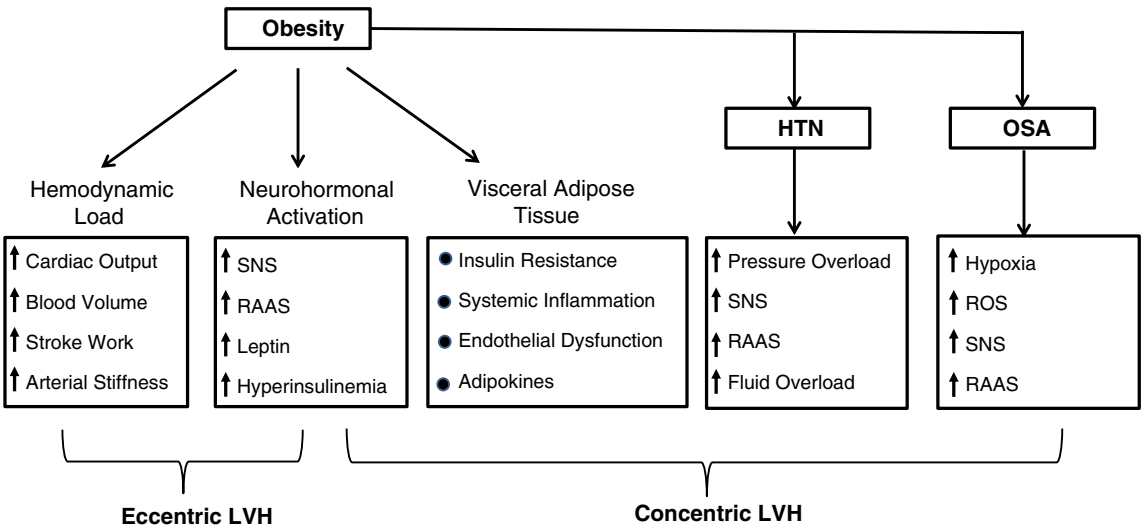

Fig. 1 Pathophysiology of eccentric and concentric left ventricular hypertrophy associated with obesity. *HTN* hypertension, *LVH* left ventricular hypertrophy, *OSA* obstructive sleep apnea, *ROS* reactive oxygen species, *RAAS* renin angiotensin aldosterone system, *SNS* sympathetic nervous system

conclude to an overall beneficial effect of bariatric surgery on LVM, Cuspidi et al. drew attention to the variable effects of bariatric surgery on LVM. They attributed the variability to the limited accuracy of 2D echocardiography for measurement of LVM, to differences in duration of obesity and follow up, as well as in pre-operative LVM and BMI, and in the various surgical procedures themselves [13]. Reviewing 11 studies that assessed the effect of bariatric surgery on LVM by 2D echocardiography, Grapsa et al. confirmed that LVM decreases after bariatric surgical procedures [4]. Nevertheless, these authors advocated for more precise measurement of LV structure and function than 2D echocardiography allows [4]. To put the bariatric surgery data in perspective, antihypertensive therapy for a mean duration of 67 months decreased LVM by 9 $g/m^{2.7}$ in the 2173 patients of the Campania Salute Network (CSN) [65].

A decrease in LVM from 192 to 146 g over a period of 15 months after RYGB was recently reported in 37 young patients [63]. The decrease in LVM was significantly related to reductions in BMI ($r = 0.53$, $p < 0.001$) and VA ($r = 0.47$, $p < 0.005$), whereas improvement in insulin resistance (assessed by homeostasis computer modeling) was only related to the reduction in VAT [66]. The mechanisms that mediate LVM regression after bariatric surgery are being actively investigated [67]. Weight loss-induced hemodynamic alterations including reductions in cardiac work load, plasma volume, and BP contribute to lower LVM. Bariatric surgery may also lower LVM by modulating release of adipocyte hormones such as leptin, resistin, and adiponectin [60, 67]. The close correlation of LVM regression with change in BMI implies weight loss-induced hemodynamic alterations as predominant mechanisms of LVM regression [9]. On another hand, the continued decline of LVM without further decrease in BMI suggests that non-hemodynamic factors factors contribute to the decrease in LVM after bariatric surgery [10]. By providing accurate and relatively inexpensive measurements of LV structure and function, 3D echocardiography, in conjunction with serial determinations of BP and serum levels of adipocyte hormones, may allow to ascertain the relative contribution of hemodynamic factors and VAT hormones to LVM regression after bariatric surgery.

LV Mass by 2D and 3D Echocardiography

By convention, LVM is measured at the end of diastole. By 2D-guided M-mode echocardiography, LVM is derived from linear measurements of the LV as previously mentioned. By 2D echocardiography, LVM can also be derived from linear measurements of LV minor radius (b), the distance from the minor axis to the endocardium of the LV apex (a), the distance from the minor axis to the mitral plane (d), and LV wall thickness (t) according to the truncated ellipsoid formula $LVM = 1.04\ \pi\{(b + t).[_{2/3}\ (a + t) + d—d^3/3(a + t)^2 - b^2[2/3a + d - d^3/3a^2]\}$ [68].

By 3D echocardiography, LVM is derived from the difference between LV epicardial and endocardial volumes. LV mass is equal to (LV epicardial − LV endocardial volume) × 1.05 g [68]. In contrast to M mode and 2D echocardiography, 3D echocardiography measures LVM without making any geometric assumptions [68]. 3D echocardiography is particularly suited for monitoring the LV remodeling process. 2D echocardiography with fixed geometric assumptions cannot account for changes in LV geometry with time, intervention, or both. Time-consuming data acquisition and data analysis initially thwarted the adoption of 3D echocardiography for evaluation of LV volumes [69]. The development of matrix array transducer and novel automated software has greatly facilitated full LV volume quantification by real-time 3D echocardiography [70]. Automated algorithm-derived LV volumes by 3D echocardiography have been validated against cardiac MRI [71]. 3D echocardiography slightly underestimates LV volume due to poor differentiation between the compacted myocardium and trabeculae, which is a greater issue in systole than in diastole [71]. However, quantifications of LVM by real-time 3D echocardiography and cardiac MRI are in high agreement [72].

Whether M-mode and 2D echocardiography provide accurate measurements of LVM has been previously questioned [34]. Poor echocardiographic windows in obese patients and geometric assumptions make it technically difficult to derive accurate LV volumes and mass by M-mode and 2D echocardiography. M-mode echocardiography overestimates LVM as the result of unintended oblique cuts, and 2D echocardiography underestimates LVM as the result of foreshortening of apical views [73–75]. M-mode echocardiography could have contributed to the high prevalence of eccentric LV remodeling in obesity due to overestimation of LV end diastolic dimension [76]. When assessed by MRI, the gold standard imaging technique, hyperglycemia/insulin resistance have consistent effects on cardiac function and structure [63] However, when assessed by 2D echocardiography, hyperglycemia/insulin resistance have markedly disparate effects that raise concerns about the accuracy of this imaging technique for assessment of cardiac function and structure [63].

Conclusion

In summary, M-mode and 2D echocardiography provide an incomplete and sometimes inaccurate assessment of LV remodeling in obesity. Further, assessment of LVM after bariatric surgery is hampered by changes in LV geometry that are not directly accounted for by M-mode and 2D echocardiography. In contrast, real-time 3D echocardiography appears particularly well suited for assessing LVM and function after

bariatric surgery, as it does not rely on geometric assumptions to quantify LVM. Serial measurements of BP and LVM may help delineate the mechanisms that mediate LVM regression after bariatric surgery.

Compliance with Ethical Standards

Conflict of Interest The authors declare no conflicts of interest relevant to this manuscript.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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- •• Of major importance

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