ESC European Society of Cardiology
European Heart Journal Open (2021) **00**, 1–10
doi:10.1093/ehjopen/oeab024

ORIGINAL ARTICLE
Special populations

One-year impact of bariatric surgery on left ventricular mechanics: results from the prospective FatWest study

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Received 21 June 2021; revised 22 July 2021; editorial decision 18 August 2021; accepted 18 August 2021; online publish-ahead-of-print 20 August 2021;

Handling Editor: Frank A. Flachskampf

Aims Patients with severe obesity are predisposed to left ventricular (LV) hypertrophy, increased myocardial oxygen demand, and impaired myocardial mechanics. Bariatric surgery leads to rapid weight loss and improves cardiovascular risk profile. The present prospective study assesses whether LV wall mechanics improve 1 year after bariatric surgery.

Methods and results Ninety-four severely obese patients [43 ± 10 years, 71% women, body mass index (BMI) 41.8 ± 4.9 kg/m^2, 57% with hypertension] underwent echocardiography before, 6 months and 1 year after gastric bypass surgery in the FatWest (Bariatric Surgery on the West Coast of Norway) study. We assessed LV mechanics by midwall shortening (MWS) and global longitudinal strain (GLS), LV power/mass as 0.222 × cardiac output × mean blood pressure (BP)/LV mass, and myocardial oxygen demand as the LV mass-wall stress-heart rate product. Surgery induced a significant reduction in BMI, heart rate, and BP ($P < 0.001$). Prevalence of LV hypertrophy fell from 35% to 19% 1 year after surgery ($P < 0.001$). The absolute value of GLS improved by—4.6% (i.e. 29% increase in GLS) while LV ejection fraction, MWS, and LV power/mass remained unchanged. In multivariate regression analyses, 1 year improvement in GLS was predicted by lower preoperative GLS, larger mean BP, and BMI reduction (all $P < 0.05$). Low 1-year MWS was associated with female sex, preoperative hypertension, and higher 1-year LV relative wall thickness and myocardial oxygen demand (all $P < 0.001$).

Conclusion In severely obese patients, LV longitudinal function is largely recovered one year after bariatric surgery due to reduced afterload. LV midwall mechanics does not improve, particularly in women and patients with persistent LV geometric abnormalities.

ClinicalTrials.gov identifier NCT01533142, 15 February 2012.

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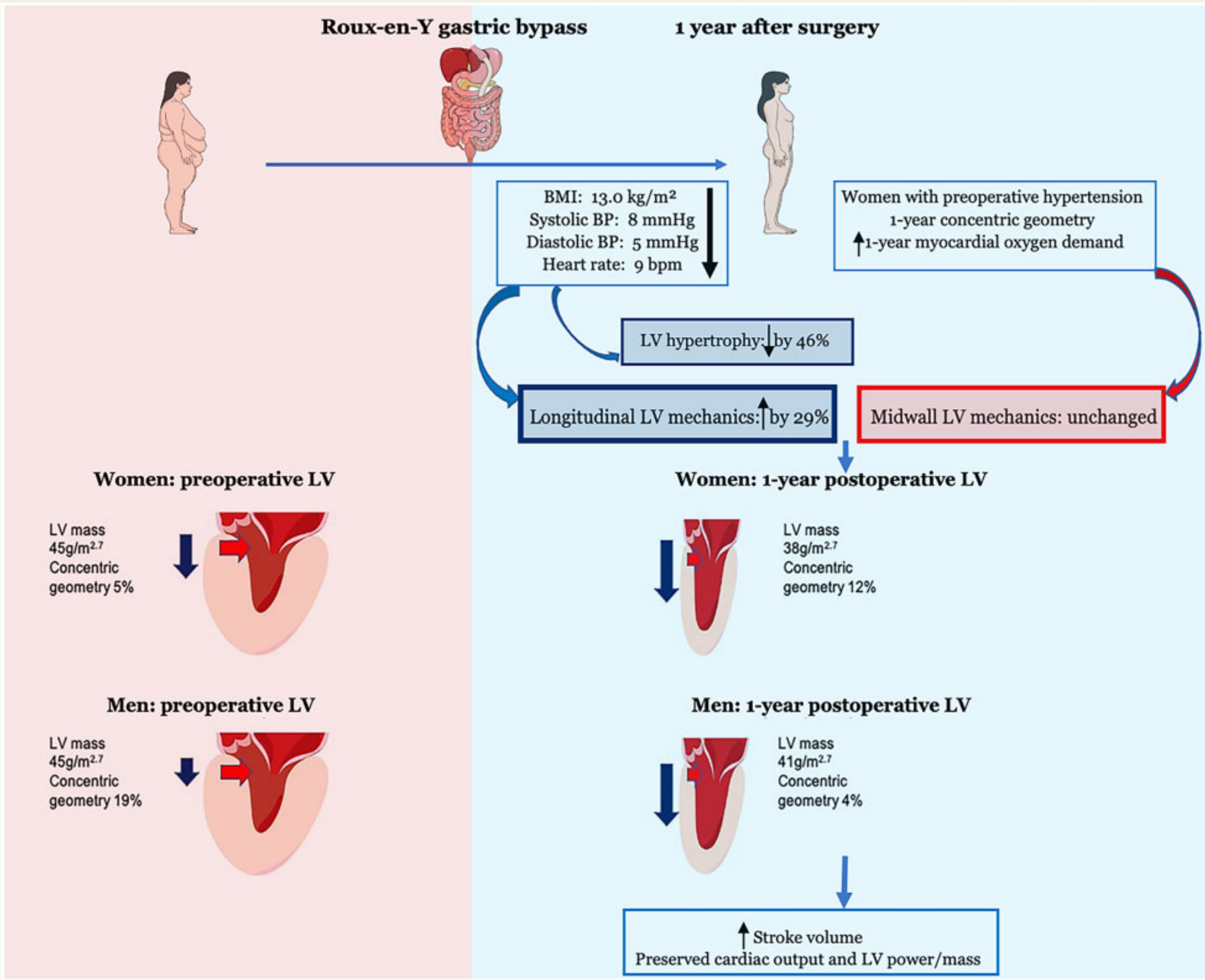
Graphical Abstract



Keywords Obesity • Left ventricular function • Echocardiography • Bariatric surgery

Introduction

Severe obesity has increased substantially and in a continuous manner during the past 20 years and is estimated to affect 177 million adults by 2025.[1] Its associations with high cardiovascular risk[2,3] and subclinical cardiac disease[4] have been well-documented. The ongoing coronavirus disease 2019 (COVID-19) pandemic has additionally demonstrated obesity as a risk factor for cardiac complications, more severe disease course, and increased COVID-19 mortality, particularly in younger patients.[5]

Bariatric surgery has established itself as an effective method of reducing the adipose mass, with an increasing number of referred patients during the last two decades, although access to surgery is still limited,[6] and reportedly worse during the COVID-19 pandemic.[7] Bariatric surgery reduces the long-term cardiovascular risk and all-cause mortality in patients with severe obesity.[8] It has also been shown to beneficially reduce left ventricular (LV) mass and left atrial size,[9] while the effect on LV systolic function and mechanics have been little explored. Earlier small studies found no postoperative change in LV ejection fraction (EF),[10,11] while a recent report in 40 patients showed improvement in LV global longitudinal strain (GLS) 10 months after bariatric surgery.[12] Lifestyle changes have also shown a beneficial effect on left atrial[13] and LV function[14] in other conditions characterized by persistent inflammation.

In the prospective FatWest (Bariatric Surgery on the West Coast of Norway) study, we have previously documented a high preoperative prevalence of impaired LV mechanics in patients with severe obesity, in particular in those with high LV myocardial oxygen demand.[4] In the present analysis, we evaluate the amplitude and the determinants of changes in LV systolic function (assessed by the LV EF) and mechanics [by LV GLS and midwall shortening (MWS)] in the FatWest population during the first year after bariatric surgery.

Methods

Study design

The FatWest study is a prospective cohort study that recruited 123 patients with severe obesity living on the West Coast of Norway and referred for a Roux-en-Y gastric bypass in the time period 2012–2016. Inclusion and exclusion criteria have been previously reported.[4] In the FatWest study patients are followed by clinical examination, echocardiography, and polysomnography preoperatively as well as 6 months, 1-, 2-, and 5-year postoperatively. The present analysis is based on the preoperative, and the first two follow-up visits planned 6 months and 1 year after surgery according to the study protocol. Among the 123 patients included in the FatWest study, LV GLS could be analysed in 116 patients (94%). Ninety-eight of these 116 patients presented at the postoperative 6 months visit, and 95 at the postoperative 1-year visit. The remaining 28 patients presented at later postoperative visits and were not included in the present analysis. This group of late presenters was younger (35 ± 11 vs. 43 ± 10 years) and included several women (82% vs. 71%) but had similar MWS and GLS with the patients that were examined earlier. Full echocardiograms at both the 6-month and 1-year controls were available in 94 patients that thus constitute the present study population.

Hypertension was defined preoperatively as a history of hypertension, blood pressure (BP) ≥140/90 mmHg at the baseline clinical visit, and/or use of antihypertensive drugs.

Early postoperative complications in the FatWest population have been previously published.[4] In the present report, we summarize the complications occurring after the perioperative period and up to 1 year after surgery.

The study was approved by the regional ethics committee (2021/22196) and was conducted by the revised Declaration of Helsinki. All patients signed a written informed consent before study examinations.

Echocardiographic measurements

Transthoracic echocardiogram including dedicated loops for myocardial deformation analyses by 2D speckle tracking was performed at all study visits by the same experienced cardiologist (S.N.) using a Vivid E9 scanner (GE Vingmed Ultrasound AS Horten, Norway) and a standardized image protocol. Digital images were stored on Digital Versatile Discs and forwarded to the study core laboratory in Bergen, Norway for analyses using Tomtec workstations with Image Arena 4.6 software (Tomtec, Unterschleissheim, Germany).

LV structure

LV size was evaluated by the 2D wall and cavity linear dimensions and the Simpson's biplane volumes following the joint American Society of Echocardiography and European Association of Cardiovascular Imaging guidelines.[15] LV hypertrophy was identified as recommended in obesity as LV mass indexed for height$^{2.7}$ ≥49.2 g/m$^{2.7}$ in men and ≥46.7 g/m$^{2.7}$ in women.[4,16] The ratio of LV end-diastolic posterior wall thickness/internal diameter (the relative wall thickness) indicated concentric LV geometry when equal to or above 0.43.[15]

Additionally, total arterial peripheral resistance was estimated from the ratio of 80 × mean BP to cardiac output.[17]

Left ventricular systolic function

LV systolic function was assessed by the Simpson's biplane EF (low if <52% in men and <54% in women, respectively).[15] LV wall mechanics were assessed in the radial direction by MWS (low if <14% in men and <16% in women), a parameter with high inter- and intra-observer agreement,[18] and in the longitudinal direction by GLS (low if less negative than -16.7% in men and < -17.8% in women).[19] MWS was additionally corrected for circumferential end-systolic stress as an expression of LV myocardial function (low if <87% in men and <90% in women).[20] GLS was analysed by using all three standard apical views in a vendor-independent

Table 1 Clinical changes 6 months and 1 year after bariatric surgery, compared to preoperative findings

	Preoperative visit	6 months postoperatively	1 year postoperatively	*P*-value
Clinical data				
Follow-up time (months)		5.8 ± 2.9	14.0 ± 2.7	NA
Age at study baseline (years)	43 ± 10			NA
Female sex	71%			NA
Weight (kg)	121 ± 19	91 ± 17	83 ± 16	<0.001
BMI (kg/m^2)	41.8 ± 4.9	31.7 ± 5.0	28.8 ± 4.7	<0.001
Heart rate (b.p.m.)	74.4 ± 12.8	64.8 ± 10.3	65.4 ± 9.3	<0.001
Systolic BP (mmHg)	135 ± 10	128 ± 8	126 ± 9	<0.001
Diastolic BP (mmHg)	87 ± 7	83 ± 5	83 ± 5	<0.001
Mean BP (mmHg)	103 ± 8	98 ± 6	97 ± 6	<0.001
Hypertension at study baseline	57%			NA
Diabetes at study baseline	16%			NA
Medical treatment				
Antihypertensive medication	29%	18%	16%	<0.001
Antidiabetic medication	16%	6%	6%	<0.01
Lipid-lowering medication	15%	5%	4%	<0.001

Results are presented as mean ± standard deviation or percentages. *P*-value is based on general linear models for repeated measures for continuous variables and Cochran's *Q* test for categorical variables.

Table 2 Echocardiographical changes 6 months and 1 year after bariatric surgery, compared to preoperative findings

	Preoperative visit	6 months postoperatively	1 year postoperatively	P-value
Echocardiographic data				
LV end-diastolic diameter (cm)	5.06 ± 0.41	4.97 ± 0.46	4.94 ± 0.42	<0.01
LV end-systolic diameter (cm)	3.43 ± 0.38	3.36 ± 0.39	3.34 ± 0.35	0.06
LV end-diastolic volume (mL)	120 ± 30	138 ± 32	135 ± 29	<0.001
LV end-systolic volume (mL)	47 ± 14	55 ± 16	55 ± 15	<0.001
LV mass index ($g/m^{2.7}$)	44.6 ± 12.2	41.3 ± 11.7	38.9 ± 10.8	<0.001
LV hypertrophy	35%	20%	19%	<0.001
Relative wall thickness	0.34 ± 0.07	0.36 ± 0.08	0.33 ± 0.07	0.02
LV EF (%)	61 ± 5	60 ± 4	60 ± 5	0.32
Stroke volume (mL)	74 ± 19	82 ± 18	80 ± 16	<0.001
Cardiac output (L/min)	5.4 ± 1.6	5.3 ± 1.3	5.2 ± 1.2	0.49
LV power/mass	0.93 ± 0.30	0.88 ± 0.23	0.91 ± 0.24	0.26
MWS (%)	15.8 ± 2.6	16.0 ± 2.8	16.2 ± 2.5	0.24
Circumferential end-systolic stress ($dyne/cm^2$)	119 ± 23	103 ± 21	106 ± 20	<0.001
scMWS (%)	90.9 ± 14.4	90.1 ± 15.8	91.8 ± 13.6	0.67
GLS (%)	-15.8 ± 4.8	-19.6 ± 3.8	-20.4 ± 2.8	<0.001
LV mass-wall stress-heart rate product ($\times 10^6$ g $kdyne/cm^2$ b.p.m.)	1.64 ± 0.57	1.13 ± 0.38	1.13 ± 0.41	<0.001
Total peripheral resistance (dynes $\times$ s $\times$ cm^{-5})	1637.1 ± 478.6	1562.3 ± 348.6	1558.6 ± 357.7	0.08

Results are presented as mean ± standard deviation or percentages. *P*-value is based on general linear models for repeated measures for continuous variables and Cochran's *Q* test for categorical variables.

software package (2D Cardiac Performance Analysis, Image Arena, Tomtec).[19]

LV pump performance was assessed by the biplane stroke volume, cardiac output, and cardiac power normalized for LV mass using the following validated formula: 0.222 $\times$ cardiac output $\times$ mean BP/LV mass.[21] LV myocardial oxygen demand was estimated from the LV mass-wall stress-heart rate product.[22]

Statistical analyses

All statistical analyses were performed using IBM SPSS Statistics 26.0 (IBM Corp., Armonk, NY, USA). To compare clinical and echocardiographic findings in the study population at the three study visits, general linear models for repeated measures with Bonferroni *post hoc* test were used for continuous, normally distributed data and Friedman's test for not normally distributed data. When sphericity was not obtained, the level of significance was tested using the Greenhouse–Geisser method. Chi-square and Cochran's *Q* test with *post hoc* analysis were used for categorical variables. Findings are reported as mean ± standard deviation for continuous variables and as percentages for categorical variables.

Predictors of improvement in LV GLS from baseline to the 1-year postoperative visit were identified in multivariate regression analysis with backward stepwise selection of covariates out of the following parameters: age, sex, preoperative hypertension, preoperative GLS and EF, and the 1-year absolute change in heart rate, mean BP, body mass index (BMI), and myocardial oxygen demand. All variables were previously entered in the regression model at the same time by using an entry procedure to ensure that variables selected in a stepwise fashion in the final model account for most of the model variance. Results are presented as standardized β coefficients with *P*-values of significance. Similarly, covariates of low MWS at the 1-year visit were identified by backward stepwise logistic regression analysis among the following: age, sex, preoperative hypertension, and 1-year BMI, mean BP or total peripheral resistance, heart rate, LV relative wall thickness, and myocardial oxygen demand. Results are presented as *B* coefficients, Wald, and *P*-values of significance. Covariates in all multiple regression models were selected purposefully based both on clinical relevance and a $P < 0.1$ level of significance for the association with the dependent variables in univariate analyses.[23] A two-tailed $P < 0.05$ was considered significant in all analyses.

Results

Changes in clinical and haemodynamic factors

The patients experienced on average a weight reduction during follow-up of 38 ± 12 kg, and 63% of the initially severely obese cohort became overweight at the 1-year visit. The BMI change was accompanied by an 8 ± 10 and 5 ± 6 mmHg reduction in systolic and diastolic BP, respectively, and a 9 ± 12 b.p.m. reduction in heart rate, with 80% of the anthropometric and haemodynamic changes taking place during the first six postoperative months (*Table 1*). Forty-eight percent and 60% of patients on preoperative antihypertensive and antidiabetic medication, respectively, did no longer use this medication at the 1-year follow-up (*Table 1*).

Changes in LV geometry and mechanics

During the first postoperative year, LV wall thickness and mass decreased gradually, with a mild increase in LV biplane internal volumes (*Table 2*). Consequently, the prevalence of 1-year LV hypertrophy was reduced from 35% to 19%, most prominently among those with eccentric hypertrophy (*Figure 1A*).

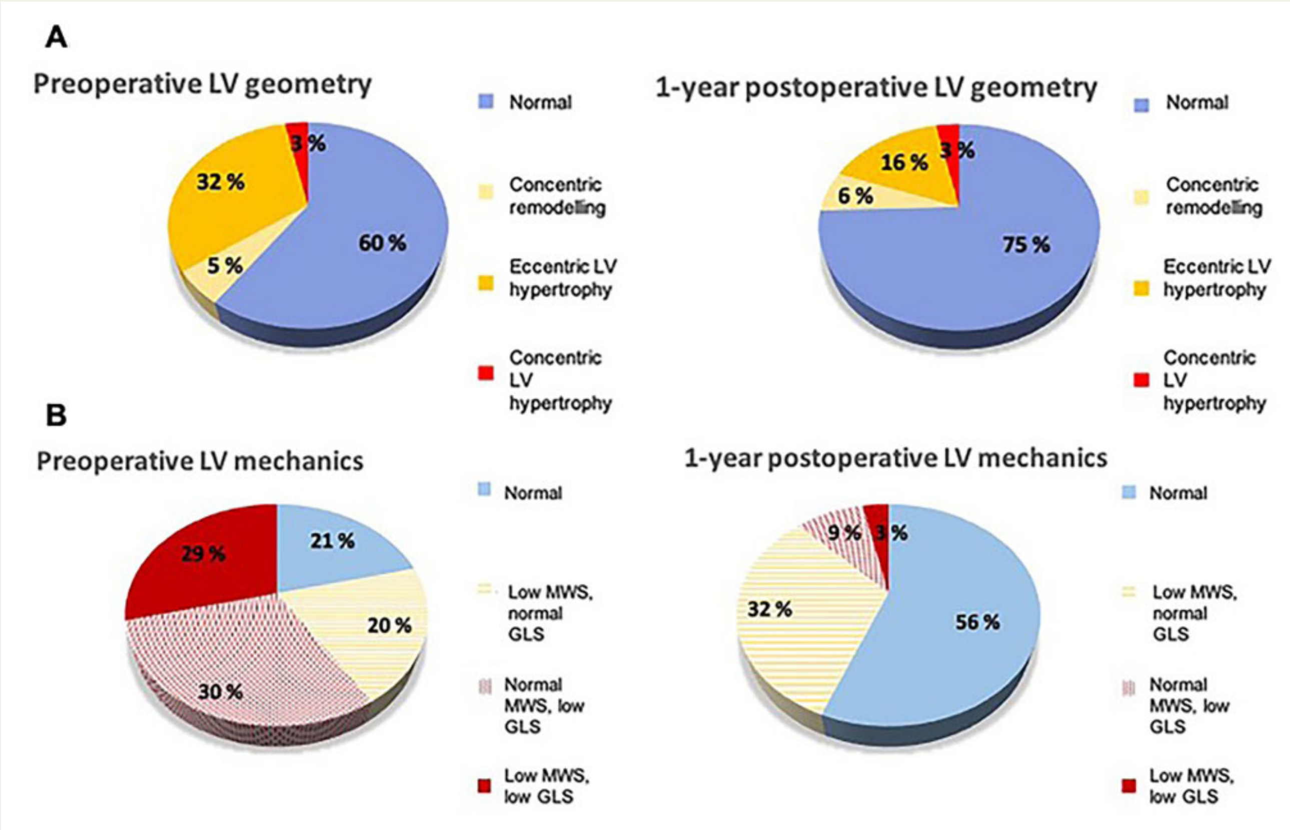


Figure 1 (*A*) LV geometry (normal geometry, concentric remodelling, eccentric hypertrophy, and concentric hypertrophy) preoperatively (left diagram) and 1 year after bariatric surgery (right diagram). (*B*) Patterns of LV mechanics (normal MWS and GLS, low MWS with normal GLS, normal MWS with low GLS, and combined low MWS and low GLS) preoperatively (left diagram) and 1 year after bariatric surgery (right diagram)

LV mechanics also improved following bariatric surgery, but abnormal LV mechanics remained more prevalent than abnormal LV geometry during follow-up (*Figure 1*). In particular, GLS increased markedly (*Table 2*), and the proportion of patients with low GLS was reduced by 80% at the 1-year control. In contrast, the prevalence of low MWS remained high 1 year after surgery (35% vs. 49% preoperatively, $P = 0.07$) (*Figure 1B*). Similar results were found when MWS was corrected for circumferential end-systolic stress. LV EF and power remained normal and unchanged during follow-up (*Table 2*).

In backward stepwise multivariate linear regression analysis, a larger 1-year improvement in GLS was associated with lower preoperative GLS, and larger reductions in mean BP and BMI during follow-up ($P < 0.001$, *Table 3*). Age, preoperative hypertension, change in myocardial oxygen demand and preoperative EF did not enter the model. In backward stepwise logistic regression analysis, the presence of low MWS 1 year after surgery was associated with preoperative hypertension, female sex, and higher 1-year LV relative wall thickness and myocardial oxygen demand (all $P < 0.001$, *Table 3*). Neither age nor 1-year heart rate, mean BP, or total peripheral resistance entered the model. When preoperative diabetes was forced into the multivariate models, the results remained unchanged.

Sex differences in LV geometry and mechanics

At study inclusion, hypertension was more common in men (74% vs. 51%, $P < 0.05$), while age, BMI, and prevalence of diabetes did not differ by sex. Men had a higher prevalence of LV concentric geometry, ($P < 0.05$), while LV mass index did not differ (*Figure 2*). Bariatric surgery resulted in a similar BMI reduction ($13.0 \pm 3.8\,kg/m^2$, i.e. 31% of baseline value in women and 32% in men), mean BP drop (6 ± 7mmHg), and pulse pressure reduction (4 ± 7mmHg) in women and men. Septum thickness and LV mass decreased significantly during follow-up in both sexes, while LV internal diameter was reduced only in women (all $P < 0.05$), resulting in a numerically higher proportion of women than men with LV concentric remodelling at the 1-year control (*Figure 2*).

Abnormal LV GLS was highly prevalent preoperatively and improved in both sexes after surgery, though significantly more in men than in women (6.7% vs. 3.9%, $P < 0.05$) (*Figure 3*). However, midwall mechanics, although showing a positive trend, particularly in men, did not improve significantly in either sex and remained abnormal in 42% of women at the 1-year visit (*Figure 3*).

Table 3 Multivariate regression analyses with a stepwise backward procedure

1-year improvement in GLS	**R^2 = 0.74, P < 0.001. Excluded variables: age, preoperative hypertension, change in myocardial oxygen demand, EF**		
	Beta	*t*	*P*-value
Preoperative GLS	0.88	14.46	<0.001
Female sex	0.11	1.76	0.08
1-year fall in heart rate	-0.12	-1.88	0.06
1-year fall in mean BP	0.11	1.98	0.05
1-year decrease in BMI	0.13	2.14	0.04
1-year low MWS	**Nagelkerke R^2 = 0.41, P < 0.01. Excluded variables: age, 1-year mean BP or 1-year total peripheral resistance, 1-year heart rate**		
	B	Wald	*P*-value
Female sex	2.81	10.53	0.001
Preoperative hypertension	1.19	3.83	0.05
1-year BMI	-0.11	3.05	0.08
1-year LV relative wall thickness	16.50	9.88	0.002
1-year myocardial oxygen demand	2.13	5.71	0.02

Predictors of improvement in GLS from baseline to 1 year after surgery are identified in multivariate linear regression analysis, after stepwise removal of non-significant variables due to lack of significance. Covariates of low MWS at a 1-year visit are identified in logistic regression analysis, after stepwise removal of non-significant variables.

Postoperative complications

The total prevalence of complications was low during the 1-year follow-up. Paroxysmal atrial fibrillation was documented in three patients (3%), all in sinus rhythm at study visits. The most common complications (8%) involved the gastrointestinal tract and included peptic ulcers, ventral hernias, gastritis, esophagitis, gallstone diseases, and oesophageal strictures. Two patients (2%) developed severe malnutrition.

Discussion

In this prospective study of a cohort of patients with severe obesity and a high preoperative prevalence of subclinical LV dysfunction, we demonstrate that although LV hypertrophy is reduced by 46%, and LV geometry normalized in 75% of participants, many patients remain with reduced LV midwall mechanics 1 year after bariatric surgery. Contrasting a substantial improvement in GLS with an 80% reduction in the prevalence of low GLS, low MWS was still common in particular in women and patients with preoperative hypertension. Changes in LV function were not detected by LV EF or power normalized for mass, both parameters unaltered and in the normal range at all study visits.

LV mechanics after bariatric surgery

Bariatric surgery is associated with a significantly lower long-term risk of cardiovascular events, including a lower risk of incident heart failure in prospective studies with up to 11 and 22 years follow-up.[8,24] Several reports from small studies have documented unchanged EF after bariatric surgery,[9,25–28] while few studies have assessed changes in LV myocardial function. Recently, improvement in GLS (measured in QLab, Phillips Medical Systems) from -17% at baseline to -20% 10 months after bariatric surgery was reported in a study of 40 patients.[12] The present study adds to current knowledge by demonstrating a large improvement in LV's longitudinal mechanics, with a 29% increase in the absolute GLS value 1 year postoperatively. In line with previous observations, the postoperative GLS increase was largest in those with the lowest preoperative GLS and associated with the postoperative reduction in afterload (lower mean BP and BMI). Consequently, the largest GLS improvement took place during the first six postoperative months, paralleling when most of the bodyweight and BP reduction occurred.

Fewer patients had improved postoperative LV midwall mechanics when assessed by the LV MWS, a prognostically validated parameter in patients with hypertension[29] and valve disease.[18] Low MWS 1 year postoperatively was associated with the presence of concentric LV geometry, as also observed in other patient cohorts with hypertension, aortic valve stenosis, and type 2 diabetes.[18,30] Furthermore, low MWS has been associated with higher myocardial oxygen demand both in obesity and in aortic valve stenosis.[4,31] Interestingly, preoperative hypertension was associated with persistently low MWS 1 year after surgery despite the significant postoperative BP reduction, raising the question of whether these patients, despite younger age and due to combined hypertension and longstanding obesity, have permanent hypertension-mediated myocardial damage with increased midwall fibrosis.[32] Fibrosis would though be expected to impact GLS also, unless if concentrated at midwall and not extensive. Longer follow-up of these patients with advanced echocardiography will help clarify the reversibility of abnormal midwall function.

LV EF remained normal throughout the study, confirming once again its low sensitivity in detecting myocardial dysfunction.[33] LV power/mass quantifies LV energy rate per 100 g of LV mass and has been shown to predict the development of heart failure in patients with initially normal EF followed-up for a median of 3.9 years.[21] In the present cohort, LV power/mass at rest did not change at postoperative visits, reflecting the parallel improvements in BP, flow, and LV

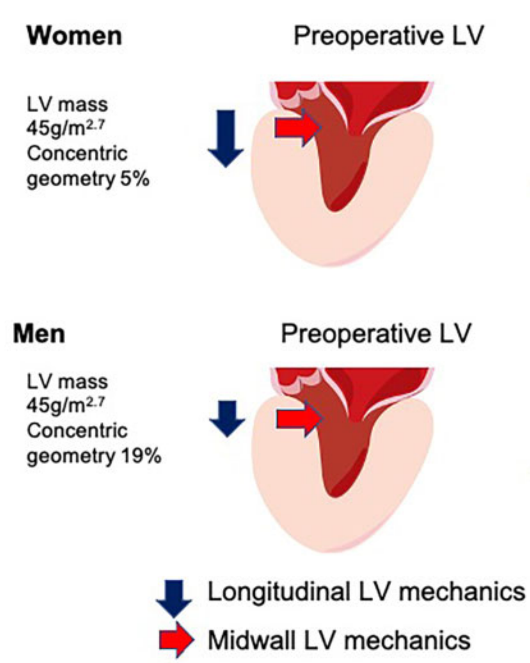


Figure 2 Changes in LV size and mechanics illustrated in women (top) and men (bottom) preoperatively (left illustration) and 1 year after bariatric surgery (right illustration). Changes in LV GLS are depicted with blue arrows, and in LV MWS with red arrows. The length of the arrows illustrates the magnitude of the changes

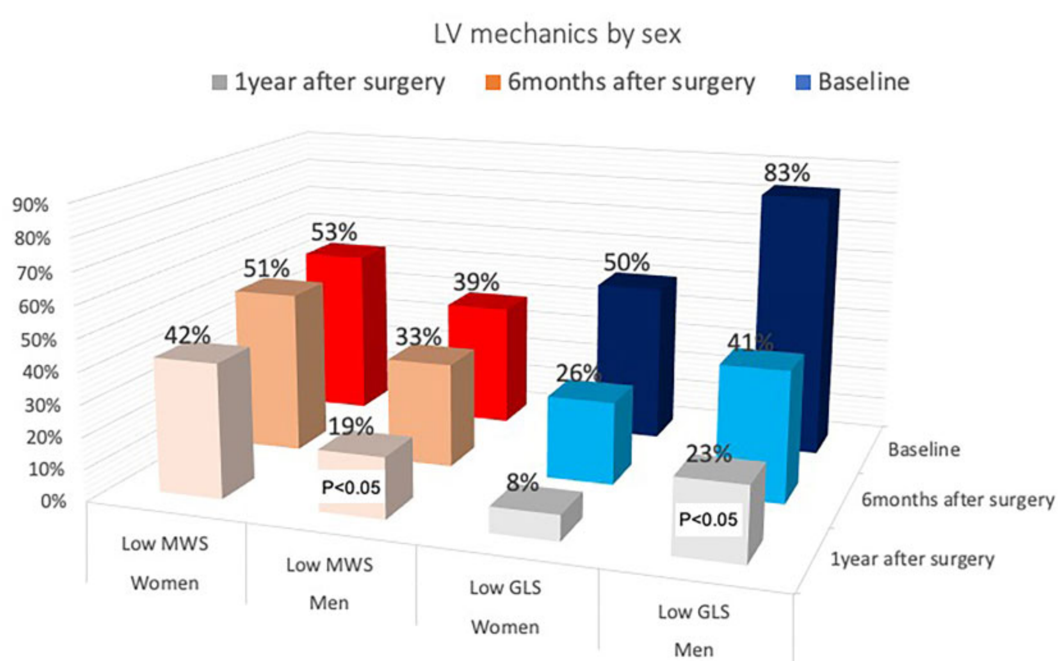


Figure 3 Sex-specific improvement in LV myocardial dysfunction during follow-up. The *P*-value indicates a significant difference between women and men at the respective visit

mass. However, stroke volume increased significantly at the 1-year control, allowing a stable cardiac output despite the postoperative reduction in heart rate. The improved LV filling underscores an improved energy balance which is consistent with the expected higher insulin sensitivity after bariatric surgery.

LV mechanics in relation to geometry and sex

LV mass decreased significantly following bariatric surgery, in line with several previous reports.[9–11] Data on LV volumes have been contradictory, reflecting the different methodologies used.[11] By the biplane Simpson's method, we find a mild postoperative increase in LV volumes, possibly due to reduced LV wall thickness, in addition to improved LV compliance due to less adipose tissue on the thoracic wall and less epicardial fat.

LV geometrical changes are often thought to precede and explain LV functional disturbances.[34] In our population abnormal LV mechanics was more prevalent than abnormal LV geometry both pre- and postoperatively, probably reflecting that longstanding obesity, often in combination with hypertension and diabetes, may alter myocardial quality through a relative increase of myocardial interstitial tissue and fibrosis without large mass increase.[35] Furthermore, heterogeneous regional geometric remodelling, that is not identified by the traditional definition of LV geometry[36] and would require 3D visualization, may have been present.

In women, low MWS remained more prevalent than in men 1 year after bariatric surgery, despite a lower incidence of preoperative hypertension, similar initial BMI, and similar postoperative BMI and BP reduction. This is partially explained by the LV geometric changes, with increasing concentric remodelling in women. However, based on the fact that women retain higher MWS in other types of chronic LV overload conditions as hypertension[37] and aortic stenosis,[38] and considering the large proportion of women with abnormal midwall function following bariatric surgery, other factors unaccounted for in our study, as the level of inflammation and sex-differences in arterial function and load,[39] might play a role.

Clinical implications

The global obesity epidemic has worsened during the COVID-19 pandemic,[5] and its therapeutic options have become even more limited.[7] In the context of the expected repercussions on population health post-pandemic, understanding the cardiac changes induced by an effective weight-reducing method like bariatric surgery is important. Even if the documented positive effects of bariatric surgery on long-term cardiovascular outcomes are solid,[8] detailed analyses of structural and functional cardiac changes induced by bariatric surgery are necessary for refining the indications for surgery and selecting the most appropriate candidates.

Study limitations

Both geometric and functional LV remodelling might have heterogeneous regional distribution and be better diagnosed by 3D echocardiography or cardiac magnetic resonance imaging. However, these techniques have limited availability and are less feasible in patients with severe obesity. Good echocardiographic image quality is mandatory for strain analyses, and, despite being often a challenge in obese patients, it was achieved by one experienced cardiologist (SN) in 94% of the preoperative examinations. We can however not exclude that changing image quality during follow-up could partially have contributed to the observed postoperative improvement in GLS. The level of physical fitness has been reported to correlate with LV GLS in overweight and mildly obese subjects.[40] Systematic data on physical fitness was not recorded in the FatWest study, but severely obese patients are generally sedentary, and most remain physically inactive also postoperatively.[41] Peak stress LV power/mass estimated by exercise echocardiography is a more powerful predictor of adverse cardiovascular outcome than rest power/mass.[21] Exercise testing was not part of the FatWest study.

Conclusion

Despite a significant reduction in abnormal LV geometry following bariatric surgery, longitudinal LV function improves more than radial midwall function in response to the reduced afterload. Midwall mechanics remain abnormal in 35% of patients and more commonly in women, reflecting persistent LV geometric abnormalities and preoperative comorbidities.

Lead author biography

Dr Lisa M.D. Grymyr obtained her medical degree from the University of Bergen in 2013 and is currently in training in internal medicine and cardiology at Haukeland University Hospital, Bergen, Norway. She is a PhD fellow affiliated with the Bergen Hypertension and Cardiac Dynamics Group at the University of Bergen where she is presently involved in projects on cardiac function in severely obese patients. Her research interests include myocardial deformation analysis by echocardiography and subclinical cardiac dysfunction in obesity and hypertension.

Data availability statement

The data underlying this article will be shared on reasonable request to the corresponding author.

Funding

The FatWest study was funded by grants from the Western Regional Health Authorities and Department of Medicine, Haugesund Hospital. The funding source has not been involved in the study design, in the collection, analysis, and interpretation of data, in the writing of the present article, or in the decision to submit the article for publication.

Conflict of interest: the authors have no conflict of interest.

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