



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

Lisa M.D. Grymyr ^{1,*}, Saied Nadirpour², Eva Gerdts³, Bjørn G. Nedrebø², Johannes Just Hjertaas², Knut Matre¹, and Dana Cramariuc ¹

¹Haukeland University Hospital, Jonas Liesvei 65, 5021 Bergen, Norway; ²Helse Fonna HF, Karmsundgata 120, 5508 Haugesund, Norway; and ³Department of Clinical Science, Center for Research on Cardiac Disease in Women, Jonas Liesvei 65, 5021 Bergen, Norway

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/m², 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 $0.222 \times$ cardiac output \times 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.

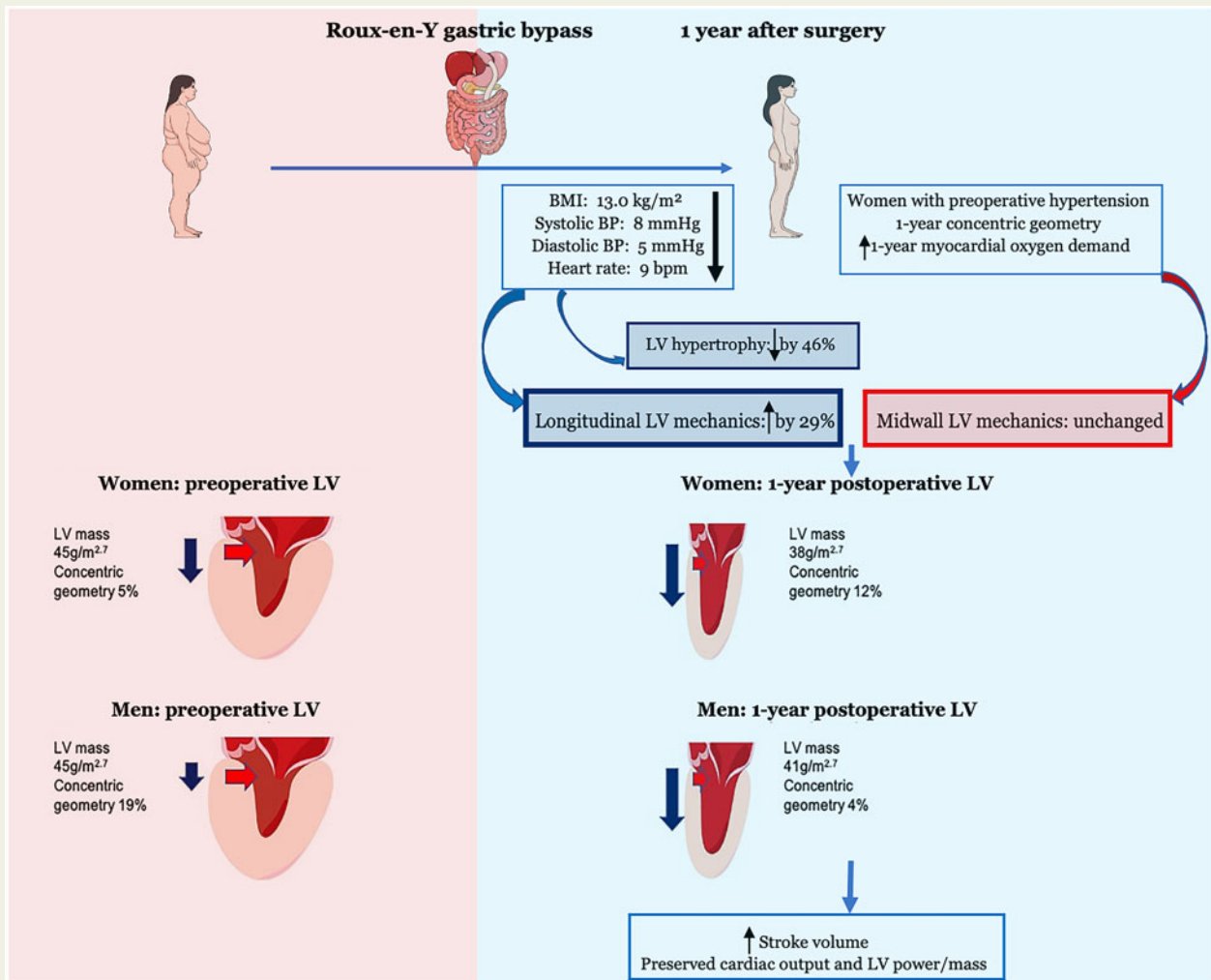
ClinicalTrials.gov NCT01533142, 15 February 2012.
identifier

* Corresponding author. Tel: +47 55972205, Fax: +47 55975150, Email: lisagrymyr@gmail.com

© The Author(s) 2021. Published by Oxford University Press on behalf of the European Society of Cardiology.

This is an Open Access article distributed under the terms of the Creative Commons Attribution-Non-Commercial License (<http://creativecommons.org/licenses/by-nc/4.0/>), which permits non-commercial re-use, distribution, and reproduction in any medium, provided the original work is properly cited. For commercial re-use, please contact journals.permissions@oup.com

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 disease⁴ 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 atrial¹³ and LV function¹⁴ 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) $\geq 140/90$ 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 $80 \times$ 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 \pm 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 (× 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 × s × 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: $0.222 \times \text{cardiac output} \times \text{mean BP/LV mass}$.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.

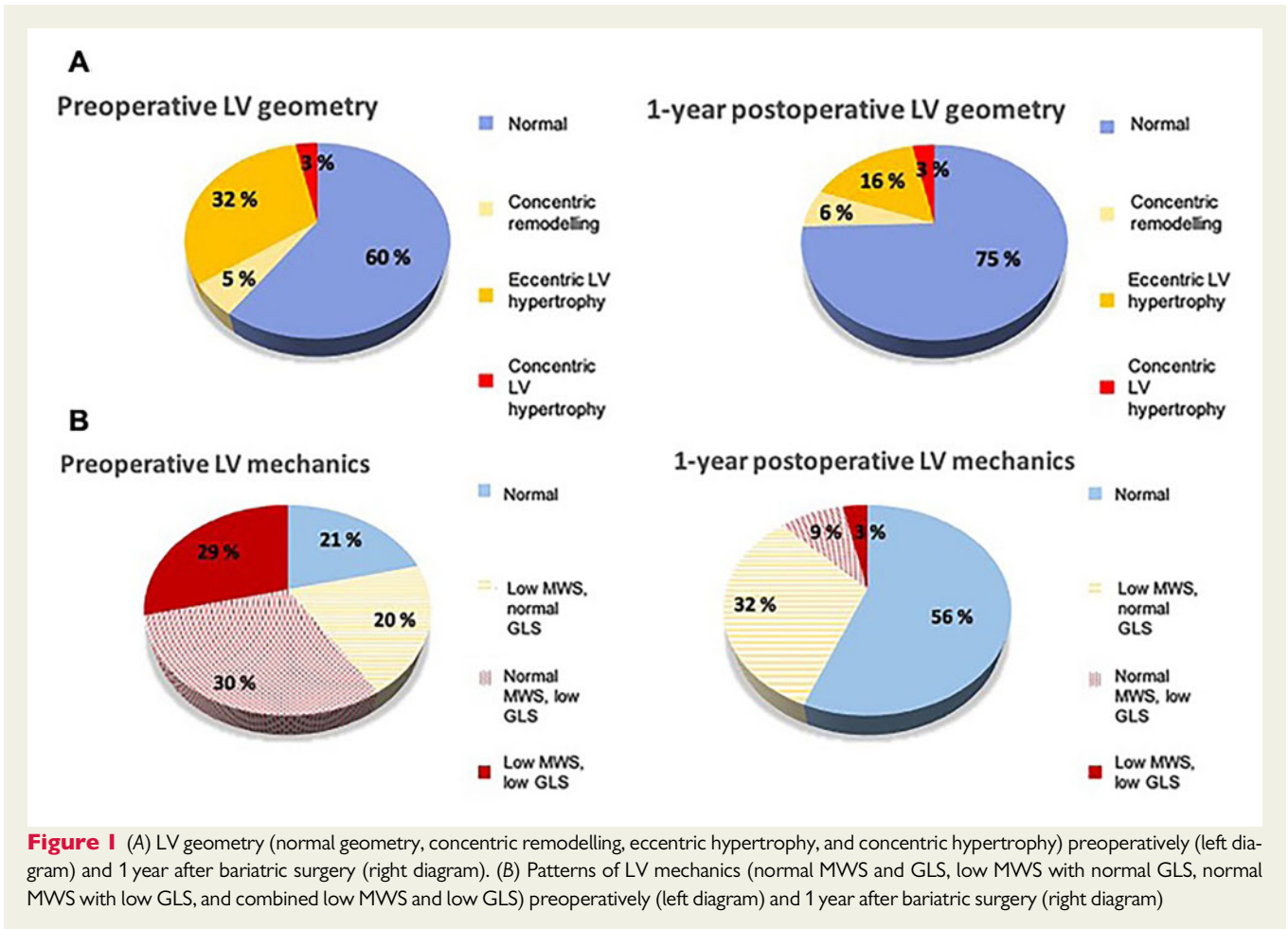
Results

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).



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 ($13.0 \pm 3.8 \text{ kg/m}^2$, i.e. 31% of baseline value in women and 32% in men), mean BP drop ($6 \pm 7 \text{ mmHg}$), and pulse pressure reduction ($4 \pm 7 \text{ mmHg}$) 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 body-weight 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 hypertension²⁹ 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

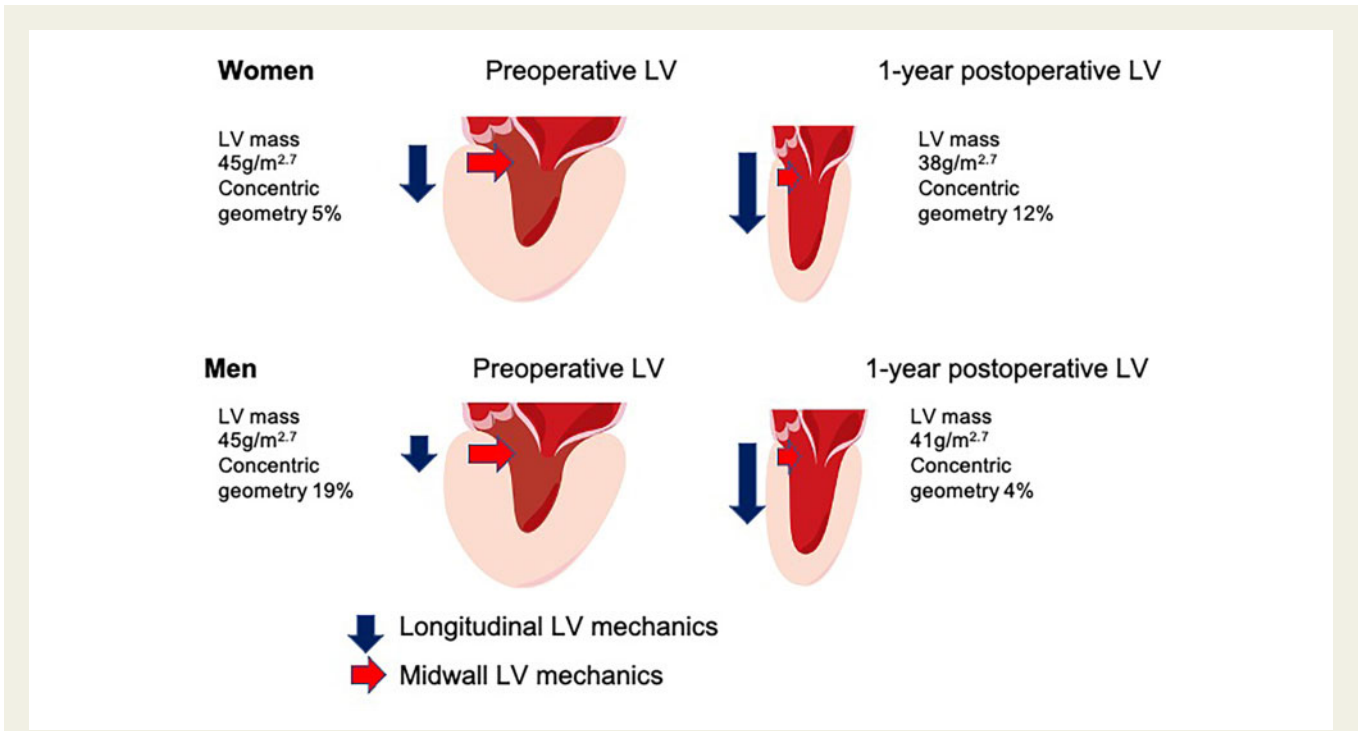


Figure 2 Changes in LV size and mechanics illustrated in women (top) and men (bottom) preoperatively (left illustration) and 1 year after bariatric surgery (right illustration). Changes in LV GLS are depicted with blue arrows, and in LV MWS with red arrows. The length of the arrows illustrates the magnitude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

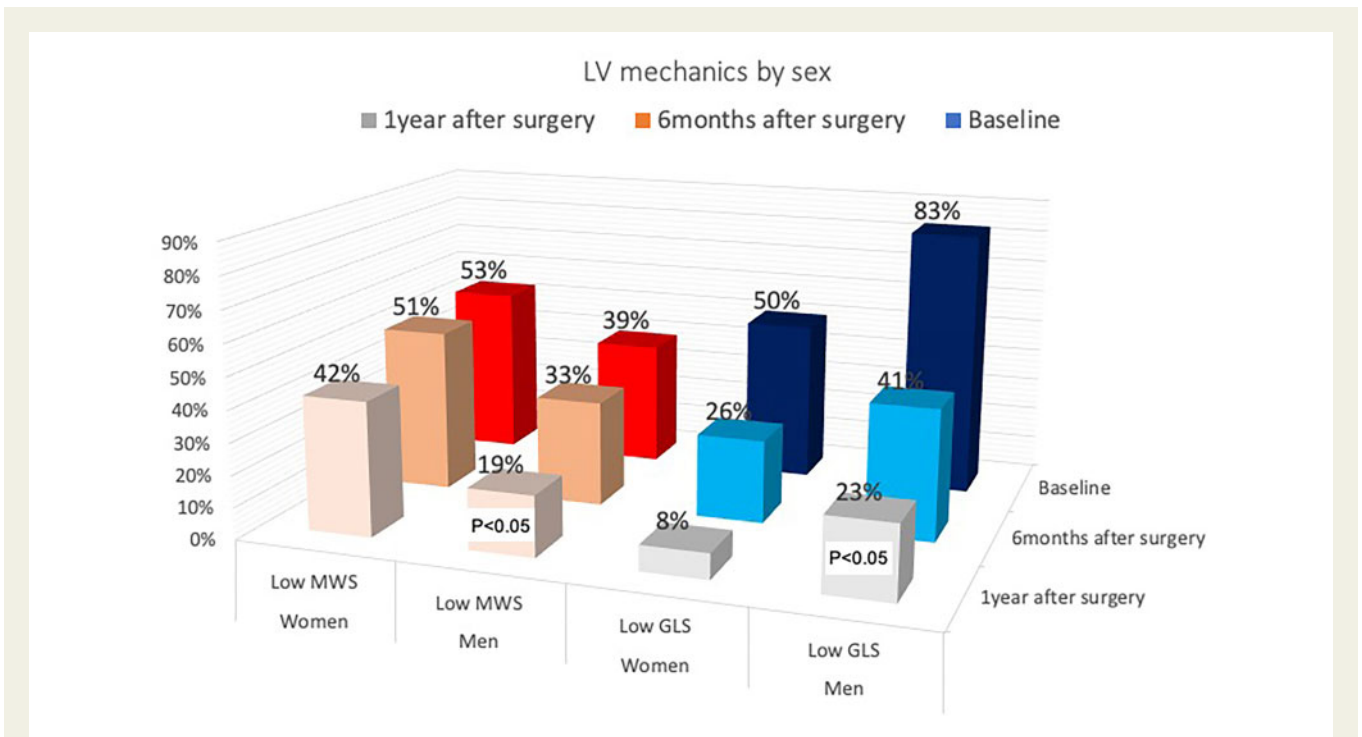


Figure 3 Sex-specific improvement in LV myocardial dysfunction during follow-up. The P-value indicates a significant difference between women and men at the respective visit

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 geometry³⁶ 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 hypertension³⁷ 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.

References

- Palmer MK, Toth PP. Trends in lipids, obesity, metabolic syndrome, and diabetes mellitus in the united states: an NHANES analysis (2003–2004 to 2013–2014). *Obesity (Silver Spring)* 2019;**27**:309–314.

2. Yusuf S, Hawken S, Ôunpuu S, Dans T, Avezum A, Lanas F, McQueen M, Budaj A, Pais P, Varigos J, Lisheng L. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. *Lancet* 2004;**364**:937–952.
3. Flotsos M, Zhao D, Rao VN, Ndumele CE, Guallar E, Burke GL, Vaidya D, Delaney JCA, Michos ED. Body mass index from early-, mid-, and older-adulthood and risk of heart failure and atherosclerotic cardiovascular disease: MESA. *J Am Heart Assoc* 2018;**7**:e009599.
4. Grymyr LMD, Nadirpour S, Gerdtz E, Nedrebø BG, Hjertaas JJ, Matre K, Cramariuc D. Left ventricular myocardial oxygen demand and subclinical dysfunction in patients with severe obesity referred for bariatric surgery. *Nutr Metab Cardiovasc Dis* 2021;**31**:666–674.
5. Stefan N, Birkenfeld AL, Schulte MB. Global pandemics interconnected – obesity, impaired metabolic health and COVID-19. *Nat Rev Endocrinol* 2021;**17**:135–149.
6. Gulliford MC, Charlton J, Prevost T, Booth H, Fildes A, Ashworth M, Littlejohns P, Reddy M, Khan O, Rudisill C. Costs and outcomes of increasing access to bariatric surgery: cohort study and cost-effectiveness analysis using electronic health records. *Value Health* 2017;**20**:85–92.
7. Rubino F, Cohen RV, Mingrone G, Le Roux CW, Mechanick JL, Arterburn DE, Vidal J, Alberti G, Amiel SA, Batterham RL, Bornstein S, Chamseddine G, Del Prato S, Dixon JB, Eckel RH, Hopkins D, McGowan BM, Pan A, Patel A, Pattou F, Schauer PR, Zimmet PZ, Cummings DE. Bariatric and metabolic surgery during and after the COVID-19 pandemic: DSS recommendations for management of surgical candidates and postoperative patients and prioritisation of access to surgery. *Lancet Diabetes Endocrinol* 2020;**8**:640–648.
8. Moussa O, Ardissino M, Heaton T, Tang A, Khan O, Ziprin P, Darzi A, Collins P, Purkayastha S. Effect of bariatric surgery on long-term cardiovascular outcomes: a nationwide nested cohort study. *Eur Heart J* 2020;**41**:2660–2667.
9. Owan T, Avelar E, Morley K, Jiji R, Hall N, Krezowski J, Gallagher J, Williams Z, Preece K, Gundersen N, Strong MB, Pendleton RC, Segerson N, Cloward TV, Walker JM, Farney RJ, Gress RE, Adams TD, Hunt SC, Litwin SE. Favorable changes in cardiac geometry and function following gastric bypass surgery: 2-year follow-up in the Utah obesity study. *J Am Coll Cardiol* 2011;**57**:732–739.
10. Cuspidi C, Rescaldani M, Tadic M, Sala C, Grassi G. Effects of bariatric surgery on cardiac structure and function: a systematic review and meta-analysis. *Am J Hypertens* 2014;**27**:146–156.
11. Aggarwal R, Harling L, Efthimiou E, Darzi A, Athanasiou T, Ashrafian H. The effects of bariatric surgery on cardiac structure and function: a systematic review of cardiac imaging outcomes. *Obes Surg* 2016;**26**:1030–1040.
12. Frea S, Andreis A, Scarlatta V, Rovera C, Vairo A, Pistone E, Anselmino M, Golzio P, Toppino M, Giustetto C, Gaita F. Subclinical left ventricular dysfunction in severe obesity and reverse cardiac remodeling after bariatric surgery. *J Cardiovasc Echogr* 2020;**30**:22–28.
13. Venkateshvaran A, Sarajlic P, Lund LH, Fridén C, Nordgren B, Opava CH, Lundberg IE, Larsson SC, Manouras A, Bäck M. Impaired left atrial dynamics and its improvement by guided physical activity reveal left atrial strain as a novel early indicator of reversible cardiac dysfunction in rheumatoid arthritis. *Eur J Prev Cardiol* 2018;**25**:1106–1108.
14. Sarajlic P, Fridén C, Lund LH, Manouras A, Venkateshvaran A, Larsson SC, Nordgren B, Opava CH, Lundberg IE, Bäck M. Enhanced ventricular-arterial coupling during a 2-year physical activity programme in patients with rheumatoid arthritis: a prospective substudy of the physical activity in rheumatoid arthritis 2010 trial. *J Intern Med* 2018;**284**:664–673.
15. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, Flachskampf FA, Foster E, Goldstein SA, Kuznetsova T, Lancellotti P, Muraru D, Picard MH, Rietzschel ER, Rudski L, Spencer KT, Tsang W, Voigt J-U. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *Eur Heart J Cardiovasc Imaging* 2015;**16**:233–270.
16. de Simone G, Izzo R, Chinali M, De Marco M, Casalnuovo G, Rozza F, Girefoglio D, Iovino GL, Trimarco B, De Luca N. Does information on systolic and diastolic function improve prediction of a cardiovascular event by left ventricular hypertrophy in arterial hypertension? *Hypertension* 2010;**56**:99–104.
17. Palmieri V, de Simone G, Arnett DK, Bella JN, Kitzman DW, Oberman A, Hopkins PN, Province MA, Devereux RB. Relation of various degrees of body mass index in patients with systemic hypertension to left ventricular mass, cardiac output, and peripheral resistance (The Hypertension Genetic Epidemiology Network Study). *Am J Cardiol* 2001;**88**:1163–1168.
18. Cramariuc D, Bahlmann E, Egstrup K, Rossebø AB, Ray S, Kesäniemi YA, Nienaber CA, Gerdtz E. Prognostic impact of impaired left ventricular midwall function during progression of aortic stenosis. *Echocardiography* 2021;**38**:31–38.
19. Sugimoto T, Dulgheru R, Bernard A, Ilardi F, Contu L, Addetta K, Caballero L, Akhaladze N, Athanassopoulos GD, Barone D, Baroni M, Cardim N, Hagedorff A, Hristova K, Lopez T, de la Morena G, Popescu BA, Moonen M, Penicka M, Ozyigit T, Rodrigo Carbonero JD, van de Veire N, von Bardeleben RS, Vinereanu D, Zamorano JL, Go YY, Rosca M, Calin A, Magne J, Cosyns B, Marchetta S, Donal E, Habib G, Galderisi M, Badano LP, Lang RM, Lancellotti P. Echocardiographic reference ranges for normal left ventricular 2D strain: results from the EACVI NORRE study. *Eur Heart J Cardiovasc Imaging* 2017;**18**:833–840.
20. Bella JN, Palmieri V, Roman MJ, Paranicas MF, Welty TK, Lee ET, Fabsitz RR, Howard BV, Devereux RB. Gender differences in left ventricular systolic function in American Indians (from the Strong Heart Study). *Am J Cardiol* 2006;**98**:834–837.
21. Anand V, Kane GC, Scott CG, Pislaru SV, Adigun RO, McCully RB, Pellikka PA, Pislaru C. Prognostic value of peak stress cardiac power in patients with normal ejection fraction undergoing exercise stress echocardiography. *Eur Heart J* 2021;**42**:776–785.
22. Devereux RB, Bang CN, Roman MJ, Palmieri V, Boman K, Gerdtz E, Nieminen MS, Papademetriou V, Wachtell K, Hille DA, Dahlöf B. Left ventricular wall stress-mass-heart rate product and cardiovascular events in treated hypertensive patients: LIFE study. *Hypertension* 2015;**66**:945–953.
23. Bursac Z, Gauss CH, Williams DK, Hosmer DW. Purposeful selection of variables in logistic regression. *Source Code Biol Med* 2008;**3**:17.
24. Jamaly S, Carlsson L, Peltonen M, Jacobson P, Karason K. Surgical obesity treatment and the risk of heart failure. *Eur Heart J* 2019;**40**:2131–2138.
25. Di Bello V, Santini F, Di Cori A, Pucci A, Talini E, Palagi C, Delle Donne MG, Marsili A, Fierabracci P, Valeriano R, Scartabelli G, Giannetti M, Anselmino M, Pinchera A, Mariani M. Effects of bariatric surgery on early myocardial alterations in adult severely obese subjects. *Cardiology* 2008;**109**:241–248.
26. Jhaveri RR, Pond KK, Hauser TH, Kissinger KV, Goepfert L, Schneider B, Jones DB, Manning WJ. Cardiac remodeling after substantial weight loss: a prospective cardiac magnetic resonance study after bariatric surgery. *Surg Obes Relat Dis* 2009;**5**:648–652.
27. Garza CA, Pellikka PA, Somers VK, Sarr MG, Collazo-Clavell ML, Korenfeld Y, Lopez-Jimenez F. Structural and functional changes in left and right ventricles after major weight loss following bariatric surgery for morbid obesity. *Am J Cardiol* 2010;**105**:550–556.
28. Graziani F, Leone AM, Cialdella P, Basile E, Pennestri F, Della Bona R, Iaconelli A, Luzzo G, Biasucci LM, Cardillo MT, Iaconelli A, Guidone C, Nanni G, Mingrone G, Crea F. Effects of bariatric surgery on cardiac remodeling: clinical and pathophysiological implications. *Int J Cardiol* 2013;**168**:4277–4279.
29. de Simone G, Devereux RB, Koren MJ, Mensah GA, Casale PN, Laragh JH. Midwall left ventricular mechanics. An independent predictor of cardiovascular risk in arterial hypertension. *Circulation* 1996;**93**:259–265.
30. Cioffi G, Rossi A, Targher G, Zoppini G, de Simone G, Devereux RB, Bonora E, Vassanelli C. Usefulness of subclinical left ventricular midwall dysfunction to predict cardiovascular mortality in patients with type 2 diabetes mellitus. *Am J Cardiol* 2014;**113**:1409–1414.
31. Gerdtz E, Saeed S, Midtbø H, Rossebø A, Chambers JB, Einarsen E, Bahlmann E, Devereux R. Higher left ventricular mass-wall stress-heart rate product and outcome in aortic valve stenosis. *Heart* 2019;**105**:1629–1633.
32. Ishizu T, Seo Y, Kameda Y, Kawamura R, Kimura T, Shimojo N, Xu D, Murakoshi N, Aonuma K. Left ventricular strain and transmural distribution of structural remodeling in hypertensive heart disease. *Hypertension* 2014;**63**:500–506.
33. Litwin SE, Adams TD, Davidson LE, McKinlay R, Simper SC, Ranson L, Hunt SC. Longitudinal changes in cardiac structure and function in severe obesity: 11-year follow-up in the Utah Obesity Study. *J Am Heart Assoc* 2020;**9**:e014542.
34. Stokke TM, Hasselberg NE, Smedsrud MK, Sarvari SI, Haugaa KH, Smiseth OA, Edvardsen T, Remme EW. Geometry as a confounder when assessing ventricular systolic function: comparison between ejection fraction and strain. *J Am Coll Cardiol* 2017;**70**:942–954.
35. Eschalier R, Rossignol P, Kearney-Schwartz A, Adamopoulos C, Karatzidou K, Fay R, Mandry D, Marie P-Y, Zannad F. Features of cardiac remodeling, associated with blood pressure and fibrosis biomarkers, are frequent in subjects with abdominal obesity. *Hypertension* 2014;**63**:740–746.
36. Baltabaeva A, Marciniak M, Bijnsen B, Mogridge J, He FJ, Antonios TF, MacGregor GA, Sutherland GR. Regional left ventricular deformation and geometry analysis provides insights in myocardial remodelling in mild to moderate hypertension. *Eur J Echocardiogr* 2008;**9**:501–508.
37. Gerdtz E, Zabalgaitia M, Björnstad H, Svendsen TL, Devereux RB. Gender differences in systolic left ventricular function in hypertensive patients with electrocardiographic left ventricular hypertrophy (the LIFE study). *Am J Cardiol* 2001;**87**:980–983; A984.

38. Cramariuc D, Rieck ÅE, Staal EM, Wachtell K, Eriksen E, Rossebø AB, Gerds E. Factors influencing left ventricular structure and stress-corrected systolic function in men and women with asymptomatic aortic valve stenosis (a SEAS sub-study). *Am J Cardiol* 2008;**101**:510–515.
39. Biobaku F, Ghanim H, Monte SV, Caruana JA, Dandona P. Bariatric surgery: remission of inflammation, cardiometabolic benefits, and common adverse effects. *J Endocr Soc* 2020;**4**:bvaa049.
40. Halland H, Matre K, Einarsen E, Midtbø H, Saeed S, Pristaj N, Lønnebakken MT, Gerds E. Effect of fitness on cardiac structure and function in overweight and obesity (the FATCOR study). *Nutr Metab Cardiovasc Dis* 2019;**29**:710–717.
41. King WC, Hsu JY, Belle SH, Courcoulas AP, Eid GM, Flum DR, Mitchell JE, Pender JR, Smith MD, Steffen KJ, Wolfe BM. Pre- to postoperative changes in physical activity: report from the longitudinal assessment of bariatric surgery-2 (LABS-2). *Surg Obes Relat Dis* 2012;**8**:522–532.