Obesity, particularly severe obesity, produces a variety of alterations of cardiac performance and morphology that may lead to the development of heart failure.1-9 Excess adipose accumulation in association with increased lean body mass produces an increase in central blood volume.1-4,8-9 In patients with normotensive obesity, this predisposes to an increase in cardiac output which is facilitated by a decrease in systemic vascular resistance.1,4 Because heart rate changes little if at all with obesity, the increase in cardiac output is predominantly caused by augmentation of left ventricular (LV) stroke volume.1-4 In many obese individuals, the high cardiac output state is associated with LV dilatation, a response that may lead to eccentric LV hypertrophy (LVH).1-5,7,8 However, recent studies have suggested that concentric LVH or remodeling occurs as often or perhaps more often than eccentric LVH in obese subjects.10-12 Factors that may predispose to concentric LVH or remodeling in such individuals include the presence of systemic hypertension, increased sympathetic activity, stimulation of the renin–angiotensin–aldosterone system, insulin resistance with hyperinsulinemia (which may predispose to release of insulin-related growth factors), hyperleptinemia, and possibly, lipotoxicity and lipoapoptosis.1-5,12,13 Because LVH is a key morphological alteration in obesity, the predominant functional change is LV diastolic dysfunction, manifested as elevated LV end-diastolic pressure (particularly during exercise) and impaired LV diastolic filling on noninvasive cardiac imaging.1,5,7,8 LV systolic dysfunction (LVSD) is relatively uncommon in obesity. LV ejection phase indices are usually normal or supranormal, and when abnormal, they are usually mildly reduced.1-5,7,8,14 LVSD in uncomplicated obesity may be related in part to inadequate LVH leading to persistently high LV wall stress.1,2,14 Recent studies have reported decreased mitral annular velocities in systole on tissue Doppler imaging and abnormal LV deformation in systole on strain imaging.1,2,16,17 LVSD has also been reported with several murine models of lipotoxicity.1,2,4,11,14 In this issue of Circulation: Heart Failure, Vest et al18 indicate that leptin, resistin, adiponectin, glucagon-like peptide, and glucose-dependent insulinotropic polypeptide may affect the relation between obesity and LV systolic function. The presence of moderate to severe LVSD in patients with obesity should elicit a search for comorbidities, such as coronary artery disease.1,2 The aforementioned alterations in cardiac performance and morphology may predispose to LV failure, which in association with pulmonary arterial hypertension from sleep apnea and obesity hypventilation may contribute to right ventricular failure in patients with severe obesity.1,4

Obesity has been identified in epidemiological studies as a risk factor for the development of heart failure in all classes of obesity.1-6,8 It may serve as the sole or predominant cause of heart failure in patients with severe obesity, a condition often referred to as obesity cardiomyopathy.1,2,4 Substantial weight loss is capable of reversing many of the changes in cardiac structure and function associated with severe obesity.1-5,7,8,19-21 Bariatric surgery has been more successful than diet ± exercise or pharmacotherapy in achieving regression of LVH and improvement in LV diastolic filling and systolic function (when impaired).1-5,19,24 This presumably is related to greater weight loss which contributes to favorable changes in hemodynamics and LV loading conditions, a more favorable metabolic milieu, and improvement in sleep-disorder breathing.1-5,7,16-22 In addition, several small studies have demonstrated that substantial weight loss from bariatric surgery is capable of reversing clinical manifestations, improving New York Heart Association functional class, and enhancing quality of life in severely obese subjects with heart failure.1,5,23,24

Vest et al18 present the results of the largest study to date of the effects of bariatric surgery on LVSD in patients with severe obesity. They defined LVSD as LV ejection fraction (LVEF) <50% on a transthoracic echocardiogram. The main focus of this retrospective analysis was a cohort of 42 patients with severe obesity treated with various types of bariatric surgery (most commonly Roux-en-Y gastric bypass). Thirty-eight of these subjects underwent transthoracic echocardiography before surgery and after substantial postoperative weight loss. The group of 42 patients with LVSD was compared with a cohort of 2588 patients with no known preoperative diagnosis of LVSD. It is unclear whether all these control patients had normal LV systolic function or whether LVEF was not assessed in all subjects. A wide variety of demographic, clinical, perioperative, and postoperative outcomes were evaluated.

The opinions expressed in this article are not necessarily those of the editors or of the American Heart Association.

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Control patients were younger, more commonly women and, in general, had fewer comorbidities than patients with LVSD. Major differences in outcomes between the 2 groups included a higher incidence of postoperative heart failure in those with LVSD (10% versus 0.2%; \( P<0.001 \); 4 in each group) and a higher incidence of new myocardial infarction in the cohort with LVSD (2% versus 0.04%; \( P=0.032 \); 1 in each group). However, there were no significant differences in mortality at 30 days, 6 or 12 months, and no deaths in the LVSD cohort. Similarly, there were no significant differences in the incidence of other cardiovascular, noncardiovascular, and postoperative complications or in hospital re-admission at 30 days.

The cohort of 38 patients who underwent transthoracic echocardiography before and after bariatric surgery was compared with a nonsurgical control group of 38 patients who underwent transthoracic echocardiography at similar time intervals. The nonsurgical group was less severely obese than the surgical group, but was otherwise well-matched for clinical characteristics and preoperative LVEF. The main end point in this subanalysis was change in LVEF. In those undergoing bariatric surgery, mean LVEF increased significantly (+5.21±8.3%; \( P=0.0005 \)). In the nonsurgical control group, mean LVEF increased to a lesser extent (+3.4±10.5%; \( P=0.056 \)). When comparing those whose LVEF increased >10% with those whose LVEF increased <10%, there were no significant differences in baseline characteristics. However, linear regression analysis identified initial LVEF and history of preoperative myocardial infarction as predictors of postoperative LVEF. This may explain why the overall increase in mean LVEF after bariatric surgery in those with LVSD was less robust than that observed in most previous studies.

Although the results of this study have expanded our knowledge of the effects of bariatric surgery and subsequent substantial weight loss on LV systolic function in severely obese patients with LVSD before surgery, limitations exist as is commonly the case with retrospective analyses. The main focus of this study was patients with LVSD, but it is not clear whether and to what extent heart failure was present in this group before surgery. Heart failure independently predicts increased mortality risk at 1 year after bariatric surgery.25 In addition, the authors did not provide information relating to LV morphology, diastolic function, or duration of obesity, factors that may influence the development of heart failure and prognosis.1–5,26,27 The small number of postoperative major adverse outcomes (heart failure, myocardial infarction and mortality) is both reassuring and concerning. On the basis of the results of this study, bariatric surgery in severely obese patients with LVSD seems to be reasonably safe from a cardiovascular standpoint. However, the low number of events and small sample size of the cohort with LVSD raise the question of a type 2 error for some of the outcomes, particularly mortality.

Several previous studies of bariatric surgery in patients with LVSD have included patients with prior myocardial infarction, but did not analyze its effect on outcomes.18,22 The fact that a history of myocardial infarction predicted a less robust increase in LVEF after bariatric surgery is an important observation.

Other strengths of this study include enrollment of older patients who are not usually selected for studies of this type and the fact that the percentages of women and men in the LVSD cohort were equal. Many previous investigations of the effects of weight loss from bariatric surgery on LVSD used study populations that consisted of younger patients and were predominantly composed of either women or men.

In summary, the results of the study by Vest et al seem to support their conclusion that bariatric surgery is effective and reasonably safe in severely obese patients with LVSD, although not without some degree of risk. Future studies should ideally be prospective and should strive for enrollment of larger numbers of patients with LVSD. They should characterize preoperative heart failure status, including neurohormonal alterations, and define cardiac morphology, LV diastolic function, and duration of obesity in addition to LV systolic function. A multicenter study may be required to accomplish these goals. It is likely that patients with severe obesity comprise a heterogeneous population from both a clinical and echocardiographic perspective. The observations by Vest et al represent an advance in our understanding of outcomes after bariatric surgery in those with LVSD. Whether intentional weight loss from bariatric surgery or any other weight loss modality reduces mortality in other groups of patients with severe obesity, and particularly in those with heart failure, remains to be determined.

Disclosures

None.

References


Bariatric Surgery in Patients With Left Ventricular Systolic Dysfunction: Effective, But Is It Safe?
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Circ Heart Fail. 2016;9:
doi: 10.1161/CIRCHEARTFAILURE.116.002960
Circulation: Heart Failure is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 1941-3289. Online ISSN: 1941-3297

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circheartfailure.ahajournals.org/content/9/3/e002960

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