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Bariatric Surgery as a Treatment for Heart Failure: Review of the Literature and Potential Mechanisms

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Introduction

Obesity cardiomyopathy is heart failure due primarily to the underlying metabolic disease of severe obesity and is separate from ischemic, familial, hypertensive or diabetic heart failure. The spectrum of obesity cardiomyopathy continues from asymptomatic diastolic dysfunction to end-stage left ventricular (LV) dilation with reduced systolic function [(1)]. Obesity cardiomyopathy may comprise more than 50% of heart failure diagnoses in severely obese individuals [(2,3)]. The risk of heart failure increases 30–100% in obese individuals [(1)]. The duration of morbid obesity increases the risk of heart failure development and is positively associated with higher LV mass, impaired diastolic and systolic function [(4)]. Bariatric surgery significantly improves cardiac geometry, function and symptoms related to obesity cardiomyopathy. The mechanisms for the improvement of cardiac failure after bariatric surgery are unknown but likely include the effect of significant body mass reduction on cardiac work load, inflammation, and metabolism as well as positive weight-loss independent alterations in the entero-cardiac axis.

Obesity Cardiomyopathy Pathogenesis

The relationship between obesity and heart failure development is complex involving multiple different pathways that ultimately affect cardiac function. Severe obesity not only affects cardiac hemodynamics due to changes in systemic mass and blood volume but cardiac structure and function is also affected by the deranged metabolic environment of obesity which includes hyperleptinemia, hyperinsulinemia and activation of the renin-angiotensin system [(5,6)]. These changes may include increased LV mass, LV hypertrophy, left atrial enlargement, as well as the development of diastolic and systolic dysfunction [(4)]. The increased metabolic demands of morbid obesity switches myocyte substrate utilization from fatty acids to glucose. When this occurs in the setting of innate insulin resistance (which exists independent of systemic type 2 diabetes mellitus), the cardiac myocyte is

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unable to uptake glucose effectively. This impairment in carbohydrate utilization with inadequate fatty acid β-oxidation leads to mitochondrial dysfunction, reactive oxygen species generation, and calcium mishandling ultimately resulting in myocardial dysfunction. Fatty infiltration or cardiac steatosis within the myocardium also leads to cardiac fibrosis and impairment of cardiac function [7]. Morbid obesity often co-exists with co-morbidities of type 2 diabetes mellitus, hypertension, obstructive sleep apnea, metabolic syndrome and arteriosclerosis which all increase the risk of cardiac dysfunction [1,8]. Obesity hypoventilation syndrome (OHS), a disease that develops due to the excessive mechanical load of obesity coupled with awake hypercapnia, and sleep-disordered breathing, is frequently associated with pulmonary hypertension and heart failure [9]. In conclusion, the direct and indirect effects of severe obesity results in left ventricular hypertrophy and dilation with cardiac failure causing maladaptive left ventricular remodeling and progression to cardiomyopathy [10].

Weight reduction is the only effective long-term treatment for obesity cardiomyopathy [1]. Weight loss is associated with decreases in left and right ventricular mass, end-diastolic volume and diastolic dysfunction and increases aortic distensibility [6]. There are minimal studies comparing the effectiveness of substantial purposeful weight loss by exercise/caloric restriction in morbidly obese patients due to the lack of efficacy of long-term dieting in this patient population. Alexander et al studied 9 severely obese patients who lost 39–84 kg with dieting over a period of 4–34 months. While cardiac output, stroke volume, and systemic arterial pressure reduced with weight loss through dieting, myocardial hypertrophy and reduced ventricular compliance persisted [11]. Modest weight loss through hypocaloric diets decreases left ventricular mass with conflicting changes in the literature of any positive effects on systolic or diastolic function [12,13].

Bariatric Surgery as a Treatment for Obesity Cardiomyopathy

There are reports back to the era of the jejunoileal bypass on the positive effects of surgical weight loss on cardiac structure [14]. Roux-en-Y gastric bypass (RYGB) surgery decreases the incidence of new heart failure development compared to intensive lifestyle modification by almost 50% (hazard ration 0.54, 95% CI 0.36–0.820) [15]. RYGB improves predictors of future cardiovascular morbidity in adolescents undergoing surgery with significant improvements in left ventricular hypertrophy, diastolic dysfunction and cardiac workload [16].

The preventative effect on the development of heart failure likely lies within the substantial effect of surgery on cardiac mass and diastolic function. Post-surgical cardiac changes include significant improvements in cardiac geometry and function [10]. Obese subjects without functional disease who are treated with bariatric surgery have a significant reduction in relative wall thickness, left ventricular mass and blood pressure [17]. In morbidly obese women, substantial weight loss reduced not just left ventricular but also right ventricular mass as measured by cardiac magnetic resonance imaging independent of reduction in blood pressure [18]. Cuspidi et al published a systemic review of 23 studies examining the effect of bariatric surgery on cardiac structure and function. In patients with preserved systolic function, bariatric surgery induces significant reductions in absolute and relative left

ventricular mass and wall thickness and improved diastolic function [19]. A recent systematic review also demonstrated a statistically significant weighted proportional reduction in left ventricular mass with an improvement in left ventricular hypertrophy [10].

There are multiple reports of improved diastolic dysfunction in patients with preserved ejection fraction after bariatric surgery [10,20,21]. Leichman et al found that bariatric surgery normalized left ventricular relaxation impairment by 9 months post-operatively [20]. Table 1 is a review of the currently available publications on the effect of weight loss surgery on diastolic function in morbidly obese patients. There is almost a universally reported increase in post-operative E/A ratio with significant reductions in intraventricular relaxation time. There is a less consistent effect of bariatric surgery on other metrics of diastolic function including e′, E/e′, left atrial diameter and deceleration time. Of note, these studies were concluded prior to the 2016 update of the American Society of Echocardiography guidelines to determine diastolic function [40].

Bariatric surgery significantly decreases hypercarbia and improves arterial oxygen concentrations in OHS patients with an improvement in pulmonary hypertension and left ventricular filling pressures [9]. Significant weight loss improves OHS likely in part due to the reduction of central obesity. Central obesity with increased intra-abdominal pressure restricts diaphragm excursion and allows for inefficient respiratory patterns which results in impaired lung volumes, respiratory muscle performance and compliance in OHS patients [41]. After bariatric surgery, the significant reduction in waist circumference, central obesity and thus intra-abdominal pressure significantly correlates to improvement in pulmonary function mechanics [42].

Bariatric surgery can significantly improve systolic function in patients with heart failure. In 2008, Ramani published the results of 12 patients with markedly depressed LV ejection fraction (EF) in 12 patients undergoing bariatric surgery. Unlikely matched morbidly obese controls, bariatric surgery patients had a significant improvement in left ventricular ejection fraction from 21.7% pre-op to 35% post-operatively [43]. Vest et al found a more modest although significant improvement in LVEF at 6 months after surgery in patients with LV systolic dysfunction (+5.1% increase in LVEF post-op) [44]. Due to vast patient heterogeneity, small sample size, different surgical procedures applied, and lack of control groups for many studies, there has been markedly variability in the reported improvement in post-operative systolic function after bariatric surgery. In a meta-analysis, Aggarwal et al found a modest yet significant improvement in LVEF after surgery. Improvements in systolic function primarily occurs only in patients with depressed pre-operative function with non-ischemic cardiomyopathy and long periods of obesity [4,10,45].

Bariatric surgery is also associated with a decline in the rate of heart failure exacerbations requiring emergency department evaluation or hospitalization among obese patients with heart failure [46]. Bariatric surgery improves quality of life in heart failure patients compared to non-operatively managed heart failure patients. There is a significant reduction in the average NHYA class after bariatric surgery from 2.9 to 2.3 compared to a significant worsening in control patients with heart failure (NYHA class 2.4 to 3.3, p=0.02) [43].

Bariatric surgery patients also have marked improvement in frequency of exertional dyspnea unlike control patients [47].

Bariatric Surgery as a Bridge to Transplantation

Morbidly obese patients with severe heart failure are ineligible or endure long donor wait-times for cardiac transplantation due to organ-specific weight and BMI limitations [48]. For advanced heart failure patients including those requiring mechanical circulatory support, bariatric surgery has been successfully used for weight loss as a bridge to cardiac transplantation [49–51]. In a case series of six patients including 3 with a pre-operative left ventricular assist device, at 12 months after bariatric surgery all patients met listing criteria for cardiac transplant [48]. Patients with left ventricular systolic failure may have a reduced post-surgical weight loss compared to patients with normal systolic function [44]. Patients with systolic failure are significantly more likely to have a complication of heart failure decompensation (10% vs 0.2%) and myocardial infarction (2% vs 0.04%) compared to non-heart failure patients but have no difference in 30 day, 6 month or 1-year mortality [44]. Bariatric programs committed to managing these complex patients should incorporate a multi-disciplinary team approach with surgery, endocrinology, pulmonary, cardiac anesthesia and advanced heart failure specialists with particular attention to the aggressive pre-operative management of obesity-associated co-morbidities including obstructive sleep apnea, pulmonary hypertension, type 2 diabetes mellitus, hypervolemia and cardiopulmonary deconditioning. In conclusion, in high volume bariatric centers with the hospital infrastructure to support pre-operative cardiac optimization and intense cardiac support peri-operatively, bariatric surgery can be performed safely with successful progression to cardiac transplant listing and transplantation for patients with end-stage heart failure.

Potential Mechanisms for Cardiac Improvement after Bariatric Surgery

The mechanisms for the improvement of cardiac failure after bariatric surgery are unknown but likely involve a combination of multiple beneficial mechanisms. These mechanisms may include the effect of significant body mass reduction on cardiac work load and metabolism and beneficial alterations in the entero-cardiac axis. First of all, the significant reduction in adipose tissue and mass with surgical weight loss decreases both preload and afterload as well as systemic and local energy demands. Myocardial oxygen consumption significantly decreases with weight loss and correlates to improved diastolic function [52]. Lin et al studied 30 obese subjects with positron emission tomography both before and after gastric bypass or moderate weight loss induced by dieting. Myocardial fatty acid utilization and oxidation decreased only after surgery [53]. The reduction in myocardial fatty acid utilization after surgery is coupled with significant post-operative improvements in insulin resistance. As measured by an intravenous glucose tolerance test, gastric bypass significantly increased insulin sensitivity and disposition index, decreased the acute insulin response to glucose with improved β-cell function [53]. Improvements in heart rate variability after bariatric surgery are also associated to improvements in insulin resistance [54].

Patients undergoing gastric bypass or sleeve gastrectomy have an increases in both cellular and tissue mitochondrial respiration compared to pre-operatively [55]. Specifically, skeletal muscle mitochondrial function improves to the level of lean patients by one year after weight loss surgery [56]. There is a reduction in systemic free fatty acids and consequently decreased skeletal muscle triglyceride deposition after surgery representing decreased lipotoxicity [20]. Although these findings are likely applicable to cardiac muscle and function, there are minimal clinical studies available on specific post-operative improvements in cardiac mitochondrial function from patients undergoing bariatric surgery.

There is a precedent for critical weight loss-independent effects of bariatric surgery on metabolic disease including type 2 diabetes mellitus. Other beneficial effects of bariatric surgery include alteration of the gastrointestinal microbiome, increases in the post-prandial bile acid pool, and increases in post-prandial glucagon-like peptide-1 (GLP-1) and receptor signaling within days after surgery [57,58]. It is possible that gastrointestinal manipulation by surgery beneficially alters the entero-cardiac axis through one of these mechanisms providing additional improvement in cardiac function beyond the impact of weight loss. We recently published that in a rodent of model of sleeve gastrectomy and diet-induced cardiac dysfunction, sleeve gastrectomy significantly improved systolic function in 44% of rats in a weight-loss independent manner [59]. Rats with improved systolic function also had significantly smaller left ventricular internal diameter in systole and end systolic volume without differences in diastolic function. These findings support that bariatric surgery alters the entero-cardiac axis in a weight-loss independent manner with the potential for reverse cardiac remodeling in addition the known beneficial impact of weight loss.

The increase in post-operative GLP-1 is an attractive mechanistic target to mediate an effect of bariatric surgery on cardiac function. GLP-1 is secreted from enteroendocrine L cells primarily in the distal small intestine and colon. GLP-1 increases glucose-dependent insulin secretion, decreases glucagon levels, decreases gastric emptying and suppresses appetite [60]. The GLP-1R is a G protein-coupled receptor located on β-cells of the pancreas, lung, central and peripheral nervous system, blood vessels, and the heart of rodents and humans [60]. GLP-1R signaling in the cardiomyocyte leads to a reduction in apoptosis and increase in glucose uptake independent of the classical insulin pathway [61]. GLP-1R agonists which mimic GLP-1 action of the entero-cardiac axis have been used for the treatment of ischemia-reperfusion injury and heart failure [62–64]. Future mechanistic studies are needed to determine how the entero-cardiac axis is altered after bariatric surgery to mediate weight-loss independent improvements in cardiac function.

Conclusions

Obesity cardiomyopathy is a morbid disease affecting cardiac geometry and diastolic and systolic function. Bariatric surgery reduces the risk of heart failure development and reverses abnormalities in cardiac mass, workload and metabolism with improved diastolic function, and potentially enhancing native cardiac systolic function. Most studies of patients with heart failure and bariatric surgery have focused on patient outcomes and not the mechanisms for cardiac recovery. Future studies aimed at understanding the mechanisms for cardiac recovery after bariatric surgery will allow for the development of novel surgical and non-

surgical therapies for heart failure which replicate the beneficial metabolic mechanisms generated from the entero-cardiac axis in additional to substantial weight loss.

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Table 1

Effect of bariatric surgery on diastolic function.

Surgery type	(n)	BMI loss (kg/m^2)	FU (mo)	LAD (mm)	e′ (m/s)	E/A	E/e′	DT	IVRT
RYGB [22]	37	11.8	15.6	NA	↑	↑	↓	↔	NA
Unspecified [23]	32	NA	6	↔	NA	↑	↑	↓	NA
SG [24]	8	9	9	NA	↑	↔	↓	NA	NA
RYGB [25]	34	15.6	12	NA	NA	↑	NA	NA	NA
AGB [26]	83	6.8	12	NA	↔	↔	↔	↓	↔
SG [27]	34	14.7	12	NA	↑	↑	↓	↓	NA
RYGB, BPD, AGB [28]	28	14	22.7	NA	NA	↔	↔	↓	NA
RYGB, SG [29]	52	10.9	6	↓	NA	↑	↔	↑	↓
RYGB [30]	9	7.2	4	NA	NA	↔	↔	NA	NA
Unspecified [31]	51	12.2	24	↔	NA	↑	↔	↔	NA
Unspecified [32]	41	17.0	12	NA	↓	↑	↑	↑	↔
RYGB, SG [33]	66	9.2	3	↓	↑	↑	↓	NA	↓
RYGB [16]	38	20	10	↔	↑	↑	↓	NA	NA
Unspecified [34]	13	11	6–24	↓	NA	↑	NA	NA	↓
DS [35]	10	14.6	6.8	↔	NA	↔	NA	NA	↔
RYGB [21]	60	16.7	36	↓	NA	↔	NA	↓	↓
RYGB, AGB [20]	22	6.7	3	NA	NA	↔	NA	↔	NA
RYGB [36]	23	17.0	36	↔	NA	↑	NA	NA	↓
RYGB [37]	17	14	7.6	↔	NA	↑	NA	↔	NA
VBG [38]	16	15	6	NA	NA	↑	NA	NA	↓
VBG [39]	41	10	12	NA	NA	↑	NA	NA	↓

RYGB: Roux-en-Y gastric bypass; SG: sleeve gastrectomy; AGB: adjustable gastric band; VGB: vertical banded gastroplasty; DS: Duodenal Switch; e′: peak mitral annular velocity during early filling; E/A: the peak early (E) divided by late (A) mitral flow velocity; DT: deceleration time; IVRT: intraventricular relaxation time; LAD: left atrial diameter; NA: data not available