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Reductions in Cardiovascular Risk After Bariatric Surgery

Fethi Benraoune[1] and **Sheldon E. Litwin**[1]

[1]Cardiology Division, Georgia Health Sciences University, Augusta, GA

Abstract

Purpose of review—Obesity is commonly associated with multiple conditions imparting adverse cardiovascular risk including, hypertension, dyslipidemia and insulin resistance or diabetes. In addition, sleep disordered breathing, inflammation, left ventricular hypertrophy, left atrial enlargement and subclinical left ventricular systolic and diastolic dysfunction may collectively contribute to increased cardiovascular morbidity and mortality. This review will describe improvements in cardiovascular risk factors after bariatric surgery.

Recent findings—All of the cardiovascular risk factors listed above are improved or even resolved after bariatric surgery. Cardiac structure and function also have shown consistent improvement after surgically-induced weight loss. The amount of improvement in cardiac risk factors is generally proportional to the amount of weight lost. The degree of weight loss varies with different bariatric procedures. Based on the improvement in risk profiles, it has been predicted that progression of atherosclerosis could be slowed and the 10 year risk of cardiac events would decline by ~ 50% in patients undergoing weight loss surgery. In keeping with these predictions, 2 studies have demonstrated reductions in 10-year total and cardiovascular mortality of approximately 50% in patients who had bariatric surgery.

Summary—These encouraging data support the continued, and perhaps expanded use of surgical procedures to induce weight loss in severely obese patients.

Keywords

obesity; cardiovascular risk factors; hypertension; diabetes; dyslipidemia; bariatric surgery; weight loss

Introduction

Large population studies have shown that obesity is associated with increased long-term mortality.(1–3) The hazard ratios for mortality in severely obese subjects (BMI > 35 kg/m^2) are typically increased 2.0–2.5 fold compared to normal weight individuals. Cardiovascular disease and cancer are responsible for much of the excess mortality.(4) This review will focus on the data supporting the use of bariatric surgery as a means to reduce cardiovascular risk in severely obese individuals.

Address for correspondence: Sheldon E. Litwin, Chief, Cardiology Division, Georgia Health Sciences University, 1120 15th St, BBR 6513, Augusta, GA 30912, slitwin@georgiahealth.edu.

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Association of obesity with cardiovascular risk factors

Cardiovascular ailments associated with obesity include early and accelerated coronary atherosclerosis(5), myocardial infarction or acute coronary syndrome(5, 6), congestive heart failure(7), stroke(8, 9), atrial fibrillation(10) and sudden cardiac death.(11)

The frequent presence of coronary risk factors in obese subjects explains at least part of the surplus cardiovascular disease. Conventional cardiac risk factors that are commonly associated with obesity include hypertension, diabetes and dyslipidemia (particularly low HDL and high triglycerides). In addition, insulin resistance, sleep disordered breathing and systemic inflammation, are all potential contributors to the cardiovascular risk profile in obesity. Lastly, there appear to be some undefined factors associated with obesity that play a role in coronary atherosclerosis and cardiovascular risk, independent of traditional atherosclerotic risk factors.(12)

Coronary arterial calcification (CAC) is a highly specific marker of coronary atherosclerosis. Obese subjects are more likely to have CAC than nonobese subjects.(12) Moreover, obesity is a risk factor for more rapid CAC progression.(13) Obesity is associated with other markers of vascular disease such as increased carotid intima-medial thickness(14, 15) and impaired endothelial-dependent vasodilation.(16)

Cardiac structure and function in obesity

In addition to coronary atherosclerosis, obesity is associated with adverse structural changes in the heart including left ventricular (LV) hypertrophy.(14, 17–23) The majority of published works have shown increases in both LV cavity size and wall thickness, with a relatively greater increase in the latter.(14, 17, 24–28) These geometric changes result in a predominance of obese patients with concentric LV remodeling or concentric hypertrophy, the severity of which increases in proportion to BMI.(17, 24) In addition to the degree of obesity, the presence and severity of hypertension and the degree of nocturnal hypoxemia appear to contribute to LV hypertrophy.(17, 26)Left ventricular hypertrophy is a well-validated marker of increased mortality.(29) It is unknown whether the presence of LV hypertrophy directly contributes to the high cardiovascular mortality that occurs in obese people.

Although the LV ejection fraction has generally been found to be normal in obese patients(17, 24, 30, 31), many groups of investigators have found evidence of subclinical myocardial dysfunction when sensitive measures such as LV midwall fractional shortening, tissue Doppler or strain measurements are used. (14, 17, 23, 25, 27, 32, 33) Most often, mild abnormalities of both systolic and diastolic function have been present by tissue Doppler and strain imaging. Interestingly, the concept of a “cardiomyopathy of obesity” characterized by progressive LV remodeling or overt systolic dysfunction has not been supported by recent studies. In one study with more than 5,000 patients undergoing cardiac magnetic resonance imaging, there was no relationship between body mass index and LV ejection fraction.(24) Indeed, there are no longitudinal studies showing that the subclinical abnormalities of systolic and diastolic function progress over time, as would be expected if obesity indeed caused a true myopathic condition.

Left atrial enlargement is known to be a marker of cardiovascular mortality.(34) Left atrial dimensions and volume are increased in obese patients.(25, 35, 36) Findings from the Framingham heart study suggest that over 15 years of follow up, obese patients are more likely to develop incident atrial fibrillation than overweight or nonobese patients.(10) This predilection for developing atrial fibrillation appears to be explained entirely by increased left atrial size.

Effects of bariatric surgery

Severe obesity is generally refractory to lifestyle modification, including diet and exercise. Pharmacological treatment is also of limited efficacy. Even when lifestyle modification or drug treatments are successful, the lost weight is usually completely regained within 1 year.

Amount of weight loss with bariatric surgery

Bariatric surgery is an extremely effective method of producing weight loss. The nadir of weight loss occurs at 1 – 2 years postoperatively.(37) The amount of weight loss depends upon the technique used. Purely restrictive procedures, such as gastric banding, usually produce about 50 lbs of weight loss, or 47% of excess body weight.(37–39) Roux en Y gastric bypass surgery is usually associated with almost 100 of weight loss, or 62% of excess body weight.(38, 39) The most aggressive procedures cause larger amounts of malabsorption. The pancreatic-biliary diversion procedure is associated with the greatest amount of weight loss (70% of excess body weight lost)(39), but this procedure is rarely performed any more. After 1.5–2 years, there is a slow but steady weight regain in the majority of patients. After 5 years, ~ 15 lbs of weight regain (5–10% body weight) has usually occurred with only a small amount of additional weight gain by 10 years.(37)

The two largest and longest, prospective, controlled studies of weight loss surgery are: 1) the Swedish Obese Subjects (SOS) study(37) which included 2010 surgery (mostly gastric banding) and 2037 control subjects, and 2) the Utah Obesity Study which included 420 surgery (all gastric bypass) and 736 control subjects.(38, 40) The SOS study has reported 10 year outcome data(37, 41) while the Utah Obesity Study has reported 2-year follow up data. (17, 38)

Changes in cardiovascular risk factors after weight loss

Weight loss achieved by lifestyle modification, pharmacotherapy and bariatric surgery have all been associated with favorable changes in cardiovascular risk factors. In general, the magnitude of improvement in cardiovascular risk factors and cardiac geometry is proportional to the amount of weight loss, regardless of the mechanism of weight loss (diet vs. surgical). Interestingly, liposuction has not been associated with beneficial effects on the cardiovascular risk profile.(42) Presumably this relates to the fact that liposuction selectively removes subcutaneous, rather than visceral fat.

There are remarkable improvements in cardiovascular risk factors after bariatric surgery (summarized in Table 1 and Table 2).(37, 38, 43–50)

Bariatric surgery generally is associated with significant lowering of systolic blood pressure of 4–15 mmHg at 1–2 years. There are smaller (2–5 mmHg), but usually significant reductions in diastolic blood pressure. Twenty to forty % of subjects undergoing bariatric surgery have complete resolution of hypertension (normal blood pressure without antihypertensive medication) at 2 years (Table 2).(37, 51) In the SOS study, much of the improvement in blood pressure observed in the surgical group at 2 years was lost at 10-year follow up.(37)

Following bariatric surgery there are reductions in several components of serum lipids. In particular, triglycerides markedly decrease (−50 to −100 mg/dl). LDL cholesterol levels also decline (−5 to −40 mg/dl) while HDL cholesterol leves generally increase (+5 – + 15 mg/dl). The magnitude of favorable changes in triglycerides and HDL are typically as large or larger than what can be achieved with currently available pharmacological treatment of dyslipidemia.

Diabetes and insulin resistance are dramatically improved after bariatric surgery. Such improvements begin to occur within days after surgery – a time when the amount of weight loss seems to be insufficient to explain the improved glycemic regulation.(52) These findings have been interpreted as evidence that bariatric surgery, particularly GBS or the gastric sleeve procedure, produce changes in gut-secreted hormones that acutely regulate metabolism.(52) At 1–2 years after bariatric surgery, up to 80% of diabetic patients have completely resolution of that condition (normal fasting glucose without glucose lowering medication; Table 2).(37, 38) By 10 years, 36% of patients suffering from diabetes prior surgery, continue to be free of diabetes.(37)

Obesity, particularly visceral obesity, is believed to be an inflammatory state. Systemic inflammation is proposed to contribute to several aspects of cardiovascular risk including acceleration of atherosclerosis progression or development of unstable atherosclerotic plaques. Obese subjects have elevated levels of high sensitivity C-reactive protein (CRP) and various cytokines or adipocytokines. Following gastric banding or gastric bypass surgery, essentially all inflammatory markers show significant decreases.(50, 53–56)

Changes in cardiac geometry and function after weight loss

There are also favorable changes in cardiac geometry following bariatric surgery in both adults and adolescents (Table 3). (19, 30, 31, 57, 58) The magnitude of these changes appears to be proportional to the amount of weight loss. Left ventricular mass decreases by ~ 25–30 g and left atrial volume is relatively reduced (6–15 ml) compared to control subjects with continued obesity.(35, 57) Although LV ejection fraction does not change(19, 30, 57), the more favorable LV geometry leads to improvements in LV midwall fractional shortening and tissue Doppler or strain measurements of LV systolic and diastolic function. (17, 25, 59)

Cardiovascular outcomes following bariatric surgery

Predicted cardiovascular event rates following bariatric surgery

Several groups have calculated the changes in medium and long-term risk of events based on the favorable effects of bariatric surgery on cardiac risk factors. Based on Framingham and Prospective Cardiovascular Munster Heart Study (PROCAM) risk scores, Batsis et al calculated that 10-year event rates would decline from 7.0 to 3.5% and 4.1 to 2.0 %, respectively.(60) Using Framingham risk scores, Vogel and colleagues estimated that 10 year cardiovascular risk would decline from 6% to 4% after gastric bypass surgery. Similarly, using data from 500 patients undergoing gastric bypass surgery, Torquati et al calculated an absolute decline in 10-year risk of cardiovascular events from 5.4% to 2.7%. (48)

Evidence that bariatric surgery may influence the progression of atherosclerosis

Based on the highly favorable changes in risk profiles, weight loss surgery could have demonstrable effects on the progression of atherosclerosis. However, there are currently only limited data regarding this key question The Program on the Surgical Control of Hyperlipidemias (POSCH) study used partial ileal diversion in 421 patients with known coronary artery disease as a means of lowering serum lipids rather than as a means of weight loss.(61) Although not used for weight loss in this study, this procedure is analogous to the most aggressive bariatric surgeries. Patients in the surgical arm had less progression of coronary atherosclerosis over 10 years compared to a control group (n=417) as assessed by serial invasive coronary angiography.(61) Preliminary data from the Utah Obesity Study showed lower coronary calcium scores and a significantly higher probability of having a zero calcium score in patients undergoing gastric bypass surgery 5 years previously (n=61) compared to a subjects in a nonsurgical control group (n=72).(62) Lastly, measurements of carotid intima-medial thickness done at baseline and 3–4 years later showed that obese subjects undergoing bariatric surgery (n=20) had a rate of IMT progression similar to a lean control group (n=35), whereas patients with continued obesity (n=19) had a rate of progression ~ 3 times higher.(15) Taken together, these data show consistent evidence that weight loss surgery may, over a period of years, fundamentally slow the process of atherosclerosis.

Cardiovascular events and mortality after bariatric surgery

Given the aforementioned observations regarding atherosclerosis, it follows that bariatric surgery could lead to reductions in cardiovascular events, such as myocardial infarction, heart failure or atrial fibrillation. Because bariatric surgery is most often performed in females in their mid 40’s, a group with low short-term cardiovascular risk, it is likely it will take 10–20 years to demonstrate conclusively whether bariatric surgery reduces cardiac events. At the present time, the longest duration of prospective observation after bariatric surgery comes from the SOS study, which has reported outcome data with a mean of 10.9 years of follow up.(41) In that study, Sjostrom and colleagues reported that the adjusted hazard ratio for mortality was 0.71 in the surgery group compared to the control group. The risk of myocardial infarction, the 2nd leading cause of death after cancer, was reduced by almost 50% in the surgery group (13 vs. 25 subjects). Adams et al reported total and cause-

specific 10-year mortality in 7,925 subjects undergoing gastric bypass surgery compared to that in an age and BMI matched control group obtained from drivers license records.(63) They reached similar conclusions to those from the SOS study. Namely, bariatric surgery was associated with a 40% reduction in all cause mortality and a 56% reduction in mortality due to coronary artery disease. Figure 1 shows a proposed scheme by which obesity related comorbidities lead to cardiovascular complications and increased mortality. The cascade of deleterious events can be largely interrupted by bariatric surgery.

Conclusions

In conclusion, abundant evidence shows that the adverse cardiovascular risk profile seen in obese subjects is profoundly improved 2–10 years after weight loss surgery. Similarly, there are improvements in cardiac geometry and structure. The significant improvements in cardiac risk factors appear to translate into slowed progression of atherosclerosis and significant reduction in total and cardiovascular mortality over 10 years. Although head to head comparisons of different weight loss procedures are still lacking, most data support a direct relationship between the magnitude of improvement in risk factors or cardiac geometry and the amount of weight that is lost. On the basis of these findings, procedures with a malabsorptive component such as gastric bypass surgery, would be predicted to be more beneficial than purely restrictive procedures such as gastric banding.(64) Given the unequivocal sustained benefits of surgically induced weight loss, it is likely that bariatric surgery will continue to evolve and to have an expanding role in the prevention of cardiovascular disease.

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Key Points

1. Obesity is associated with increased total and cardiovascular mortality.
2. The frequent association of cardiovascular risk factors with obesity likely contributes to premature and accelerated atherosclerosis, which then leads to the high risk of cardiovascular events.
3. Structural changes in the heart including concentric LV hypertrophy and left atrial enlargement may predispose to the development of heart failure and atrial fibrillation.
4. Bariatric surgery produces marked weight loss, impressive reversal or even resolution of coronary risk factors including, hypertension, diabetes, dyslipidemia and inflammation. There is also significant reduction in LV mass and prevention of LA enlargement.
5. Surgically-induced weight loss is associated with ~ 50% reduction in the risk of cardiovascular events and a similar reduction in 10-year cardiovascular mortality.

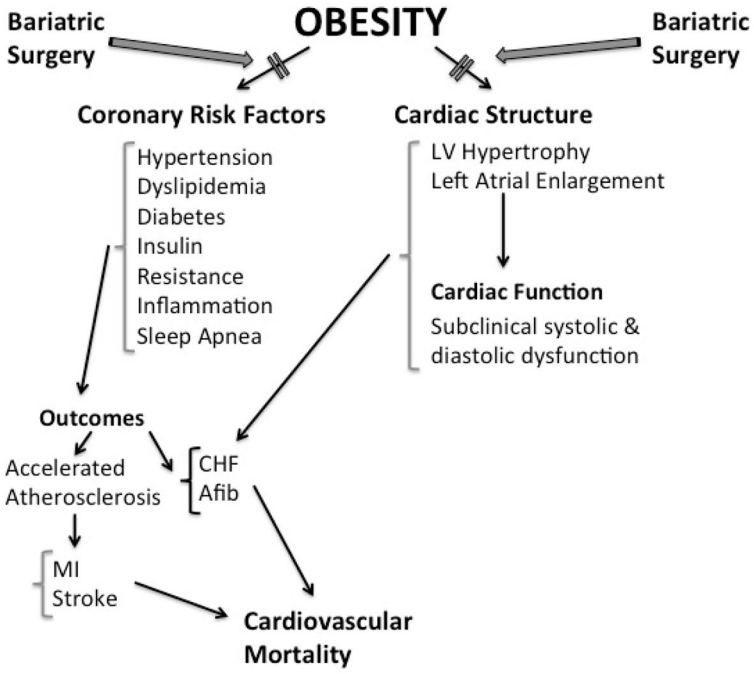

Figure 1.

Proposed model by which obesity contributes both directly and indirectly to adverse cardiovascular outcomes. Bariatric surgery can effectively reduce or eliminate most of the comorbid conditions that are responsible for the elevated morbidity and mortality associated with obesity.

Table 1

Changes in cardiovascular risk factors after bariatric surgery

	Pontiroli (46)		Stoopen-Margain (47)		Busetto (44)		He & Stubbs (45)		Batsis (43)		Sjostrom (37)			Adams (38)		Torquati (48)		Williams (50)		Vogel (49)	
	Base	F/U	Base	F/U	Base	F/U	Base	F/U	Base	F/U	Base	F/U	F/U	Base	F/U	Base	F/U	Base	F/U	Base	F/U
Study duration		1		1.7		1.3		1		3.3		2	10		2		1		1		2
N	143		100		650		310		197		1845			420		500		356		109	
Age (years)	43		31		38		42		43		42			43		45		43		46	
BMI	45	37	50	36	47	38	46	NA	50	34	47	32	35	48	32	53	33	47	36	49	36
Women (%)	81		63		76		77		80		71			84		81		84		75	
Systolic BP	133	128	155	123	146	131	144	125	134	121	144	137	145	129	113	130	121	155	123	133	116
Diastolic BP	83	81	97	79	94	87	85	82	80	72	89	84	87	73	69	83	80	97	79	80	71
Total cholesterol	201	205	204	179	209	203	244	208	199	154	226	220	220	186	161	NA	NA	192	166	201	176
HDL	48	53	NA	NA	46	46	36	52	45	55	46	51	57	44	53	51	61	46	54	50	54
LDL	NA	NA	123	95	132	134	145	131	117	77	140	134	129	107	90	112	83	125	88	113	100
TG	151	106	246	153	151	115	315	129	188	111	204	144	171	195	121	NA	NA	133	92	205	132
DM (%)	46	21	24	14	11	4	17	1	32	11	74	21	18	19	4	28	6	33	14	36	11
Glucose	112	97	134	95	104	94	NA	NA	117	95	97	84	95	97	82					108	94

Study duration, years); N, number of patients in each study; BMI, kg/m^2; BP, blood pressure in mmHg; cholesterol, HDL, LDL, TG and glucose (mg/dl); DM, diabetes mellitus

Table 2

Baseline prevalence, incidence and resolution of comorbidities at 2-year follow up after bariatric surgery.

Variables	Bariatric surgery			Control groups			Study
	Prevalence	Incidence	Resolved	Prevalence	Incidence	Resolved	
Hypertension	31.9	1.4	37.6	34–37	7.3–9.8	1.3–3.7	Adams (38)
Dyslipidemia	57.8	5.1	54.2	51–64	35.3–39.6	7.2–13.9	
Diabetes	19.5	0	78.7	22–23.4	5.6–9.5	0–4.7	
Hypertension	NA	41	19	NA	11	1.3–3.7	Sjostrom (37)
Dyslipidemia	NA	27	62	NA	17	22	
Diabetes	NA	7	36	NA	24	13	

Numbers are shown as percent of subjects. The majority of subjects in the study of Sjostrom et al had gastric banding while all of those in the study by Adams et al had gastric bypass.

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Table 3

Changes from baseline to 2-year follow up in cardiac geometry in control (nonsurgical) and gastric bypass surgery patients.

Variable	Baseline		2 Years	
	Control	GBS	Control	GBS
LA volume (ml)	53.9±19.1	55.3±17	56.7±14.5	54.4±15.2*
IVSd (cm)	1.14±0.24	1.08±0.24	1.13±0.24	1.03±0.21*
PWd (cm)	1.13±0.22	1.08±0.23	1.11±0.23	0.99±0.18*
LVIDd (cm)	4.43±0.65	4.58±0.64	4.43±0.59	4.44±0.58*
RWT	0.53±0.16	0.49±0.15	0.52±0.16	0.46±0.12*
LVMI ($g/m^{2.7}$)	44.0±13.0	44.0±12.0	44.0±12.0	38.0±10.0*
% with LV hypertrophy	67%	64%	64%	56%*
LV EF (%)	64±9.0	63±11	65%	65±8

LA, left atrial; IVSd, interventricular septal thickness; PWd, posterior wall thickness; LVIDd, left ventricular internal diastolic dimension; RWT, relative wall thickness; LVMI, left ventricular mass index; EF, ejection fraction.

* $p < 0.05$ vs. GBS vs. control. Data are from Owan, et al.(57)