Bariatric Surgery for Severe Obesity

HARVEY J. SUGERMAN
Emeritus Professor of Surgery
Virginia Commonwealth University
Richmond, Virginia

SEVERE OBESITY is associated with a large number of comorbid diseases. These start at the head (stroke, diabetic retinopathy, pseudotumor cerebri, tinnitus) and go to the toes (diabetic neuropathy, foot ulcers, venous stasis disease) and affect almost every organ in between: lungs, heart, liver, spleen, gall bladder, esophagus, intestines, colon, kidneys, bladder, ovaries, prostate, breast, kidneys, bladder, legs, etc. Many of these comorbidities can be divided into two major groups: the metabolic syndrome (also known as “Syndrome X”) and comorbidities secondary to an increased intra-abdominal pressure (1). Metabolic syndrome comorbidities include type 2 diabetes mellitus (T2DM), hypercholesterolemia, hypertriglyceridemia, non-alcoholic liver disease (NALD) or steatohepatitis (NASH), polycystic ovary syndrome, hypertension, and gallstones. The comorbidities associated with an increased intra-abdominal pressure include pseudotumor cerebri, obesity hypoventilation, venous stasis disease (venous thrombosis, venous stasis ulcers, pulmonary embolism), gastroesophageal reflux disease (GERD), urinary stress incontinence, abdominal hernias (inguinal, umbilical, incisional), hypertension, and the nephrotic syndrome. Other problems such as sleep apnea, diverticulitis, necrotizing pancreatitis, and musculoskeletal disorders (low back, hip, and knee pain), as well as depression and quality of life (QoL) issues are unrelated to either the metabolic syndrome or increased intra-abdominal pressure. Severe obesity also increases the risk of developing cancer (esophagus, liver, pancreas, kidney, colon, breast, uterus, ovary, prostate, leukemias, lymphomas), some of which are associated with the metabolic syndrome (e.g., breast, uterus, prostate) and some secondary to an increased intra-abdominal

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1 Read 24 April 2009. Correspondence: 290 Southwinds Dr., Sanibel, Florida 33957; hsugerman@comcast.net.
pressure (e.g., esophagus). Severe obesity is also associated with severe discrimination, problems with employability, and absenteeism, as well as presenteeism (limited quality and quantity of work when employed).

Unfortunately, dietary management with or without pharmaceutical therapy has not been effective over the long term (three to five years) for severely obese individuals. Weight loss peaks at approximately 10% of weight, but recidivism is almost invariable. Bariatric surgery is associated with the loss of approximately one-third of pre-operative weight or two-thirds of excess weight, and this weight loss is reasonably stable over ten years, with a relatively small degree of recidivism (2–4). In 1991 the National Institutes of Health Consensus Conference supported bariatric surgery in patients with a body mass index (BMI) of 35 kg/m² and obesity-related comorbidities or a BMI of 40 kg/m² regardless of comorbidity status (5).

The current operations for severe obesity include Roux-en-Y gastric bypass (RYGB), laparoscopic adjustable gastric band (LAGB), biliopancreatic diversion (BPD) with or without a sleeve gastrectomy (SG), and an SG by itself (figs. 1–4) (6–9).

Surgically induced weight loss is associated with a significant improvement in or remission of all obesity-related comorbidities. In a study by Christou et al. there were significant improvements after RYGB in patients from Quebec, Canada, in musculoskeletal disorders, infectious complications, endocrinological problems, cardiovascular complications (myocardial infarction, peripheral vascular disease), respiratory problems (sleep apnea, obesity hypoventilation), mental problems, genitourinary problems, and, for the first time, a significant decrease in cancer care (10). In a meta-analysis with 22,094 patients, Buchwald et al. found a significant improvement in T2DM (86%), hyperlipidemia (70%), hypertension (62%), and sleep apnea (86%) following bariatric surgery (11). In that study, there was significantly more weight loss with the RYGB and BPD than with the LAGB, and these were associated with a better improvement in T2DM than the LAGB. The LAGB had the lowest mortality risk (0.1%), RYGB intermediate (0.5%), and BPD highest (1.1%). In another, more recent meta-analysis with more than 135,000 patients, Buchwald et al. noted that T2DM improved or went into remission in 87% of the patients who underwent bariatric surgery (12). In a cohort study from Sweden known as the Swedish Obese Subjects (SOS) study, in which 10,000 severely obese patients were enrolled, of whom 1,000 were to undergo bariatric surgery, 851 patients who were ten years or more past their operation were matched to a similar number of control patients (13). Significant improvements were noted in hypertriglyceridemia and T2DM at ten years, but not in hypertension. This study found significantly greater weight loss in the patients who had undergone
RYGB than in those who had a gastroplasty or banding procedure; however, there were very few patients (only 34) who had undergone the RYGB. In another publication, this group found a significant decrease in both systolic and diastolic blood pressure at eight years in the patients who had RYGB, but not in those who had gastroplasty or banding (14).

There appears to be a much more rapid improvement in T2DM after RYGB than after LAGB; RYGB is also associated with a greater number of patients who achieve this improvement. Rarely, patients may develop beta cell hyperplasia, or nesidioblastosis, after RYGB, necessitating a partial pancreatectomy (15). This RYGB effect is thought to be secondary to alterations in gut peptide release. There are two main theories as to the special effects of RYGB: the proximal gut theory: the “distal” and “proximal” gut hypotheses (16). The “distal” hypothesis postulates that nutrients reach the distal ileum within five minutes of ingestion, and that that stimulates the secretion of GLP-1 by ileal L-cells, which then stimulates beta cell production of insulin. The “proximal” theory postulates that the exclusion of the duodenal passage may offset an abnormality of gastrointestinal physiology responsible for insulin resistance and T2DM secondary to the release or inhibition of a peptide from the duodenum. In a study of the Goto-Kakizaki (GK) model of T2DM, a gastrojejunostomy was constructed for one group of rats, and a gastrojejunostomy with transaction of the first portion of the duodenum was constructed for a duodenal exclusion, and the two groups of experimental rats and a sham group underwent an oral glucose tolerance test (OGTT) (17). The duodenal exclusion rats had a significantly lower area under the curve (AUC) than either the sham or gastrojejunostomy rats for glucose. In another study, a plastic sleeve was placed at the gastroduodenal junction, which prevented nutrients from contacting the duodenal mucosa in one group of GK rats, and a fenestrated sleeve placed in a second group (18). Again there was a significantly lower AUC for glucose in the duodenal exclusion group as compared with either the sham or fenestrated sleeve group, and, as in the previous study, there was no difference in the AUC for the sham or fenestrated sleeve groups (fig. 6). There was also a significant suppression of gastric inhibitory polypeptide (GIP) at thirty minutes in the animals with the duodenal exclusion group as compared with either the sham or fenestrated tube groups. There have recently been reports from Brazil that non-morbidly obese T2DM patients who have undergone a duodenal exclusion procedure have had a significant improvement in their T2DM, including improvements in their glycated hemoglobin (HbA1c) levels (19). These data support the “proximal” gut hypothesis, but they do not exclude the “distal” hypothesis, as glucagon-like peptide 1 (GLP-1) and pancreatic polypeptide-Y (PPY) levels do increase after an RYGB (17).
Figure 1. Laparoscopic Roux-en-Y gastric bypass (RYGB), showing transected stomach, gastrojejunostomy and jejunojunostomy. With permission from the Cleveland Clinic Foundation.

Figure 2. Laparoscopic adjustable gastric band (LAGB). With permission from the Cleveland Clinic Foundation.
Figure 3. Biliopancreatic Diversion with Duodenal Switch (BPD/DS). With permission from the Cleveland Clinic Foundation.

Figure 4. Laparoscopic sleeve gastrectomy. With permission from the Cleveland Clinic Foundation.
Reactive hypoglycemia is a not unusual complication of an RYGB that is due to the Somogyi effect, or an excess of insulin after a meal. It can usually be prevented by having the patient drink half a glass of orange juice about an hour after a meal. Severe and persistent hypoglycemia is cause for evaluation for beta cell hyperplasia, for which a partial pancreatectomy may, rarely, become needed.

Surgically induced weight loss has been shown to significantly improve obesity venous stasis ulcers, polycystic ovary syndrome, pso-tumor cerebri, gastroesophageal reflux disease (GERD), and asthma (1). In the early 1900s Sir William Osler coined the term “Pickwickian Syndrome” for individuals who had a combination of obesity hypoventilation (OHS) and obstructive sleep apnea (SAS) syndromes. This was named after Joe, the servant in The Pickwick Papers, who was continually falling asleep—while driving his master in his horse and buggy and even while knocking on a door. The syndrome was forgotten until resurrected in a case report in the Annals of Internal Medicine describing a fifty-two-year-old gentleman who had fallen asleep in a poker game with three aces and two kings, known as a “full house”(20). Charles Dickens had a close friend, a physician, whom Dickens accompanied when he visited patients in the hospital; there must have been a patient there like Joe in The Pickwick Papers. The obesity hypoventilation syndrome describes severely obese individuals who have hypoxemia and hypercarbia while awake plus sleep apnea syndrome, causing them to fall asleep when they should be awake, driving a car—or truck on interstate highways—or listening to a lecture or watching a movie. Should you be in line for a taxi and the next driver is severely obese, I suggest you let the next person in line take that cab. One of my first patients was such a taxi driver. He weighed 580 lbs. and had fallen asleep multiple times while driving his taxi. He broke the toilet off the wall after his surgery, endearing me to the nursing staff. (Centers of Excellence certification requires hospitals in which bariatric surgery is performed to have specially designed toilets to prevent this problem.) Three years after his surgery he weighed 245 lbs. and his sleep apnea and obesity hypoventilation had resolved; he no longer had sleep apnea, hypercarbia, or hypoxemia (21). Patients with OHS have pulmonary hypertension; this resolves following correction of hypoxemia and hypercarbia after surgically induced weight loss (figs. 5,6). Bariatric surgery in patients with OHS has a tenfold higher risk than surgery in those without OHS, 2.2% vs. 0.2% mortality in our experience, but that means that 98% of these patients survive the surgery and their respiratory insufficiency goes into remission (22).

Bariatric surgery also improves heart conditions. It decreases the risk for atherogenesis and coronary artery disease with a decrease in low LDL, increasing HDL with an overall decrease in cholesterol and

Triglyceride levels (13). This is associated with a significant decrease in deaths due to myocardial infarction (23,24). There is also a decrease in arterial systolic and diastolic blood pressure, which is associated with a decrease in left ventricular wall thickness and cardiac hypertrophy (25). Improving OHS and SAS, as previously stated, decreases pulmonary artery pressure and right ventricular hypertrophy. These changes are associated with an increased ejection fraction and may be beneficial to some patients with obesity-associated congestive heart failure (26,27). These are also high-risk patients, but successful surgery is very rewarding to both the patient and the surgeon.

Surgically induced weight loss is also beneficial to obesity-related complications of pregnancy, including a decreased risk of gestational diabetes, preeclampsia, cephalo-pelvic disproportion, and Caesarian section, as well as obesity in the offspring of mothers who have had bariatric surgery (28–32).

Severe obesity is associated with fat infiltration of the liver, known as non-alcoholic fatty liver disease (NAFL). This may produce hepatic inflammation with non-alcoholic steatohepatitis (NASH). NASH will probably be the greatest cause of cirrhosis and liver failure in the future.
because of the epidemic of severe obesity, a frequency greater than Hepatitis A, B, and C combined. Surgically induced weight loss is associated with resolution of fatty infiltration, a decrease in hepatic inflammation, and even some resolution of fibrosis. Major weight loss may permit an obese cirrhotic patient to become eligible for a liver transplant (32–35).

Bariatric surgery improves patient mobility and decreases hip, knee, and lower back pain and may make hip or knee joint replacements unnecessary or improve their long-term outcomes (36–38). There is also a significant improvement in patient quality of life, formerly often miserable, as well as employment status (39–41). There is an initial improvement in depression; however, over time this may dissipation, and there has been an increased risk of suicide several years after bariatric surgery (24,42).

Obesity has been shown to cause a marked increase in the risk of death, which becomes logarithmic after a BMI of 30 kg/m². Patients who have undergone bariatric surgery have been found in five studies to have a significant decrease in mortality compared with matched cohorts of those who have not had bariatric surgery (figs. 7,8) (10,23,24,43,44).
Three other studies have also shown a decreased mortality in bariatric surgical patients, compared with those who were unable to undergo surgery for personal or insurance reasons (45–47). This decreased mortality is a result of a decrease in rates of myocardial infarction, diabetes, and cancer deaths. There is a decreased risk of breast cancer, as well as other endocrine-related cancer deaths (10,23,24,48,49).

Because of the improvements in all of these obesity comorbidities, bariatric surgery has been shown to pay for itself in two years (after laparoscopic surgery) to four years (after open surgery) (50,51). There is a decrease in physician visits, medications, and hospitalizations. There is an increase in plastic surgical procedures after bariatric surgery (abdominoplasty, arm tucks, thigh and breast procedures), but almost all of these are self-paid and not a burden to health insurance.

As with anything in life, there are risks to bariatric surgery. Some of these are nutritional, some procedural. The greatest nutritional risk is beriberi or thiamine deficiency, which can occur after any bariatric procedure and is usually secondary to persistent nausea and vomiting, which is usually a result of a stricture at the gastrojejunostomy after RYGB or too tight a band after LAGB (52). This complication is totally preventable with endoscopic dilatation or loosening of the band, but if it occurs, it can be devastating and irreversible, with an encephalopathy or peripheral neuropathy (53).

Other nutritional complications include calcium and vitamin B₁₂ deficiencies and, especially in menstruating women, iron deficiency after either RYGB or BPD/DS. The BPD/DS may be associated with fat-soluble vitamin (A, D, E, K) deficiencies, as this operation leads to fat malabsorption and steatorrhea. These deficiencies are usually preventable. If they do occur, they respond to vitamin and mineral supplementation (53).

Conclusion

Bariatric surgery saves lives with a decreased death rate from myocardial infarction, diabetes, and cancer. Surgically induced weight loss leads to improvement or remission of numerous comorbidities, including obesity hypoventilation, sleep apnea, type 2 diabetes, dyslipidemia, venous stasis, pseudotumor cerebri, non-alcoholic liver disease and steatohepatitis, hypertension, cardiac dysfunction, urinary incontinence, polycystic ovary syndrome, and pregnancy-related complications, and is associated with a marked improvement in quality of life. An epigenetic effect has been found, with a decreased rate of obesity in the offspring of women who have had surgically induced weight loss. Furthermore, bariatric surgery has been shown to pay for itself in two to
four years. It is difficult to understand why health insurance companies, supposedly designed to help their beneficiaries, work hard to impede access to this surgery. Clearly, bariatric surgery is not the answer to the huge problem of obesity throughout the world, but for those whose lives are broken from severe obesity, it is currently the only effective solution.

References


