The Effect of Obesity Surgery on Obesity Comorbidity

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ABSTRACT: Obesity is epidemic in the modern world. It is becoming increasingly clear that obesity is a major cause of cardiovascular disease, diabetes, and renal disease, as well as a host of other comorbidities. There are at present no generally effective long-term medical therapies for obesity. Surgical therapy for morbid obesity is not only effective in producing long-term weight loss but is also effective in ameliorating or resolving several of the most significant complications of obesity, including diabetes, hypertension, dyslipidemia, sleep apnea, gastroesophageal reflux disease, degenerative joint disease, venous stasis, pseudotumor cerebri, nonalcoholic steatohepatitis, urinary incontinence, fertility problems, and others. The degree of benefit and the rates of morbidity and mortality of the various surgical procedures vary according to the procedure. **KEY INDEXING TERMS:** Obesity; Obesity surgery; Comorbidity. **[Am J Med Sci 2006;331(4):183–193.]**

Background

Obesity is a stronger predictor of mortality and morbidity than either poverty or smoking.[1] In the Framingham cohort, obesity has been found to be equivalent to smoking in terms of years of life lost. Obesity is now more prevalent in the world than malnutrition, and that prevalence continues to increase.[2] The fastest rate of increase has been among the severely obese, so that recent estimates suggest that 8% to 10% of American women and 5% of American men are now morbidly obese (body mass index [BMI] $\geq$40).[3] Morbid obesity is estimated to account for about 5% of total U.S. health costs (approximately $60 billion/year).[4] Most of this cost is associated with the morbidity and mortality arising from obesity associated comorbidities. These have been defined by the National Institutes of Health to include hypertension, diabetes mellitus type 2, dyslipidemia, obstructive sleep apnea, hypoventilation, hypertrophic cardiomyopathy, gallbladder disease, degenerative arthritis, psychosocial impairments, and certain forms of cancer.[5] Unfortunately, dietary and pharmacological therapies are relatively ineffective in treating obesity in the long term, especially for morbidly obese patients.[6,7] The only effective weight loss therapy for severely obese patients with BMI of 35 or greater has proven to be surgical (Table 1).[3]

The most commonly performed surgical procedure for severe obesity in the United States is gastric bypass, whereas restrictive procedures (vertical banded gastroplasty, gastric banding, gastroplasty) are more common internationally. Gastric bypass via a laparoscopic approach and adjustable gastric banding are becoming increasingly utilized. The number of obesity surgery procedures has skyrocketed in the last few years, by more than 40% in 2002 to 80,000, and by 2003 was expected to climb to 120,000.[3,8] U.S. spending on obesity surgery is now approximately $3 billion per year, with an average cost of $25,000 per procedure.[3] This paper reviews the effects of obesity surgery on the comorbidities of severe obesity (Figures 1 and 2).

The Effect of Bariatric Surgery on Obesity Comorbidity

Diabetes

More than 95% of prevalent type 2 diabetes is attributable to overweight/obesity. The risk of type 2 diabetes increases as weight increases.[9] Multiple cross-sectional studies have demonstrated the strong association of weight with type 2 diabetes in men and women.[10] Compelling evidence exists to show that type 2 diabetes and insulin resistance improve across all types of bariatric surgery. Buchwald et al., in a recent meta-analysis of 136 controlled trials and case series, found that type 2 diabetes was resolved (as defined by the ability to discontinue all diabetes medications and maintain

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Table 1. National Institute of Health Consensus Criteria: Surgical Eligibility

BMI	Comorbidity
35–39	Type 2 diabetes, hypertension, hypoventilation, sleep apnea, venous stasis, pseudotumor cerebri, polycystic ovary, degenerative joint disease, NASH, and others
>40	Any or none

BMI, body mass index; NASH, nonalcoholic steatohepatitis. From Lara MD, Kothari SN, Sugerman HJ. Surgical management of obesity: a review of the evidence relating health benefits to risks. Treat Endocrinol 2005;4:55-64.

normal blood glucose levels) in 76.8% of patients.[11] When resolution was analyzed by procedure, there was a clear gradation according to procedure, with a 98.9% rate for biliopancreatic diversion, 83.7% rate for gastric bypass, 71.6% rate for gastroplasty, and 47.9% rate for gastric banding. Of those patients not experiencing complete resolution, more than half showed significant improvement. Interestingly, two recent gastric bypass case series have supported the findings of this meta-analysis, showing almost identical resolution rates of 83%[12] and 86%[13].

Pories et al. showed, in a case series including 146 diabetic gastric bypass patients, that type 2 diabetes remained resolved in 83% of patients at 14 years, with normal fasting glucose and plasma insulin levels and glycosylated hemoglobin (A1c).[14] Moreover, of the 152 patients in that series with impaired glucose tolerance (IGT) who were at risk for type 2 diabetes, 99% had complete resolution of their IGT and none developed diabetes after 14 years. There was no significant weight regain in these cohorts. By contrast, in the Swedish Obesity Study (SOS), a 10-year case-controlled study of primarily vertical banded gastroplasty or gastric banding patients (94%), the recovery rate from type 2 diabetes was 72% at 2 years, but at 10 years had fallen to only 36%.[15] Recurrence of type 2 diabetes was accompanied by a significant increase in weight after the 2-year mark. Also, the initial amount of weight lost was less than that reported in Pories' bypass study. Weight regain is a common problem associated with restrictive procedures but is much less frequent with diverting procedures. Interestingly, improvement in diabetic neuropathy after obesity surgery has been noted.[16]

The time-course of diabetes resolution is often abrupt after obesity surgery, with improvement in insulin resistance occurring before significant weight loss. This observation and the more pronounced changes observed with combined restrictive and diverting procedures (gastric bypass, biliopancreatic diversion) have sparked a great deal of speculation about the role of gut hormones in insulin resistance and the possibility of novel regulatory mechanisms. Accordingly, the impact of various obesity surgery procedures on leptin, ghrelin, resistin, adiponectin, glucagon-like peptide 1, and other satiety mechanisms is receiving increasing attention.[17–21] However, all forms of weight loss surgery lead to weight loss, caloric restriction, decrease in fat mass, and improvement in type 2 diabetes, and the degree of these improvements in the long term do seem to be stratified by weight. These observations alternatively suggest that the improvements in glucose metabolism and insulin resistance after obesity surgery may primarily arise from known mechanisms: in the short term due to decreased stimulation of the enteroinsular axis by decreased calorie intake, and in the long term from decreased fat mass and resultant changes in the release of adipocytokines.[22]

Hypertension

Epidemiologic studies indicate that in the United States excess weight gain accounts for as much as 75% of prevalent hypertension.[23] The continuous linear relationship between BMI and blood pressure is well known.[24] Indeed, almost all individuals show

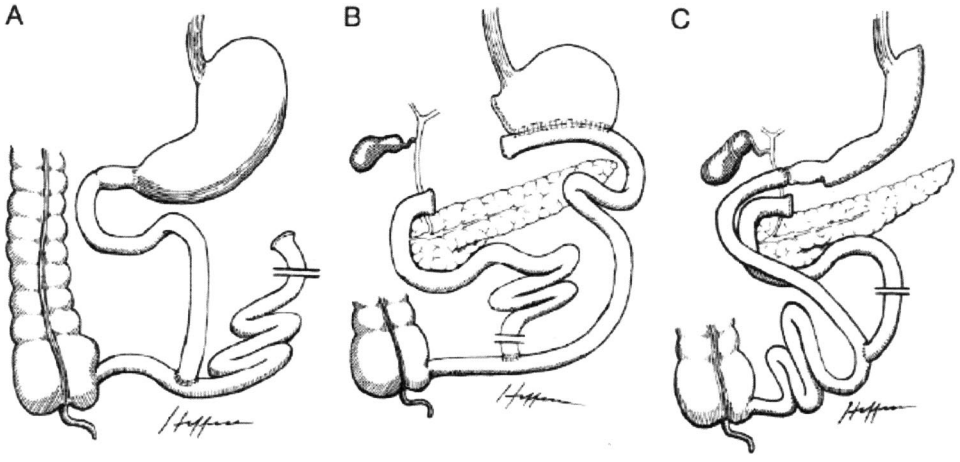

Figure 1. Malabsorptive bariatric operations: jejunoileal bypass (A); biliopancreatic diversion (B); duodenal switch (C). (From Cummings CE, Joost O, Foster-Schubert KE. Gastric bypass for obesity: mechanisms of weight loss and diabetes resolution. J Clin Endorin Metab 2004;89:2608-15.)

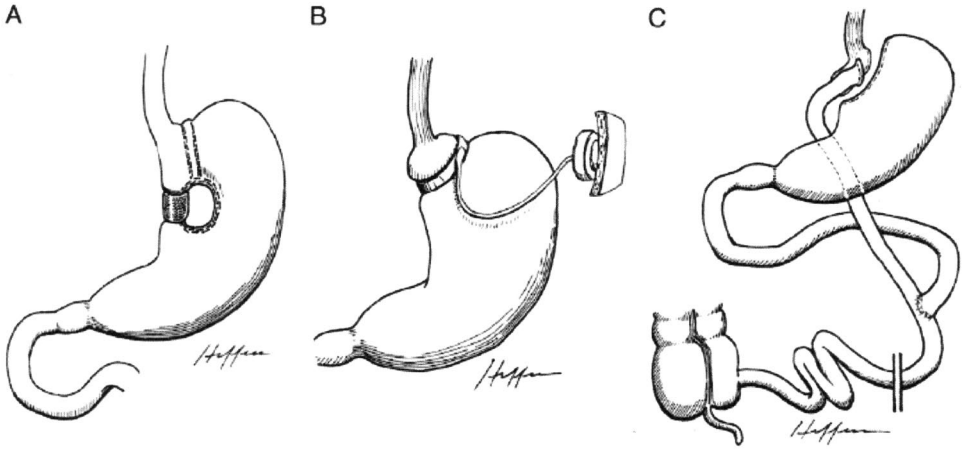

Figure 2. Restrictive bariatric procedures: vertical banded gastroplasty (A); adjustable gastric banding (B). Mixed restrictive and malabsorptive procedure: roux-en-Y gastric bypass (C). (From Cummings CE, Joost O, Foster-Schubert KE. Gastric bypass for obesity: mechanisms of weight loss and diabetes resolution. J Clin Endocrin Metab 2004; 89:2608-15.)

an increase in blood pressure with weight gain, and weight loss is almost always associated with a decreased blood pressure, even in normotensive individuals. Generally, a 1% decrease in body weight has been associated with about a 1 mm Hg decrease in systolic blood pressure and about a 2 mm Hg decrease in diastolic blood pressure.[25–27] In obese, hypertensive patients, a number of randomized controlled trials and prospective cohort studies have shown that intentional weight loss, whether dietary or surgical, leads to reductions in systolic blood pressure, diastolic blood pressure, and decreased use of antihypertensive medications.[28–36]

It is therefore not surprising to find that obesity surgery is commonly associated with marked improvements in or resolution of clinical hypertension.[3,11] Sugarman, in a large series of 1000 obese subjects undergoing gastric bypass, found that of the diabetic subset, 75% had a diagnosis of hypertension. Of these, at 1 year after bypass, 69% had resolution of hypertension, and at 7 years, 66% had resolution.[37] In this series, patients with resolution of diabetes or hypertension had a greater percent excess weight loss and a greater percentage weight loss than patients with nonresolved cases.

In SOS, a long-term prospective, case-controlled trial, 2-year and 10-year recovery rates from hypertension were greater for obesity surgery subjects than for traditional weight loss control subjects. However, the 2- and 10-year incidence rates for hypertension were not changed by obesity surgery.[15] In this study, net weight lost at 2 years was 23.4%, and at 10 years was only 16.1%, likely reflecting the fact that 94% of the procedures involved were restrictive (vertical banded gastroplasty or gastric banding). In the 6% of procedures involving gastric bypass, a greater percentage of weight loss was achieved initially and maintained (25% at 10 years), with a correspondingly greater decrease in systolic blood pressure and diastolic blood pressure.[15] Incidence rates of hypertension among the gastric bypass subgroup were not reported, apparently due to statistical limitations.

Buchwald et al., in a recent systematic review and meta-analysis of 136 controlled trials and case series relating to obesity surgery, found that hypertension was significantly improved in the total population analyzed across all surgical procedures. Specifically, their meta-analysis found the rate of reported hypertension resolution to be 62% and the rate of combined resolution or significant improvement to be 78.5%.[11] Although it is difficult to apply these specific numbers to individual cases due to the nature of meta-analysis, their relative magnitude as well as the diversity of studies from which they were derived emphasizes the profound effect that obesity surgery has on improving or resolving hypertension.

Indeed, successful resolution of hypertension is a frequent outcome of obesity surgery. Cure of hypertension seems to be proportional to the amount of weight lost, and not the final weight. Maintenance of resolution seems to depend on the amount of weight regained. It is important to note that hypertension is often resolved even though patients do not achieve ideal body weight.[38]

Dyslipidemia

Several series have reported significant improvement in lipid profiles after obesity surgery, including marked reductions in low-density lipoprotein, increased high-density-lipoprotein (HDL), and decreased triglycerides.[39–45] Average reductions of as much as 40% of total triglycerides and 25% of total cholesterol have been reported within 6 to 12 months following gastric bypass surgery.[40] The SOS found significant improvements in triglycerides and HDL in the surgical group at 2 and 10 years. Though total cholesterol was not significantly different at 2 and 10 years in the entire cohort, in the gastric bypass subgroup total cholesterol, triglycerides, and HDL were all significantly improved.[15]

In general, dyslipidemia, hypercholesterolemia, and hypertriglyceridemia all appear to significantly improve with all types of bariatric surgical procedures. The percentage of patients experiencing improvement is generally in excess of 70% in most studies. The greatest improvement in these variables seems to occur with the malabsorptive procedures (biliopancreatic diversion, duodenal switch, gastric bypass surgery). In contrast, low HDL improved significantly with restrictive procedures (gastric banding, vertical banded gastroplasty, GP), with mean changes of up to 5 mg/dL.[11]

Cardiovascular Disease

No clear outcome data exist from controlled trials on the effect of obesity surgery on rates of cardiovascular disease incidence or events. Nevertheless, from the data reported above it would seem that, for the morbidly obese, obesity surgery is perhaps the most effective therapy for resolution or improvement of hypertension, type 2 diabetes, high LDL, high triglycerides, and high total cholesterol, all major modifiable risk factors for cardiovascular disease.[3] Improvements in each of these risk factors have been associated with reductions in myocardial infarction, stroke, and cardiovascular death. Data do exist that relate long-term weight loss to direct cardiovascular event reduction. For example, in the Framingham cohort, a 10% reduction in body weight resulted in a 20% decrease in the risk of cardiovascular disease.[46] Several randomized controlled trials in obese and nonobese adults with cardiovascular disease have shown that multiple risk factor reduction programs coupled with weight reduction or weight maintenance results in slower progression or regression of anatomic coronary artery disease and decreased anginal symptoms.[47–49] It seems very likely, therefore, that obesity surgery will be found to have a marked effect on cardiovascular mortality and morbidity.

Congestive heart failure and cardiomyopathy have also been associated with obesity.[50] Significant improvements in left ventricular ejection fraction, chamber size, and ventricular wall thickness have been observed after weight loss in the morbidly obese.[51] Similar echographic improvements in cardiovascular dynamic function have been described following surgical weight loss.[52,53]

Metabolic Syndrome

Metabolic syndrome has several definitions and has been described under several names, including obesity syndrome, insulin-resistance syndrome, the deadly quartet, and syndrome X. Its common components include visceral obesity, elevated blood pressure, microalbuminuria, insulin resistance, characteristic lipid abnormalities, and signs of inflammatory and coagulation pathway activity.[54,55] It is a known risk factor for cardiovascular disease and death, as well as a risk factor for and precursor to type 2 diabetes and hypertension.[56]

Gastric bypass surgery has been shown to prevent type 2 diabetes and cure IGT in up to 99% of obese patients manifesting the insulin-resistance component of metabolic syndrome for intervals as long as 14 years.[14] It is worth contrasting these results with the effect of lifestyle modification in the recent Diabetes Prevention Program and the Finnish Diabetes Study, which both achieved a 58% reduction in diabetes risk, though it is important to stress that the latter studies were randomized controlled trials. Also, in a series of female obese patients, resolution of metabolic syndrome at 1 year after surgery was described in 89% of those undergoing laparoscopic gastric bypass.[57]

The improvements observed in type 2 diabetes, hypertension, and dyslipidemia with weight loss surgery have already been commented on. The roots of these disorders in metabolic syndrome are unclear but are thought to relate to visceral adiposity through a number of mechanisms including intraorgan and intramuscular adipose deposition, renin-angiotensin system activation, sympathetic activation, direct mechanical compressive effects, and inflammatory and coagulation pathway abnormalities.[58] Gastric bypass surgery has been shown to decrease intramyocellular lipid content, decrease insulin resistance, and increase plasma adiponectin.[59–61] Gastric bypass has also been shown to decrease plasma concentrations of inflammatory markers (including C-reactive protein, interleukin-6, and tumor necrosis factor-α), increase procoagulant activity (PAI-1), decrease angiotensinogen production, and decrease leptin levels.[62,63] Finally, caloric intake and physical activity are both increased after obesity surgery up to 10 years after the procedure compared with case-matched control subjects.[15]

Sleep Apnea and Obesity Hypoventilation Syndrome

Pulmonary manifestations of obesity include dyspnea on exertion, shortness of breath at rest, asthma, obstructive sleep apnea (OSA), obesity hypoventilation syndrome (OHS), and pulmonary hypertension. OSA consists of frequent nocturnal apneas accompanied by oxygen desaturations during sleep. The degree of OSA is measured by the apnea index, which is also proportional to the degree of obesity.[64] OHS includes both waking and sleeping disorders of ventilation with resultant hypoxemia and hypercapnia, which is thought to arise principally from pulmonary compression because of increased intra-abdominal pressure, diaphragmatic elevation, and chest wall restriction.[65,66] Pulmonary hypertension is very common in these patients.

Several prospective cohort studies in obese patients have shown that weight loss results in im-

provement in sleep apnea symptoms.[67–69] In one long-term series of GBS patients, 93% had improved respiratory indices post procedure, as well as marked improvement in sleep quality.[70] Sugarman found OSA and OHS improved or cured in nearly all patients post gastric bypass.[71,72] In OHS patients, significant improvement of resolution of symptoms, pulmonary hypertension, hypoxemia, and hypercapnia has been described.[73] Buchman et al. in their meta-analysis found that OSA (OHS was included in this group) was significantly improved in the total patient population analyzed and across all surgical procedures. Furthermore, resolution was reported in 85.7% of patients.[11] Finally, the atrial and ventricular arrythmias associated with OSA have been reported to significantly improve after gastric bypass surgery.[74]

Gastroesophageal Reflux Disease and Asthma

Obesity is a major risk factor for gastrointestinal reflux disease. Frezza et al. found that gastric bypass surgery significantly decreased gastroesophageal reflux disease symptoms for up to 3 years after surgery, with 97% of patients reporting satisfactory relief.[75] Others have described similar improvement.[76] Although fundoplication has been shown to be effective at least in the short term, it has no effect on weight loss or on comorbidities.[3] Obesity surgery has been shown to improve asthma, presumably through the mechanism of limiting reflux.[77,78]

Degenerative Joint Disease

Obesity is clearly a cause of large joint degeneration and arthritis. Gastric bypass surgery has been shown to improve symptoms of degenerative joint disease in the hips, knees, feet, and lower back.[79] Arthroplasty, however, may be difficult or contraindicated in morbidly obese patients.[80] Obesity surgery may be considered to bring the morbidly obese into a safe and effective weight range for joint replacement.[81]

Venous Stasis

Morbid obesity is thought to contribute to chronic venous stasis disease of the lower extremities through the mechanism of increased abdominal pressure leading to impaired venous and lymphatic return.[82] Morbidly obese patients are at increased risk for pulmonary embolism, deep venous thrombosis, thrombophlebitis, stasis ulcers, lower extremity edema, lower extremity lymphedema, and pretibial bronze discoloration. Gastric bypass surgery has been shown in at least one series to improve the symptoms of severe venous stasis disease.[83]

Urinary Incontinence

Obesity appears to be an important etiologic factor in stress urinary incontinence, probably through the mechanism of increased abdominal pressure.[84] Two prospective studies have demonstrated that the weight loss after gastric bypass surgery has been associated with decreased intra-abdominal pressure, decreased sagittal abdominal diameter, and decreased stress urinary incontinence.[66,85] Other series have described the frequent alleviation of stress urinary incontinence after gastric bypass surgery.[3,86]

Nonalcoholic Steatohepatitis

Nonalcoholic liver disease and nonalcoholic steatohepatitis are strongly associated with obesity and type 2 diabetes. Nonalcoholic steatohepatitis may result in some cases of cirrhosis and liver failure. The pathophysiology whereby fat deposition in the liver may lead eventually to cirrhosis is unknown.[87] Obesity surgery has been shown to significantly decrease the severity of hepatic steatosis in the morbidly obese.[88–91]

Pseudotumor Cerebri

The cause of pseudotumor cerebri is unknown but is thought to be related to increased intracranial pressure, producing symptoms including headache, nausea, vomiting, and pulsating noises.[92] It is usually associated with young, obese women between the third and sixth decades. Animal models have implicated increased intra-abdominal pressure as a cause via the mechanism of increased intrathoracic pressure leading to decreased intracerebral venous drainage.[93] Gastric bypass surgery has been very successful in alleviating the symptoms of pseudotumor cerebri in severely obese patients over long periods. Interestingly, the success rates reported for gastric bypass surgery have been better than those reported for cerebrospinal fluid-peritoneal shunting.[94,95]

Pregnancy

Gastric bypass surgery has been shown to improve menstrual irregularities, fertility, and sex hormone balance in the morbidly obese.[86] Pregnancy has been shown to be safe after gastric bypass surgery.[96,97] Obese women who have had gastric bypass surgery have been shown to have fewer pregnancy-related complications than women who remain obese.[96–100] However, women should not become pregnant during the rapid weight loss phase in the first post-operative year and should use contraception during this time.[3]

Psychiatric and Social Disorders

The morbidly obese frequently suffer from poor self-image and social stigmatization.[101] They are at special risk for affective disorders, anxiety, and substance abuse.[102,103] Dietary weight loss is also a risk factor for depression, anxiety, and food preoccupations. Even obesity specialists have been shown to demonstrate prejudices against the obese, and 80%

report being treated disrespectfully by medical professionals.[104,105]

Quality of life for the morbidly obese has been shown to significantly improve as a result of obesity surgery.[28,102,106–108] After gastric bypass, patients have been shown to have better mood, self-esteem, and interpersonal effectiveness.[102,103] They are more involved involved in social activities, have higher levels of exercise compared to controls, and increase their outdoor activities with their children.[15,102,103]

Self-image improves after obesity surgery.[102,103] New community and vocational challenges are often taken on by patients after the procedure. Marital satisfaction increases but only if a measure of satisfaction was present prior to surgery. If marital discord was present preoperatively, then the improved self-image postoperatively may lead to divorce.[102,103]

Risks of Obesity Surgery

Surgical Weight Loss

Several randomized prospective[109–112] and retrospective studies[113–117] have shown that greater weight loss is achieved with gastric bypass than vertical banded gastroplasty. Laparoscopic gastric bypass has been shown to produce equivalent weight loss to open gastric bypass.[3] Although adjustable gastric banding has been shown to be effective for weight loss, the magnitude of weight loss may be less than with gastric bypass.[118–121] The time-course of the weight loss phase of laparoscopic banding is considerably longer than gastric bypass. Biliopancreatic bypass with duodenal switch may be associated with greater weight loss in the super obese (BMI $>$ 50), and may have less chance for weight regain.[122] However, a higher risk of fat-soluble vitamin and calcium deficiency and protein/calorie malnutrition exists with this procedure.

Mortality

The mortality rate for gastric bypass is reported at 1% to 2%. The risk of mortality is higher among the superobese compared to those with a BMI less than 50. By comparison, the mortality rate with laparoscopic banding is less than 1.0%.[3] Later conversion from a laparoscopic band to a gastric bypass procedure has greater risk than the primary gastric bypass procedure.

Wound Complications

Open obesity surgery procedures have a high rate of wound infections and wound hernias.[123] By contrast, laparoscopic gastric bypass procedures have wound infection rates of about 1.5% and wound hernia rates of about 1.8%.[124] Compared to open gastric bypass, laparoscopic gastric bypass has decreased wound infections, incisional hernias, and mortality. However, there is an increased risk of early and late bowel obstruction, stomal stenosis, and gastrointestinal bleeding when compared to open gastric bypass.[125]

Anastomotic Leakage

Anastomotic leaks are perhaps the most feared complication of gastric bypass surgery. The leak rate at the gastrojejunostomy or jejunojejunostomy site has been reported to be about 1.8%.[124] Leaks may occur at the gastrojejunostomy site, the jejunojejunostomy site, the roux limb stump, or the gastric staple line. A delay in diagnosis may lead to peritonitis, overwhelming sepsis, and death, so the index of suspicion must remain high in spite of negative radiography. Subclinical gastrojejunal anastomotic leaks noted on routine postoperative studies may sometimes be managed by intravenous antibiotics, nothing by mouth, and parenteral nutrition if a drain has been placed at the time of surgery.[3]

Stomal Complications

Stomal complications including ulceration, pain, bleeding, and stenosis/obstruction sometimes occur at the gastrojejunostomy site, presumably as a result of exposure of the roux limb of jejunum to unbuffered gastric acid.[126] In one series of 191 patients, the frequency of stomal ulceration was 12.5% and the frequency of stomal stenosis was 12%.[127] Eighty percent of cases occurred in the first 3 months postoperatively and almost all were managed conservatively. Stomal stenosis is usually successfully alleviated by endoscopic balloon dilatation.[3]

Deep Venous Thrombosis and Pulmonary Thromboembolism

Morbidly obese patients undergoing gastric bypass surgery, especially those with severe venous stasis disease, are at high risk for perioperative venous thromboembolism.[128] Laparoscopic bypass surgery involves several additional factors, which may increase the risk, including pneumoperitoneum, reverse Trendelenberg position, and increased venous stasis.[129] Although prophylaxis for venous thromboembolism is believed to be very important for the gastric bypass surgery patient, there is no concensus as to method.[130] Podnos et al. reviewed 3464 cases and found no difference in postoperative pulmonary embolism or pneumonia between laparoscopic and open gastric bypass.[125] The death rates in this study were 0.23% and 0.87%, respectively ($P = 0.001$).

Nutritional Complications

Longer term complications of obesity surgery include various nutritional deficiencies. Patients undergoing gastric bypass are at risk for iron, calcium, thiamine, folate, and vitamin B12 deficiencies.[131] Patients after biliopancreatic diversion with duodenal switch are at risk for vitamin A, D, E deficiency

Table 2. Comparison of Common Bariatric Surgical Procedures

Procedure	Benefits	Complications
Roux-en-Y gastric bypass	Greater weight loss than gastroplasty or adjustable gastric banding	Iron, calcium, vitamin B12 deficiencies; risk of internal hernia, gastric perforation, anastomotic leak
Laparoscopic gastric banding	Lowest mortality risk; less weight loss than gastric bypass	Band erosion, band slippage, esophageal dilatation, dysphagia, band/port infections
Biliopancreatic diversion with duodenal switch	Possibly greater weight loss than gastic bypass; recidivism lower than gastric bypass or banding	Greatest risk of iron and calcium deficiencies, fat-soluble vitamin deficiencies, protein-calorie malnutrition; technically the most difficult operation

From Lara MD, Kothari SN, Sugerman HJ: Surgical management of obesity: a review of the evidence relating health benefits to risks. Treat Endocrinol 2005;4:55-64.

as well and are at a much greater risk of calcium deficiency, osteoporosis, and protein/calorie malnutrition. In contrast, patients undergoing banding procedures are not at risk for iron, calcium, or B12 deficiency.

The patient should be routinely observed for the development of these complications, with appropriate dietary and symptom review and screening laboratory studies. Most of these complications can be prevented with prophylactic multivitamins and aggressive treatment of postoperative stomal stenoses associated with severe nausea and vomiting. Menstruating women who undergo gastric bypass should take supplemental iron sulfate tablets. All gastric bypass patients should take calcium, vitamin B12, and a multivitamin daily. Calcium citrate does not require the presence of stomach acid to be absorbed. Laparoscopic banding patients usually only require a multivitamin. Biliopancreatic bypass patients need the fat-soluble vitamins (A, D, E) and additional calcium in addition to the supplements recommended for gastric bypass patients (Table 2).[3]

Conclusion

Obesity surgery provides the most effective means of weight loss and sustained weight control for the morbidly obese. The objective of obesity surgery is to decrease excess body weight and consequent co-morbidities. Obesity surgery should be considered for patients who meet the criteria defined by the 1991 National Institutes of Health Consensus Development Conference Panel. Careful screening and follow-up by a multidisciplinary team can help select appropriate patients and improve outcomes.[3]

Given the marked improvement or resolution associated with major comorbidities seen in obesity surgery, the benefit to the properly selected patient with significant comorbidities would seem substantial when compared to the risk. Gastric bypass, the most commonly performed procedure in the United States, is a safe and effective procedure in the proper hands. Laparoscopic gastric bypass procedures seem to be similarly effective with similar mortality rates but a reduction in morbidity, particularly for the most common complications, wound infections and wound hernias. Restrictive procedures have lesser morbidity and mortality but result in lesser weight loss and more weight recidivism. In general, the effect on comorbidities of obesity surgery seems to be proportional to the amount of weight permanently lost.

The effectiveness of obesity surgery on major medical comorbidities in many cases is profound. We have seen that type 2 diabetes and hypertension can be expected to resolve after gastric bypass surgery. That statement should prompt reflection. Moreover, most of the significant comorbidities associated with severe obesity have been found to be ameliorated or improved with obesity surgery, even many previously considered to be irreversible. The remarkable alteration in the natural history of type 2 diabetes, hypertension, dyslipidemia, metabolic syndrome, and cardiovascular risk associated with obesity surgery provoke new thoughts about the nature of these heretofore incurable chronic medical diseases, as well as the mechanisms associating adiposity with their pathogenesis and therapy.

References

1. **Peeters A, Barendregt JJ, Willekens F, et al.** Obesity in adulthood and its consequences for life expectancy: a life-table analysis. Ann Intern Med 2003;138:24–32.
2. **Mokdad AH, Bowman BA, Ford ES, et al.** The continuing epidemics of obesity and diabetes in the United States. JAMA 2001;286:1195–200.
3. **Lara MD, Kothari SN, Sugerman HJ.** Surgical management of obesity: a review of the evidence relating health benefits to risks. Treat Endocrinol 2005;4:55–64.
4. **Martin LF, Tan TL, Horn JR, et al.** Comparison of the costs associated with medical and surgical treatment of obesity. Surgery 1995;118:599–606; discussion 606.
5. **National Institutes of Health.** Gastrointestinal surgery for severe obesity: Consensus Development Conference Panel. Ann Intern Med 1991;115:956–61.
6. **North American Association for the Study of Obesity and the National Heart, Lung, and Blood Institute.** The practical guide: identification, evaluation, and treatment of overweight and obesity in adults. NIH publication 00-4084. Bethesda, Md: National Institutes of Health; 2000.
7. **North American Association for the Study of Obesity (NAASO) and the National Heart.** Clinical guidelines on the identification, evaluation, and treatment of overweight

and obesity in adults: the evidence report. NIH publication 98-4083. Bethesda, Md: National Institutes of Health; 1998.
8. **Fredenheim M.** Hospitals pressured by soaring demand for obesity surgery. New York Times, Aug 29, 2003.
9. **United States National Commission on Diabetes.** Report of the United States National Commission on Diabetes to the Congress of the United States. Publication number 76-1021. Bethesda, Md: US Department of Health, Education, and Welfare, 1975.
10. **Colditz GA, Willett WC, Stamfer MJ, et al.** Weight as a risk factor for clinical diabetes in women. Am J Epidemiol 1990;132:501–13.
11. **Buchwald H, Avidor Y, Braunwald E, et al.** Bariatric surgery: a systematic review and meta-analysis. JAMA 2004;292:1724–8.
12. **Schauer PR, Burguera B, Ikramuddin S, et al.** Effect of laparoscopic roux-en-Y gastric bypass on type 2 diabetes mellitus. Ann Surg 2003;238:467–84.
13. **Sugerman HJ, Wolfe LG, Sica DA, et al.** Diabetes and hypertension in severe obesity and effects of gastric bypass-induced weight loss. Ann Surg 2003;237:751–6.
14. **Pories WJ, Swanson MS, MacDonald KG, et al.** Who would have thought it? An operation proves to be the most effective therapy for adult-onset diabetes mellitus. Ann Surg 1995;222:339–52.
15. **Sjostrom L, Lindroos AK, Peltonen M, et al.** Lifestyle, diabetes, and cardiovascular risk factors 10 years after bariatric surgery. N Engl J Med;351:2683–93.
16. **Pories WJ.** Diabetes: the evolution of a new paradigm. Ann Surg 2004;239:12–3.
17. **Faraj M, Havel PJ, Phelis S, et al.** Plasma acylation-stimulating protein, adiponectin, leptin, and ghrelin before and after weight loss induced by gastric bypass surgery in morbidly obese subjects. J Clin Endocrinol Metab 2003;88: 1594–602.
18. **Yannakoulia M, Yiannakouris N, Bluher S, et al.** Body fat mass and macronutrient intake in relation to circulating soluble leptin receptor, free leptin index, adiponectin, and resistin concentrations in healthy humans. J Clin Endocrinol Metab 2003;88:1730–36.
19. **Pories WJ, Albrecht RJ.** Etiology of type 2 diabetes mellitus: role of the foregut. World J Surg 2001;25:527–31.
20. **Hickey MS, Pories WJ, MacDonald KG, et al.** A new paradigm for type 2 diabetes mellitus: could it be a disease of the foregut? Ann Surg 1998;227:637–44.
21. **Rubino F, Marescaux J.** Effect of duodenal-jejunal exclusion in a non-obese animal model of type 2 diabetes: a new perspective for an old disease. Ann Surg 2004;239:1–11.
22. **Gumbs AA, Modlin IM, Ballantyne GH.** Changes in insulin resistance following bariatric surgery: role of caloric restriction and weight loss. Obes Surg 2005;15:462–73.
23. **Garrison RJ, Kannel WB, Stokes J, et al.** Incidence and precursors of hypertension in young adults: the Framingham Offspring Study. Prev Med 1987;16:234–51.
24. **Jones DW, Kim JS, Andrew ME, et al.** Body mass index and blood pressure in Korean men and women: the Korean National Blood Pressure Survey. J Hypertens 1994; 12:1433–7.
25. **Dornfeld LP, Maxwell MH, Waks AU, et al.** Obesity and hypertension: long-term effects of weight reduction on blood pressure. Int J Obes 1985;9:381–9.
26. **Hypertension Prevention Treatment Group.** The Hypertension Prevention Trial: three-year effects of dietary changes on blood pressure. Arch Intern Med 1990;150: 153–62.
27. **Reisin E, Frohlich ED.** Effects of weight reduction on arterial pressure. J Chronic Dis 1982;35:887–91.
28. **Nguyen NT, Goldman C, Rosenquist CJ, et al.** Laparoscopic versus open gastric bypass: a randomized study of outcomes, quality of life, and costs. Ann Surg 2001;234:279–91.
29. **Stamler R, Stamler J, Grimm R, et al.** Nutritional therapy for high blood pressure: final report of a four-year randomized controlled trial. The Hypertension Control Program. JAMA 1987;257:1484–91.
30. **Hypertension Prevention Trial Research Group.** The Hypertension Prevention Trial: three-year effects of dietary changes on blood pressure. Arch Intern Med 1990;150:153–62.
31. **Elmer PJ, Grimm R Jr, Laing B, et al.** Lifestyle intervention: results of the Treatment Of Mild Hypertension Study (TOMHS). Prev Med 1995;24:378–88.
32. **Tuck ML, Sowers J, Dornfeld L, et al.** The effect of weight reduction on blood pressure, plasma renin activity, and plasma aldosterone levels in obese patients. N Engl J Med 1981;304:930–3.
33. **Weinsier RL, James LD, Darnell BE, et al.** Obesity-related hypertension: evaluation of the separate effects of energy restriction and weight reduction on hemodynamic and neuroendocrine status. Am J Med 1991;90:460–8.
34. **Davis BR, Blaufox MD, Oberman A, et al.** Reduction in long-term antihypertensive medication requirements: effect of weight reduction by dietary intervention in overweight persons with mild hypertension. Arch Intern Med 1993;153: 1773–82.
35. **Stevens VJ, Corrigan SA, Obarzanek E, et al.** Weight loss intervention in phase I of the trials of hypertension prevention: the TOHP Collaborative Research Group. Arch Intern Med 1993;153:849–58.
36. **Kirschner MA, Schneider G, Ertel NH, et al.** An eight-year experience with a very-low-calorie formula diet for control of major obesity. Int J Obes 1988;12:69–80.
37. **Sugerman HJ, Wolfe LG, Sica DA, et al.** Diabetes and hypertension in severe obesity and effects of gastric bypass-induced weight loss. Ann Surg 2003;237:751–6.
38. **Carson JL, Ruddy ME, Duff AE, et al.** The effect of gastric bypass surgery on hypertension in morbidly obese patients. Ann Intern Med 1994;154:193–200.
39. **Sjostrom CD, Lissner L, Wedel H, et al.** Reduction in incidence of diabetes, hypertension, and lipid disturbances after intentional weight loss induced by bariatric surgery: the SOS Intervention Study. Obes Res 1999;7:477–84.
40. **Cowan GS Jr, Buffington CK.** Significant changes in blood pressure, glucose, and lipids with gastric bypass surgery. World J Surg 1998;22:987–92.
41. **Gleysteen JJ.** Results of surgery: long-term effects on hyperlipidemia. Am J Clin Nutr 1992;55:591S–3S.
42. **Brolin RE, Bradley LJ, Wilson AC, et al.** Lipid risk profile and weight stability after gastric restrictive operations for morbid obesity. J Gastrointest Surg 2000;4:464–9.
43. **Buffington CK, Cowan GS Jr, Smith H.** Significant changes in the lipid-lipoprotein status of premenopausal morbidly obese females following gastric bypass surgery. Obes Surg 1994;4:328–35.
44. **Wolf AM, Beisiegel U, Kornter B, et al.** Does gastric restriction surgery reduce the risks of metabolic diseases? Obes Surg 1998;8:9–13.
45. **Busseto L, Pisent C, Rinaldi D, et al.** Variation in lipid levels in morbidly obese patients operated with Lap-Band adjustable gastric banding system: effects of different levels of weight loss. Obes Surg 2000;10:569–77.

46. **Kannel WB, Gordon T.** Obesity and cardiovascular disease. In: Burland W, Samuel PD, Yudkin J, ed. Obesity. London: Churchill Davidson, 1974.
47. **Niebauer J, Hambrecht R, Velich T, et al.** Attenuated progression of coronary artery disease after 6 years of multifactorial risk intervention: role of physical exercise. Circulation 1997;96:2534–41.
48. **Haskell WL, Alderman EL, Fair JM, et al.** Effects of intensive multiple risk factor reduction on coronary atherosclerosis and clinical cardiac events in men and women with coronary artery disease: the Stanford Coronary Risk Intervention Project (SCRIP). Circulation 1994;89:975–90.
49. **Ornish D, Brown SE, Scherwitz LW, et al.** Can lifestyle changes reverse coronary heart disease? The Lifestyle Heart Trial. Lancet 1990;336:129–33.
50. **Kotsis V, Stabouli S, Bouldin M et al.** Impact of obesity on 24-hour ambulatory blood pressure and hypertension. Hypertension 2005;45:602–7.
51. **Alpert MA, Terry BE, Kelly DL.** Effect of weight loss on cardiac chamber size, wall thickness and left ventricular function in morbid obesity. Am J Cardiol 1985;55:783–6.
52. **Sugerman HJ, Baron PL, Fairman RP, et al.** Hemodynamic dysfunction in obesity hypoventilation syndrome and the effects of treatment with surgically induced weight loss. Ann Surg 1988;207:604–13.
53. **Alaud-din A, Meterissian S, Lisbona R, et al.** Assessment of cardiac function in patients who were morbidly obese. Surgery 1990;108:818–20.
54. **The Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults.** Executive summary of the third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (adult treatment program III). JAMA 2001;285:2486–97.
55. **Alberti KG, Zimmet PZ.** Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus provisional report of a WHO consultation. Diabet Med 1998;15:539–53.
56. **Lakka HM, Laaksonen DE, Lakka TA, et al.** The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men. JAMA 2002;288:2709–16.
57. **Giusti V, Suter M, Heraief A, et al.** Effects of laparoscopic gastric banding on body composition, metabolic profile and nutritional status of obese women: 12 months follow-up. Obes Surg 2004;14:239–45.
58. **Hall JE, Brands MW, Henegar JR.** Mechanisms of hypertension and kidney disease in obesity. Ann N Y Acad Sci 1999;892:91–107.
59. **Letiexhe MR, Desaive C, Lefebvre PJ, et al.** Intact cross-talk between insulin secretion and insulin action after postgastroplasty recovery of ideal body weight in severely obese patients. Int J Obes Relat Metab Disord 2004;28: 821–3.
60. **Kelley DE.** Influence of weight loss and physical activity interventions upon muscle lipid content in relation to insulin resistance. Curr Diab Rep 2004;4:165–8.
61. **Pender C, Goldfine ID, Tanner CJ, et al.** Muscle insulin receptor concentrations in obese patients post bariatric surgery: relationship to hyperinsulinemia. Int J Obes Relat Metab Disord 2004;28:363–9.
62. **Cottam DR, Mattar SG, Barinas-Mitchell E, et al.** The chronic inflammatory hypothesis for the morbidity associated with morbid obesity: implications and effects of weight loss. Obes Surg 2004;14:589–600.
63. **van Dielen FM, Buurman WA, Hadfoune M, et al.** Macrophage inhibitory factor, plasminogen activator inhibitor-1, other acute phase proteins, and inflammatory mediators normalize as a result of weight loss in morbidly obese subjects treated with gastric restrictive surgery. J Clin Endocrinol Metab 2004;89:4062–8.
64. **Guilleminault C, Partinen M, Quera-Salva MA, et al.** Determinants of day-time sleepiness and obstructive sleep apnea. Chest 1986;94:32–7.
65. **Hackney JD, Crane MG, Collier CC, et al.** Syndrome of extreme obesity and hypoventilation: studies of etiology. Ann Intern Med 1959;51:541–52.
66. **Sugerman HJ, Windsor A, Bessos M, et al.** Abdominal pressure, sagittal abdominal diameter and obesity co-morbidity. J Intern Med 1997;241:71–9.
67. **Suratt PM, McTier RF, Findley LJ, et al.** Changes in breathing and the pharynx after weight loss in sleep apnea. Chest 1987;92:631–7.
68. **Loube DI, Loube AA, Mitler MM.** Weight loss for obstructive sleep apnea: the optimal therapy for obese patients. J Am Diet Assoc 1994;94:1291–5.
69. **Smith PL, Gold AR, Meyers DA, et al.** Weight loss in mildly to moderately obese patients with obstructive sleep apnea. Ann Intern Med 1985;103:850–5.
70. **Charuzi I, Lavie P, Peiser J, et al.** Bariatric surgery in morbidly obese sleep-apnea patients: short- and long-term follow-up. Am J Clin Nutr 1992;55:594S–6S.
71. **Sugerman HJ, Fairman RP, Baron PL, et al.** Gastic surgery for respiratory insufficiency of obesity. Chest 1986; 90:81–6.
72. **Sugerman HJ, Fairman RP, Sood RK, et al.** Long-term effects of gastric surgery for treating respiratory insufficiency of obesity. Am J Clin Nutr 1992;55:597S–601S.
73. **Sugerman HJ, Baron PL, Fairman RP, et al.** Hemodynamic dysfunction in obesity hypoventilation syndrome and the effects of treatment with surgically induced weight loss. Ann Surg 1988;207:604–13.
74. **Peiser J, Ovnat A, Uwyyed K, et al.** Cardiac arrythmias during sleep in morbidly obese sleep-apneic patients before and after gastric bypass surgery. Clin Cardiol 1985;8:519–21.
75. **Frezza EE, Ikramuddin S, Gourash W, et al.** Symptomatic improvement in gastroesophageal reflux disease (GERD) following laparoscopic roux-en-Y gastric bypass. Surg Endosc 2002;16:1027–31.
76. **Smith SC, Edwards CB, Goodman GN.** Symptomatic and clinical improvement in morbidly obese patients with gastroesophageal reflux disease following roux-en-Y gastric bypass. Obes Surg 1997;7:479–84.
77. **Macgregor A, Greenberg RA.** Effect of surgically induced weight loss on asthma in the morbidly obese. Obes Surg 1993;3:15–21.
78. **Dixon JB, Chapman L, O'Brien P.** Marked improvement in asthma after Lap-Band surgery for morbid obesity. Obes Surg 1999;9:385–9.
79. **Peltonen M, Lindroos AK, Torgerson JS.** Musculoskeletal pain in the obese: a comparison with the general population and long-term changes after conventional and surgical obesity treatment. Pain 2003;104:549–57.
80. **Winiarsky R, Barth P, Lotke P.** Total knee arthroplasty in morbidly obese patients. J Bone Joint Surg Am 1998;8: 1770–4.
81. **Parvizi J, Trousadale RT, Sarr MG.** Total joint arthroplasty in patients surgically treated for morbid obesity. J Arthroplasty 2000;15:1003–8.
82. **Foley EF, Benotti PN, Borlase BC, et al.** Impact of gastric restrictive surgery on hypertension in the morbidly obese. Am J Surg 1992;163:294–7.

83. **Sugerman HJ, Kellum JM, DeMaria EJ.** Risks and benefits of gastric bypass in morbidly obese patients with severe venous stasis disease. Ann Surg 2001;234:41–6.
84. **Bump RC, Sugerman HJ, Fantl JA, et al.** Obesity and lower urinary tract function in women: effects of surgically induced weight loss. Am J Obstet Gynecol 1992;167:392–7.
85. **Sugerman HJ, Windsor A, Bessos M, et al.** Effects of surgically induced weight loss on urinary bladder pressure, sagittal abdominal diameter and obesity co-morbidity. Int J Obes Relat Metab Disord 1998;22:230–5.
86. **Dietel M, Stone E, Kassam HA, et al.** Gynecologic-obstetric changes after loss of massive excess weight following bariatric surgery. J Am Coll Nutr 1988;7:147–53.
87. **Harrison SA, Kadakia S, Lang KA, et al.** Nonalcoholic steatohepatitis: what we know in the new millennium. Am J Gastroenterol 2002;97:2714–24.
88. **Ranlov I, Hardt F.** Regression of liver steatosis following gastroplasty or gastric bypass for morbid obesity. Digestion 1990;47:208–14.
89. **Silverman EM, Sapala JA, Appelman HD.** Regression of hepatic steatosis in morbidly obese patients after gastric bypass. Am J Clin Pathol 1995;104:23–31.
90. **Luyckx FH, Desaive C, Thiry A, et al.** Liver abnormalities in severely obese subjects: effect of drastic weight loss after gastroplasty. Int J Obes Relat Metab Disord 1998;22: 222–6.
91. **Kral JG, Thung SN, Biron S, et al.** Effects of surgical treatment of the metabolic syndrome on liver fibrosis and cirrhosis. Surgery 2004;135:48–58.
92. **Durcan FJ, Corbett JJ, Wall M.** The incidence of pseudotumor cerebri: population studies in Iowa and Louisiana. Arch Neurol 1988;45:875–7.
93. **Bloomfield GL, Blocher CR, Sugerman HJ.** The relationship between elevated intra-abdominal, intrathoracic and raised intracranial pressure. Crit Care Med 1997;25: 496–503.
94. **Sugerman HJ, Felton WL, Sismanis A, et al.** Effects of surgically induced weight loss on pseudotumor cerebri in morbid obesity. Neurology 1995;45:1655–9.
95. **Sugerman HJ, Felton WL, Sismanis A, et al.** Gastric surgery for pseudotumor cerebri associated with severe obesity. Ann Surg 1999;229:634–40.
96. **Printen KJ, Scott D.** Pregnancy following gastric bypass for the treatment of morbid obesity. Am Surg 1982;48:363–5.
97. **Rand CS, Macgregor AM.** Medical care and pregnancy outcome after gastric bypass surgery for obesity. South Med J 1989;82:1319–20.
98. **Friedman D, Cunco S, Valenzano M, et al.** Pregnancies in an 18-year follow-up after biliopancreatic diversion. Obes Surg 1995;5:308–13.
99. **Wittgrove AC, Jester L, Wittgrove P, et al.** Pregnancy following gastric bypass for morbid obesity. Obes Surg 1998; 8:461–4.
100. **Dixon JB, Dixon ME, O'Brien PE.** Pregnancy after Lap-Band surgery: management of the band to achieve health weight outcomes. Obes Surg 2001;11:59–65.
101. **Maddox GL, Lieberman V.** Overweight as a social disability with medical implications. J Med Educ 1969;44:214–20.
102. **Rand CS, Macgregor A, Hankins G.** Gastric bypass surgery for obesity: weight loss, psychosocial outcome, and morbidity one and three years later. South Med J 1986;79: 1511–4.
103. **Powers PS.** Conservative treatments for morbid obesity. In: Deitel M, ed. Surgery for the morbidly obese patient. Philadelphia (PA): Lea & Febiger, 1989. p 27–37.
104. **Schwartz MB, Chambliss HO, Brownell KD, et al.** Weight bias among health professionals specializing in obesity. Obes Res 2003;11:1033–9.
105. **Rand CS, Macgregor AM.** Morbidly obese patients' perceptions of social discrimination before and after surgery for obesity. South Med J 1990;83:1390–5.
106. **Kral JG, Sjostrom LV, Sullivan MB.** Assessment of quality of life before and after surgery for severe obesity. Am J Clin Nutr 1992;55:611S–4S.
107. **Schauer PR, Ikramuddin S, Gourash W, et al.** Outcomes after laparoscopic roux-en-Y gastric bypass for morbid obesity. Ann Surg 2000;232:515–29.
108. **Dixon JB, O'Brien PE.** Changes in comorbidities and improvements in quality of life after Lap-Band placement. Am J Surg 2002;184:51S–4S.
109. **MacLean LD, Rhode BM, Sampalis J, et al.** Results of the surgical treatment of obesity. Am J Surg 1993;165:155–62.
110. **Sugerman HJ, Starkey JV, Birkenhauer RA.** A randomized prospective trial of gastric bypass versus vertical banded gastroplasty and their effects on sweets versus non-sweets eaters. Ann Surg 1987;205:613–24.
111. **Hall JC, Watts JM, O'Brien PE, et al.** Gastric surgery for morbid obesity: the Adelaide Study. Ann Surg 1990;211: 419–27.
112. **Howard L, Malone M, Michalek A, et al.** Gastric bypass and vertical banded gastroplasty: a prospective randomized comparison and 5-year follow-up. Obes Surg 1995;5:55–60.
113. **Yale CE.** Gastric surgery for morbid obesity: complications and long-term weight control. Arch Surg 1989;124:941–6.
114. **Brolin RL, Robertson LB, Kenler HA, et al.** Weight loss and dietary intake after vertical banded gastroplasty and roux-en-Y gastric bypass. Ann Surg 1994;220:782–90.
115. **Capella JF, Capella RF.** The weight reduction operation of choice: vertical banded gastroplasty or gastric bypass? Am J Surg 1996;171:74–9.
116. **Buchwald H, Menchaca HJ, Menchaca YM, et al.** Surgically induced weight loss: gastric bypass versus gastroplasty. Prob Gen Surg 2000;17:23–8.
117. **Sugerman HJ, Londrey GL, Kellum JM, et al.** Weight loss with vertical banded gastroplasty and roux-en-Y gastric bypass for morbid obesity with selective versus random assignment. Am J Surg 1989;157:93–100.
118. **Fielding GA, Rhodes M, Nathanson LK.** Laparoscopic gastric banding for morbid obesity: surgical outcome in 355 cases. Surg Endosc 1999;13:550–4.
119. **Angrisani L, Alkilani M, Basso N, et al.** Laparoscopic Italian experience with the Lap-Band. Obes Surg 2001;11: 307–310.
120. **O'Brien PE, Dixon JB, Brown W.** The laparoscopic adjustable gastric band (Lap-Band): a prospective study of medium-term effects on weight, health and quality of life. Obes Surg 2002;12:652–60.
121. **Belachew M, Belva PH, Desaive C.** Long-term results of laparoscopic adjustable gastric banding for the treatment of morbid obesity. Obes Surg 2002;12:564–89.
122. **Marceau P, Hould FS, Simard S, et al.** Biliopancreatic diversion with duodenal switch. World J Surg 1998;22:947–54.
123. **Sugerman HJ, Kellum JM Jr, Reines HD, et al.** Greater risk of incisional hernia with morbidly obese than steroid-dependent patients and low recurrence with prefascial polypropylene mesh. Am J Surg 1996;171:80–4.
124. **DeMaria EJ, Sugerman HJ, Kellum JM, et al.** Results of 281 consecutive total laparoscopic roux-en-Y gastric bypasses to treat morbid obesity. Ann Surg 2002;235:640–5.
125. **Podnos YD, Jimenez JC, Wilson SE, et al.** Complications after laparoscopic gastric bypass: a review of 3464 cases. Arch Surg 2003;138:957–61.

126. **MacLean LD, Rhode BM, Nohr C, et al.** Stomal ulcer after gastric bypass. J Am Coll Surg 1997;185:1–7.
127. **Sanyal AJ, Sugerman HJ, Kellum JM, et al.** Stomal complications of gastric bypass: incidence and outcome of therapy. Am J Gastroenterol 1992;87:1165–9.
128. **Wu EC, Barba CA.** Current practices in the prophylaxis of venous thromboembolism in bariatric surgery. Obes Surg 2000;10:7–13.
129. **Ido K, Suzuki T, Kimura K, et al.** Lower-extremity venous stasis during laparoscopic cholecystectomy as assessed using color Doppler ultrasound. Surg Endosc 1995;9:310–3.
130. **Kothari SN, Sugerman HJ.** Complications of bariatric surgery. Healthy Weight J 2003;17:52–5.
131. **Nguyen NT, Owings JT, Gosselin R, et al.** Systemic coagulation and fibrinolysis after laparoscopic and open gastric bypass. Arch Surg 2001;136:909–16.