AHA Scientific Statement

Bariatric Surgery and Cardiovascular Risk Factors

A Scientific Statement From the American Heart Association

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The rate of obesity is rising logarithmically, especially in those with severe obesity (body mass index [BMI] >40 kg/m^2). Cardiologists, endocrinologists, internists, family practitioners, and most healthcare professionals are increasingly confronted with the severely obese patient and with postoperative bariatric patients because obesity is associated with significant morbidity and increased mortality. In addition, more adolescents these days are severely obese. Substantial long-term successes of lifestyle modifications and drug therapy have been disappointing in this population. The National Institutes of Health has suggested that surgical therapy be proposed to those patients with BMI >40 kg/m^2 or >35 kg/m^2 with serious obesity-related comorbidities such as systemic hypertension, type 2 diabetes mellitus, and obstructive sleep apnea. When indicated, surgical intervention leads to significant improvements in decreasing excess weight and comorbidities that can be maintained over time. These include diabetes mellitus, dyslipidemia, liver disease, systemic hypertension, obstructive sleep apnea, and cardiovascular dysfunction. Recent prospective, nonrandomized, observational, or case-control population studies have also shown bariatric surgery to prolong survival in the severely obese. Different types of bariatric procedures are being performed. Historically, operative mortality was between 0.1% and 2.0% with more recent data not exceeding 1%. Early complications include pulmonary embolus (0.5%), anastomotic leaks (1.0% to 2.5%), and bleeding (1.0%). Late complications include anastomotic stricture, anastomotic ulcers, hernias, band slippage, and behavioral maladaptation. The number of bariatric operations being performed is increasing tremendously as a result of increasing medical need and the evolution of safer surgical techniques and guidelines. Currently, bariatric surgery should be reserved for patients who have severe obesity in whom efforts at medical therapy have failed and an acceptable operative risk is present.

Definition and Prevalence of Severe Obesity

The terms overweight, obese, and severe obesity refer to a clinical continuum. Excess adiposity should be considered a chronic disease that has serious health consequences. An expert panel convened by the National Heart, Lung, and Blood Institute stated that "obesity is a complex multifactorial chronic disease that develops from an interaction of genotype and the environment." In 1997, the World Health Organization defined obesity as "a disease in which excess fat is accumulated to an extent that health may be adversely affected." Since 1979, the World Health Organization has listed obesity as a disease in its *International Classification of Disease*. Obesity has reached epidemic proportions in the United States and in much of the industrialized world.[1] The standard classification of obesity is expressed in terms of BMI. Obesity is defined as a BMI ≥30 kg/m^2 and may be further subdivided into classes (Table 1).[2] The most rapidly growing segment of the obese population is the severely obese.[3] Between 1986 and 2000, those with a BMI >30, 40, and 50 kg/m^2, are reported to have doubled, quadrupled, and

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This statement was approved by the American Heart Association Science Advisory and Coordinating Committee on January 25, 2011. A copy of the statement is available at http://my.americanheart.org/statements by selecting either the "By Topic" link or the "By Publication Date" link. To purchase additional reprints, call 843-216-2533 or E-mail kelle.ramsay@wolterskluwer.com.

The American Heart Association requests that this document be cited as follows: Poirier P, Cornier M-A, Mazzone T, Stiles S, Cummings S, Klein S, McCullough PA, Fielding CR, Franklin BA; on behalf of the American Heart Association Obesity Committee of the Council on Nutrition, Physical Activity, and Metabolism. Bariatric surgery and cardiovascular risk factors: a scientific statement from the American Heart Association. *Circulation.* 2011;123:1683–1701.

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(*Circulation*. 2011;123:1683-1701.)

***Circulation* is available at http://circ.ahajournals.org** **DOI: 10.1161/CIR.0b013e3182149099**

Table 1. Classification of Body Weight According to BMI in Adults and Children

Adults
Underweight: BMI <18.5 kg/m^2
Normal or acceptable weight: BMI 18.5–24.9 kg/m^2
Overweight: BMI 25–29.9 kg/m^2
Obese: BMI ≥30 kg/m^2
Class 1: BMI 30–34.9 kg/m^2
Class 2: BMI 35.0–39.9 kg/m^2
Class 3: BMI ≥40 kg/m^2 (severe, extreme, or morbid obesity)
Class 4: BMI ≥50 kg/m^2
Class 5: BMI ≥60 kg/m^2
Children (between 2 and 18 y of age)
Overweight: BMI at 85th to 94th percentile
Obese: BMI at 95th percentile or ≥30 kg/m^2, whichever is lower
Severe obesity: BMI at 99th percentile
BMI ≈30–32 kg/m^2 for youths 10–12 y of age
BMI 34 kg/m^2 for youths 14–16 y of age

BMI indicates body mass index.

The National Institutes of Health and World Health Organization classification schema do not include class 4 and 5 obesity.

Source: References 2 and 208.

Table 2. Long-Term Deleterious Health Impacts of Severe Obesity

Shorter life expectancy
Lower quality of life with fewer economic and social opportunities
Cardiovascular disease
Type 2 diabetes mellitus
Stroke
High blood pressure/hypertension
Kidney failure
Dyslipidemia
Obstructive sleep apnea
Acid reflux/gastroesophageal reflux disease
Cancer
Depression
Osteoarthritis
Joint pain

quintupled, respectively, in the United States.[4] It is projected that in the near future, there will be at least 31 million US adults who are severely obese and may qualify for bariatric surgery.

Severe obesity interferes with basic physical functions such as breathing and walking. There are important serious deleterious long-term implications of severe obesity (Table 2). The loss of years of life resulting from obesity is profound. Compared with normal-weight individuals, a 25-year-old severely obese man has a 22% reduction in his expected remaining lifespan. Thus, it was reported from National Health and Nutrition Examination Survey III data that white women 20 to 30 years of age with a BMI ≥45 kg/m^2 will lose 8 years of life and their male counterparts will lose 13 years.[5] Current data from the Behavioral Risk Factor Surveillance System indicate that increases in severe obesity have disproportionately affected blacks, women, young adults, and those of lower socioeconomic status in the American society.[6] Bariatric operations significantly reduce weight, curing or durably improving serious comorbidities. Prospective nonrandomized or case-control population studies have shown that bariatric surgery prolongs life. Various bariatric procedures are available for the management of high-risk severely obese patients. The number of bariatric operations being performed is also increasing logarithmically as a result of increasing medical need and the evolution of safer surgical techniques and guidelines. Currently, Roux-en-Y gastric bypass (RYGB) procedures account for >80% of bariatric operations although the proportion is changing with the availability of the laparoscopic adjustable gastric band procedure (LAGB).[7]

This statement reviews the indications for bariatric surgery, the different surgical options, the complications related to bariatric surgery, and cardiovascular risk factors improvement and outcome benefits of each type of operation, as well as the postoperative management from an interdisciplinary team viewpoint.

Type of Bariatric Surgical Procedures

Surgery for severe obesity has evolved over the past 50 years.[8] Many surgical techniques have been described and abandoned,[9] but numerous different techniques are still in use today. All techniques rely on 1 or both of 2 mechanisms: restriction of food intake and/or the malabsorption of food. Surgical techniques differ in terms of morbidity and mortality rate, magnitude of weight loss, weight loss maintenance, rate of resolution of comorbidities, and side-effects profile. No consensus exists as to which procedure offers the best option overall, nor is there established criterion or algorithm for a made-to-measure procedure for a given patient. Despite the lack of consensus, it is clear that obesity surgery today offers the only effective long-term treatment option for the severely obese patient.

Bariatric surgery can be performed either through a large abdominal incision or by less invasive laparoscopy. Conventional laparotomy used to be the traditional approach to all general surgery. Laparoscopically performed operations carry the advantages of decreased pain, decreased complication rates (ie, pulmonary, thromboembolism, wound infection, hernia), and shorter recovery time with comparable efficacy. Specialized training is required for minimally invasive surgery, but laparoscopic approaches may not be possible in certain situations. Three categories of operations currently exist: restrictive operations, malabsorptive operations, and combined operations. All operations have advantages and disadvantages, with no clear evidence of one being the standard of care. There are currently no large-scale head-to-head randomized trials comparing surgical procedures. Restrictive operations have a lower mortality with a lower rate of surgical and nutritional complications compared with the malabsorptive or combination operations. However, on average, restrictive operations require more frequent postoperative outpatient visits and are associated with a slower and

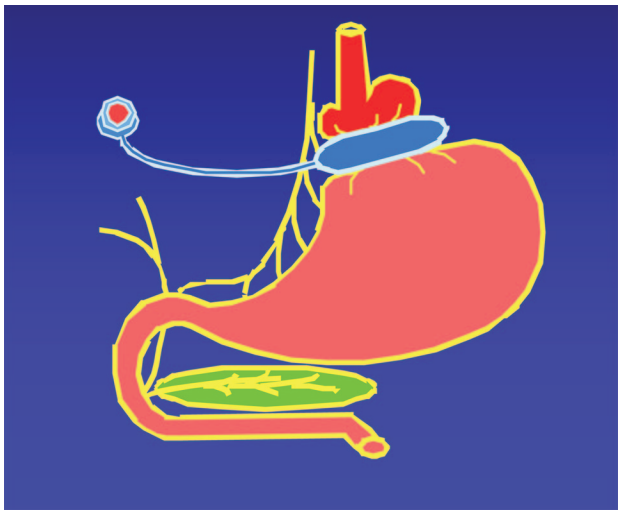

Figure 1. Gastric banding.

lesser weight loss.[10] Combination operations result in the greatest weight loss but require continuous lifelong nutritional surveillance and supplementation.[10]

Purely Restrictive Procedures

Restrictive operations induce weight loss by decreasing the amount of oral intake primarily by the small volume of the pouch and the small diameter of the opening obstructing the passage of food. Appetite suppression or early satiety may be also involved in weight loss.[11] The mechanisms have been hypothesized to be vagal nerve compression or gastric hormone diminution (ie, ghrelin, peptide YY).[12] Restrictive operations include the adjustable gastric band and the sleeve gastrectomy. The adjustable gastric band is an implanted silicone device that includes an inflatable band connected to a reservoir port. The topmost part of the upper stomach is encircled by the band, which prevents expansion circumferentially just below the esophagogastric junction when filled with saline injected through the reservoir port (Figure 1). The injections are performed in the office on a routine basis and result in "adjustment" of the upper stomach pouch outlet until the patient achieves optimum appetite control and satiety. Six band adjustments were reported to be the median adjustments required in the first year and are critical to successful weight loss. No cutting or stapling of stomach or intestine is involved. The gastric band is associated with less loss of fat-free mass compared with other operations but on average also with the slowest and least weight loss[13] (Table 3). The sleeve gastrectomy involves resection of the greater curvature of the stomach, ≈75% of the stomach. The smaller reservoir provides early satiety, and the remnant stomach is associated with a decrease in ghrelin and peptide YY levels.[14]

Hybrid Procedure

A combination of restriction and malabsorption is represented by the RYGB (Figure 2).[15] This operation involves reducing the size of the stomach to 15 mL by cutting off the topmost upper portion and connecting it to the small intestine further down in the digestive system. The stomach remains viable but is bypassed of all food intake, and the new stomach has a dramatically smaller capacity; the duodenum is bypassed of all food intake, resulting in decreased macronutrient absorption, which may modulate postprandial hormonal responses.[16,17] The reduction of appetite may be partially explained by modulations in serum peptide YY and glucagon-like peptide.[18] Bypassing the duodenum also contributes to decreased micronutrient absorption such as iron and calcium, making lifelong supplementation a necessity.[7]

Table 3. Impact of the Type of Weight Loss Surgery on Weight, Lipid Profile, Systemic Hypertension, Diabetes Mellitus, and Side Effects

	Procedures	
	Restrictive	Hybrid
Weight	↓	↓↓
Fat mass	↓	↓↓
Fat-free mass	↓	↓↓
Lipid profile		
LDL cholesterol	→	→
HDL cholesterol	↑	↑
Triglycerides	↓	↓↓
Systemic hypertension	→ or ↓	→ or ↓
Diabetes mellitus	↓	↓↓
Side effects	+	++

↓ indicates decrease; LDL, low-density lipoprotein; →, no change; HDL, high-density lipoprotein; ↑, increase.

Plus signs indicate seriousness of side effects.

The biliopancreatic diversion (BPD) involves resection of the lower two thirds of the stomach, leaving a 250-mL stomach capacity associated with an intestinal bypass, whereby one half of the jejunum and ileum are disconnected from the alimentary tract and reconnected near the terminal

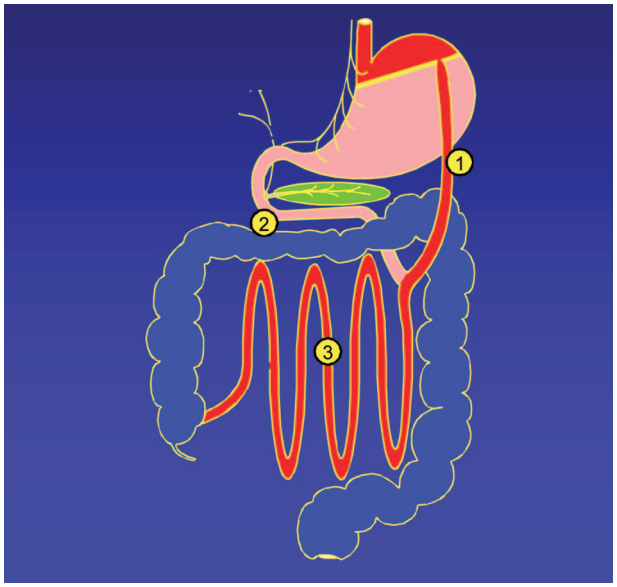

Figure 2. Roux-en-Y gastric bypass. ① indicates alimentary; ②, biliopancreatic; and ③, common.

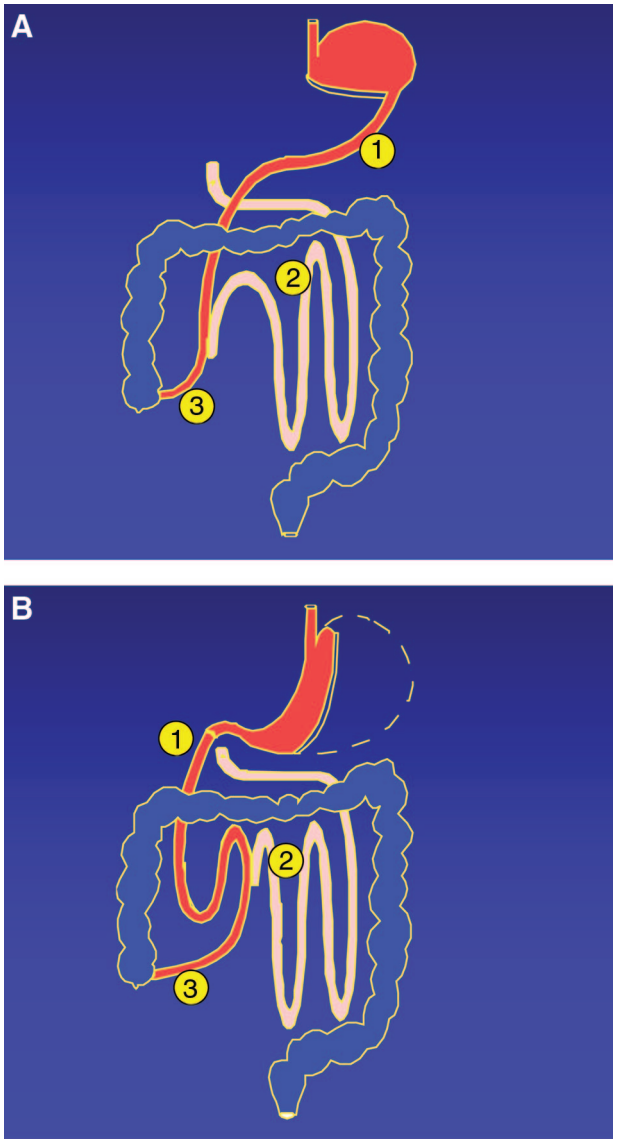

Figure 3. A, Biliopancreatic diversion. **B,** Biliopancreatic diversion duodenal switch. ① indicates alimentary; ②, biliopancreatic; and ③, common.

ileum (Figure 3A). With delayed entry of digestive enzymes into the alimentary tract, the majority of fat and a significant proportion of the protein content ingested are excreted as a result of reduced digestion. Dramatic rapid weight loss results, but many micronutrients are not absorbed and supplementation is required.[7] A modification of the BPD procedure is the BPD–duodenal switch procedure in which the stomach construction is made up of a sleeve gastrectomy with a less drastic intestinal bypass (Figure 3B).[19,20] The advantage of the BPD–duodenal switch is the preservation of the pylorus that connects the stomach to the beginning portion of the small intestine. In addition, the length of the small intestine available for absorption is increased to 100 cm. As a result of these adjustments, this variant procedure carries fewer complications but achieves comparable or even greater weight loss.[21–23] In very high-risk patients, staged approaches may be required in which 1 operation, either gastrectomy or intestinal bypass, is "followed by" the other operation in 2 separate surgical procedures.

Complications of Bariatric Surgical Procedures

Mortality

Operative (30-day) mortality for bariatric surgery ranges from 0.1% to 2%.[10,24,25] Mortality rates depend on several factors: complexity of the operation, patient comorbidities, patient body habitus, and experience of the surgeon and the center. Gastric banding typically has the lowest mortality rate of 0.1%.[24,25] Gastric bypass and sleeve gastrectomy mortality is 0.5%, and malabsorptive operations tend to carry a higher average mortality rate of 1%. More recent data reported a 30-day mortality rate not exceeding 0.3%.[26] The improved mortality rates are secondary to laparoscopic approaches, better anesthesia, and better monitoring and oversight. These numbers can be lower, however, when taking into account healthier patients with lower BMI who have operations performed by an experienced surgeon at an experienced center. Indeed, in the Longitudinal Assessment of Bariatric Surgery Consortium study, none of the 1198 patients who had undergone LAGB died, whereas 0.2% of the 2975 patients who had undergone laparoscopic RYGB and 2.1% of the 437 patients who had undergone open RYGB died.[26] Of importance, patients who underwent open RYGB in that study had a higher BMI and more severe coexisting conditions.[26] The higher end mortality rates have been correlated with visceral obesity, sex, BMI of 50 kg/m^2, diabetes mellitus, sleep apnea, and older age if the patient, particularly if the operation is performed at a lower-volume center.[2,10,26,27]

Early Complications

The severely obese patient is at risk of developing several general complications. They include thromboembolism (1%), pulmonary or respiratory insufficiency (<1%), hemorrhage (1%), peritonitis (1%), and wound infection (2%). Laparoscopy has been instrumental in decreasing these rates.[28] Preventive measures resulting in lower pulmonary complications, lower complications of medical care (ie, medical errors, iatrogenic hypotension), and lower wound infections have also decreased such early morbidity.[29]

Late Complications

Gastrointestinal obstruction can occur, but the cause of the obstruction typically depends on the type of bariatric operation performed. Gastric obstruction associated with gastric banding is due to food entrapment at the narrowed banded area, from overinflation of the band, or from band "slippage," which causes pouching over the band. Symptoms can be resolved by loosening the band, but in certain circumstances, surgical repositioning of the band is necessary.[30] Gastric obstruction associated with gastric bypass or sleeve gastrectomy is due to stenosis of the gastric outlet secondary to scar tissue and may be treated with endoscopic dilation.[31] Intestinal obstruction can occur after gastric bypass and malabsorptive operations and requires urgent surgical intervention.[32] Marginal ulceration between the stomach pouch and the small intestine after

gastric bypass surgery is relatively rare but when present is a frequent source of abdominal pain and anemia. A couple factors explain the impression that this complication may be more prevalent for the clinician[33]: The majority of the studies are retrospective, and because endoscopic studies have been performed only in selected symptomatic patients, no information on outcomes in the asymptomatic patients is available. A systematic review reported the rates of marginal ulcer to be 0% to 4.3%.[34] More recently, Csendes et al[35] published a prospective study assessing the incidence of marginal ulcer 1 month and 1 to 2 years after gastric bypass in 442 consecutive patients. Those investigators found early marginal ulcer (1 month after surgery) in 6% of patients and late marginal ulcer (1 to 2 years after surgery) in only 0.6% of patients. Tobacco, aspirin, and anti-inflammatory nonsteroidal medications should be avoided after gastric operations.[31] Incisional hernias are common after open bariatric surgery and require subsequent surgical intervention. The increased use of minimally invasive techniques have significantly decreased the incidence of this complication.[28] Device-related complications with the gastric band include malfunction of the band, tubing, or reservoir component. Complications are reported to be <1%.[30] A leak in any of these components results in lack of weight loss and subsequently requires surgical replacement. Erosion of the band into the stomach also results in weight loss failure and necessitates removal of the device. Band infection is relatively rare, but infection of the reservoir port can be seen in high-risk patients such as patients with diabetes mellitus and smokers. Most can be treated successfully with antibiotics.[30]

Hypoglycemia has been reported after gastric bypass and can typically be managed successfully with diet modulations. Steatorrhea, diarrhea, and bacterial overgrowth are more common with malabsorptive procedures than gastric banding. Diet modification and antibiotics can be helpful in controlling the severity of these side effects.[36] The most important thing to remember is that a patient with a complication may be best served by being evaluated by an experienced bariatric surgeon. With the rapid growth of bariatric surgery, the maintenance of quality care was the impetus for the creation of Centers of Excellence in the United States. However, the health benefits of this implementation are not clear.[37] A thorough nutrition evaluation can identify characteristics known to be associated with nutrition risks and/or complications of bariatric surgery. The nutrition evaluation should contain a detailed weight history, including a description of the onset, severity, and duration of obesity; a description of current eating patterns; and an assessment of overall nutrition knowledge. Because indications for bariatric surgery include failed attempts to maintain weight loss, a brief summary of self-directed, commercial plans and medically supervised programs should also be documented by the interdisciplinary bariatric surgery team. A patient's motivation for seeking bariatric surgery and knowledge of the procedure, risk, benefits, and impacts on their postsurgical diet and eating behaviors, as well as expectations about weight loss, are all part of a thorough pre–bariatric surgery nutrition evaluation. Nutritional deficiencies of micronutrients are common in obese patients seeking bariatric surgery and should be detected and corrected to avoid postoperative complications. Vitamin B_{12}, vitamin D, folate, and trace minerals such as zinc, iron, and calcium should be screened before bariatric surgery and repleted as needed.[38,39] Vitamin D deficiency is a major public health problem in the United States, and patients with obesity may be at higher risk for developing this deficiency.[40,41] 25-OH Vitamin D should be assessed preoperatively and repleted.[39] Thiamine deficiency, especially in black and Hispanic patients with obesity, is not uncommon and should be screened and repleted.[38] Reported neurological complications of bariatric surgery are peripheral neuropathy, burning feet syndrome, meralgia paresthesia, myotonic syndrome, posterolateral myelopathy, myotonic syndrome, optic neuropathy, Wernicke-Korsakoff encephalopathy, and lumbosacral plexopathy. Although encephalopathies are rare, retrospective reviews identified the prevalence of peripheral neuropathy as being between 7% and 16%.[42,43] Risk factors for peripheral neuropathy included increased glycohemoglobin levels at baseline and increased triglycerides at follow-up, longer duration of hospital stay, prolonged postoperative gastrointestinal symptoms, and a lesser rate of attendance at nutritional clinics. Rate and absolute amount of weight loss were also identified but may represent a lack of appropriate nutritional follow-up.[42,43]

No data exist to support mandatory psychological evaluation, and no predictive value has been found in a patient's psychological profile in terms of outcomes. However, psychological evaluations have become incorporated into most bariatric surgery practices. Although these evaluations often focus on screening for untreated psychopathology, they should also focus on psychoeducational matters and include an assessment of the behavioral and environmental factors that may have contributed to the development of severe obesity, as well as the potential impact of these factors on the patient's ability to make the necessary dietary and behavioral changes to experience an optimal postoperative outcome.[44] Although a psychological evaluation for untreated or undiagnosed psychological conditions may be important for identifying the need for presurgery psychological treatment, the mental health professional will play a greater role in the postoperative care of patients by conducting support groups and/or providing individual psychotherapy.

Regarding exercise, according to 1 report, <20% of severely obese patients engage in structured exercise before undergoing RYGB surgery.[45] Common barriers to regular physical activity include frustration with contemporary exercise guidelines, hesitation to exercise in public places (walking tracks, swimming pools), excessive fatigue/dyspnea with low-level effort, and associated musculoskeletal problems that hinder balance and mobility.[46] However, every patient should be counseled to engage in a preoperative exercise regimen, which may be beneficial in reducing surgical complications and enhancing postoperative recovery. Because reduced peak oxygen consumption (peak Vo_2) level ($<15.8\ mL \cdot kg^{-1} \cdot min^{-1}$ or <4.5 metabolic equivalents [METs; $1\ MET = 3.5\ mL \cdot kg^{-1} \cdot min^{-1}$]) is independently associated with increased short-term complications after bari-

atric surgery, cardiorespiratory fitness should be optimized before bariatric surgery to potentially reduce postoperative complications.[47] Activities corresponding to ≥3 METs are likely to induce somatic fatigue because these aerobic requirements may exceed the ventilatory-derived anaerobic threshold in severely obese patients.[48] This would suggest a training intensity of 2 to 3 METs, which approximates walking speeds of only 1 to 2.5 mph. Such training intensities, if performed in ≥20 min/d of continuous or accumulated exercise 3 to 4 times a week, may serve to increase aerobic capacity and improve quality of life and surgical outcomes.[49]

Effects of Bariatric Surgery on Cardiovascular Risk Factors

Weight

Bariatric surgery provides the greatest sustainable weight loss. Bariatric surgery weight loss effectiveness is commonly expressed in terms of loss of excess weight. The excess weight loss nomenclature as a measurement of weight loss after bariatric surgery has been challenged because of several limitations.[50] The term "excess weight" refers to the difference between the actual weight and "ideal weight" of a given individual. In this context, "ideal weight" is synonymous with "desirable weight," first introduced in 1943 by the Metropolitan Life Insurance Company in their standard height-weight tables for men and women of a medium frame size category. An average weight loss of 50% of excess weight at 5 years may be considered a success, although this typically varies according to the type, aggressiveness, and complexity of surgery. In a meta-analysis of 22 000 bariatric surgery patients, Buchwald et al[24] found that an average percent excess body weight loss of 61% was accompanied by improvements in type 2 diabetes mellitus, systemic hypertension, obstructive sleep apnea, and dyslipidemia. In another meta-analysis,[51] bariatric surgery resulting in a weight loss of 20 to 30 kg maintained up to 10 years was associated with a reduction of comorbidities with an overall mortality rate <1%.[51] Weight loss is more gradual for the restrictive LAGB procedure and may continue for several years.[52] The average long-term (5-year) weight loss is 50% excess body weight loss. Nonetheless, LAGB with longer follow-up has shown poor long-term outcome in a significant number of patients who present with poor primary weight loss or secondary weight regain. A long-term study has reported that only 43% of patient depicted a 7-year success rate defined as a excess weight loss >50%.[53] In addition, a recent meta-analysis found a composite percent excess weight loss of 49.4% for LAGB versus 62.6% for laparoscopic gastric bypass. The authors found percent excess weight loss outcomes for laparoscopic gastric bypass to be significantly superior to those for LAGB at all 3 time points examined (1, 2, and >3 years).[54]

Weight loss after malabsorptive bariatric surgery reaches a nadir at ≈12 to 18 months with an average of 70% excess body weight loss and 35% decrease in BMI[7] with an approximate 10% weight regain over the next decade.[55] RYGB similarly nadirs at 18 months with a 70% excess body weight loss and may have a weight regain after 3 years of up to 15%, for an average of 55% excess body weight loss out to 15 years.[7,55] Sleeve gastrectomy has weight loss patterns similar to RYGB.

Diabetes Mellitus

The vast majority of people with type 2 diabetes mellitus are overweight, with up to 50% to 60% qualifying as obese or severely obese.[56] The presence of diabetes mellitus is associated with a significant 3- to 4-fold increase in the risk of microangiopathy and large-vessel atherosclerosis as manifested by myocardial infarction, stroke, or lower-extremity claudication.[57] There have been significant advances in the management of hyperglycemia and prevention of complications of diabetes mellitus over the past several decades. However, even with currently available approaches, efficacy is incomplete, and the need for multiple medications and intensive medical follow-up leads to considerable expense and poor patient compliance. In addition, in a subset of patients with type 2 diabetes mellitus, currently available therapies are poorly effective and associated with substantial weight gain. Weight loss has long been regarded as the first approach to prevent diabetes mellitus in high-risk subjects and to manage the metabolic derangements of established diabetes mellitus. The attractiveness of weight control as a therapeutic target and the limited efficacy of producing medically induced weight loss have led to increased interest in the effect of surgically produced weight loss to correct the metabolic abnormalities in patients with established diabetes mellitus and to prevent diabetes mellitus in high-risk individuals.

Weight loss prevents the emergence of diabetes mellitus in a significant portion of high-risk patients (eg, those with insulin resistance, impaired glucose tolerance, or impaired fasting glucose).[58,59] In the Diabetes Prevention Program, lifestyle intervention with diet and exercise produced a 5.6-kg weight loss and reduced the emergence of diabetes mellitus by 58% with an average 2.8 years of follow-up.[58] Bariatric surgery with its substantial weight loss reduces the appearance of diabetes mellitus in overweight insulin-resistant subjects. In 1 study[55] in which patients underwent bariatric surgery or conventional treatment, the odds ratio for developing diabetes mellitus in the surgical group was 0.14 (95% confidence interval, 0.08 to 0.24) at 2 years and 0.25 (95% confidence interval, 0.17 to 0.38) at 10 years (both significant at $P<0.001$). Prevention of diabetes mellitus in high-risk subjects by bariatric surgery most likely relates to amelioration of insulin resistance because bariatric procedures have been shown to significantly improve systemic insulin sensitivity.[60,61] Bariatric surgical procedures have also been shown to be effective in reversing the prevalence of the metabolic syndrome or the insulin resistance syndrome as defined by National Cholesterol Education Program Adult Treatment Panel III criteria.[62,63] This perhaps is not surprising given the fact that many of the manifestations of the metabolic syndrome (waist circumference, blood pressure, high fasting blood sugar, high fasting triglycerides, low fasting high-density lipoprotein [HDL] cholesterol) can be traced back to excess adiposity and insulin resistance.

The most clinically relevant impact of surgically induced compared with medically induced weight loss on diabetes

mellitus is the ability of the former to completely reverse established diabetes mellitus in a large percentage of subjects. In a large, long-term, controlled study comparing bariatric surgery and conventional therapy for obesity, established diabetes mellitus was reversed in 21% of the control group and 72% of the surgical group at 2 years of follow-up.[55] At 10 years of follow-up, diabetes mellitus was reversed in 13% of the control group and 36% of the surgical group. The odds ratio of recovery from diabetes mellitus with surgical therapy at 2 years was 8.42 (95% confidence interval, 5.68 to 12.5) and at 10 years was 3.45 (95% confidence interval, 1.64 to 7.28; both significant at $P<0.001$). In a smaller cohort of 165 patients with type 2 diabetes mellitus, 83% experienced remission during a mean follow-up of 9.4 years.[64] All types of bariatric surgery operations have been associated with resolution of diabetes mellitus, but malabsorptive procedures have appeared most effective overall. Predictors of reversal of diabetes mellitus with bariatric surgery have been evaluated; mild disease and larger degree of weight loss predict higher likelihood of reversal.[65] Reduced mortality in subjects undergoing bariatric surgery has been specifically noted in deaths resulting from diabetes mellitus, heart disease, and cancer.[66,67] Another aspect of the reversal of diabetes mellitus observed after bariatric surgery is the rapidity of its appearance, sometimes occurring within days after operation.[68] Improvements in insulin sensitivity have also been documented within days of bariatric surgery and before any observed weight loss.[69] Some have suggested that the rapid resolution of insulin resistance and hyperglycemia that appear before observable weight loss may be related to alterations in gut regulatory peptides produced by the surgical procedure.[70] For example, gastric inhibitory peptide and glucagon-like peptide-1 are 2 incretin hormones produced by K cells, enterochromaffin cells of the proximal small intestine (enteroendocrine duodenal and jejunal mucosa), for gastric inhibitory peptide and by enteroendocrine L cells located in the ileum (glucagon-like peptide-1) that have potent effects on releasing insulin and glucagon from pancreatic islet cells. In obese women with type 2 diabetes mellitus, gastric bypass surgery produced a greater release of the intestinally derived incretin hormones, gastric inhibitory peptide, and glucagon-like peptide-1 compared with a group of women who experienced similar weight loss in response to diet.[71] In addition, postprandial glucose levels decreased more after gastric bypass surgery. However, this study was small, fasting glucose levels were not different in the surgically and medically treated obese diabetic women, and results of the glycemic measures were confounded by the differential approach to the use of hypoglycemic medications in the surgical compared with the medical group. There is also support for the role of the proximal intestine in reversing diabetes mellitus in animal studies that have shown that bypassing a segment of proximal intestine markedly improves glucose tolerance in Goto-Kakizaki type 2 rats with diabetes mellitus independently of food intake, body weight, malabsorption, or nutrient delivery to the hind gut.[72] Some recent studies, however, have questioned the importance of gut enteric hormones for the reversal of diabetes mellitus after surgery, suggesting that rapid resolution of insulin resistance and diabetes mellitus may be related more to the dramatic decreases in energy intake that immediately follow bariatric surgical procedures.[69,73] Therefore, the role of intestinal hormones in mediating the effect of bariatric surgery on the reversal of diabetes mellitus remains unsettled.[71,74]

Although bariatric surgery appears to be an effective means for preventing and/or reversing type 2 diabetes mellitus, it cannot be considered a practical response to the worldwide epidemic of diabetes mellitus because, in the United States alone, 24 million people are currently estimated to have type 2 diabetes mellitus. In addition, bariatric surgery is associated with the potential for both immediate and long-term adverse metabolic consequences.[75] In the absence of other indications for bariatric surgery, it seems most appropriate to consider this intervention for managing metabolic derangements of diabetes mellitus in the subset of obese diabetes mellitus patients whose metabolic state and cardiovascular risk factors are refractory to currently available nonsurgical approaches. In considering the usefulness of bariatric surgery, it is also important to recognize that long-term follow-up is required before assigning a beneficial therapeutic effect in patients with diabetes mellitus because of the potential for regain of weight that has been observed after some surgical procedures.[75]

Lipid Profile and Inflammation

Weight loss surgery results in significant improvements in circulating lipid concentrations and dyslipidemia.[24,55,76–79] Nguyen et al[78] found that 82% of patients requiring lipid-lowering therapy preoperatively were able to discontinue hypolipidemic drug regimens after weight loss surgery. Triglyceride levels are consistently reduced both immediately and after long-term follow-up. Greater impacts are seen after RYGB, with reductions of up to 50% to 60%, in contrast to gastric procedures, in which triglyceride levels are reduced by only 16% to 25%.[24,55,76–78,80–84] Most studies have also shown a significant increase in HDL cholesterol (13% to 47%) after weight loss surgery.[24,55,76–78,80,81,83,85] Although Sjostrom et al[55] found that HDL improvements were greater after RYGB compared with gastric banding, the meta-analysis by Buchwald et al[24] found a significant effect on HDL only with the restrictive procedures. Total cholesterol was modestly reduced[55,76,78,82,83] and low-density lipoprotein (LDL) cholesterol typically was not affected by these procedures.[83,84] As would be expected, the ratio of total cholesterol to HDL was consistently improved (driven by the improvements in HDL) after weight loss surgery[78,80,81,83,85] (Table 3). Weight loss and resultant improvements in hepatic insulin sensitivity likely explain these findings; however, the exact mechanisms for the benefits seen in dyslipidemia after weight loss surgery are not clear. Interestingly, Brolin et al[76,77] found no differences in lipids between patients who maintained weight loss for 5 years compared with those who regained the weight, suggesting that weight loss may not be the primary mechanism for lipid improvements. Dixon and O'Brien[83] also showed that weight loss was not a good predictor of lipid changes. Regardless of the underlying mechanisms, weight loss surgery results in a more favorable lipid profile and therefore an improvement in cardiovascular disease risk.

It is well documented that "at-risk" obesity is associated with an elevated inflammatory state as a result of adipose tissue secretion of a number of proinflammatory cytokines or "adipokines" such as tumor necrosis factor-α, interleukin (IL)-6, and IL-18 and elevated markers of inflammation such as C-reactive protein.[86,87] A number of studies have examined the effects of weight loss surgery on these inflammatory cytokines and markers of inflammation. C-reactive protein levels decrease consistently after weight loss surgery by ≈65% (range, 29% to 89%).[88–100] C-reactive protein concentrations appear to decrease on the basis of the amount of weight loss.[100] IL-6 levels also decrease after weight loss surgery by ≈41%,[89,93,94,98] although 1 study found increased levels of IL-6 after gastric banding.[88] IL-18 levels also appear to be modestly reduced by ≈30% after weight loss surgery.[96,97] Tumor necrosis factor-α levels, however, do not appear to be influenced by these procedures or by important weight loss.[88,89] Finally, adiponectin, an adipose tissue–derived cytokine that is positively associated with insulin sensitivity, is decreased in obesity and increased with weight loss. As expected, adiponectin levels increase significantly, anywhere from 30% to 140%, after weight loss surgery.[93,95,97,98] As with the effects of weight loss surgery on other cardiovascular disease risk factors, weight loss per se is likely the primary mechanism explaining the benefits seen on these markers of inflammation.

Nonalcoholic Fatty Liver Disease

Obesity is associated with a spectrum of liver abnormalities known as nonalcoholic fatty liver disease (NAFLD), characterized by an increase in intrahepatic triglyceride content (ie, steatosis) with or without inflammation and fibrosis (ie, steatohepatitis).[101,102] Of importance, the progression of NAFLD to cirrhosis results in a loss of intrahepatic triglycerides and resolution of steatosis. The prevalence rate of NAFLD is increasing in both children and adults, presumably because of the increase in the prevalence of obesity. In the United States, approximately one third of overweight children and adolescents (defined as BMI ≥95th percentile on the sex-specific BMI-for-age growth chart),[103,104] about one third of all adults,[105] and ≈85% of severely obese adults (BMI ≥40 kg/m^2)[106] have NAFLD. The prevalence of NAFLD is also influenced by ethnicity and race; NAFLD is more common in Hispanic populations but less common in black populations than in whites.[107] Obese people who have NAFLD are at increased risk of having metabolic risk factors for coronary heart disease, including multiorgan insulin resistance,[108,109] increased hepatic very LDL (VLDL)–triglyceride secretion rates, and dyslipidemia,[110] as well as the metabolic syndrome.[111] In addition, among those who have NAFLD, 22% develop diabetes mellitus, 22% develop systemic hypertension, and 25% die of coronary heart disease within 5 to 7 years after diagnosis.[112] The mechanisms responsible for the close relationship between NAFLD and metabolic abnormalities are not clear, and it is not known whether NAFLD is a cause or a consequence of metabolic dysfunction. It is possible that alterations in adipose tissue fatty acid metabolism and the production of inflammatory cytokines are involved. An increased rate of release of fatty acids and inflammatory adipokines from the subcutaneous and visceral fat depots into the systemic and portal circulation in obese subjects with NAFLD can increase hepatic free fatty acids uptake and triglyceride accumulation, impair insulin-mediated skeletal muscle glucose uptake, increase hepatic glucose production, and stimulate hepatic VLDL-triglyceride production.[113–116] In addition, an increased amount of intrahepatic fat, by itself, can impair hepatic insulin action[117] and stimulate VLDL-triglyceride secretion by providing a source of fatty acids for triglyceride production.[118]

Weight loss is an effective therapy for obese patients with NAFLD. Moderate diet-induced weight loss (≈10% body weight) decreases liver fat content and improves liver biochemistries.[119] There has been concern that the large and rapid weight loss induced by bariatric surgery can actually worsen NAFLD by increasing hepatic inflammation and fibrosis.[120,121] However, the preponderance of data from more recent surgical series suggests that weight loss induced by bariatric surgery can decrease steatosis, inflammation, and fibrosis.[122–124] There are also reports supporting that bariatric surgery may reverse cirrhosis.[121,125] In addition, bariatric surgery–induced weight loss has considerable beneficial metabolic effects in the liver manifested by a decrease in hepatic glucose production, hepatic VLDL-triglyceride secretion rate, hepatic gene expression of factors that regulate hepatic inflammation (monocyte chemoattractant protein-1 and IL-8), and fibrogenesis (transforming growth factor-β1, tissue inhibitor of metalloproteinase-1, α-smooth muscle actin, and collagen-α1[I]).[126] These data suggest that bariatric surgery–induced weight loss is an effective therapy for NAFLD in patients with severe obesity by normalizing the metabolic abnormalities involved in the pathogenesis and pathophysiology of NAFLD and by preventing the progression of hepatic inflammation and fibrosis to cirrhosis.

Systemic Hypertension

Most patients with high blood pressure are overweight, and hypertension is ≈6 times more frequent in obese than in lean subjects.[127] In obese patients, both body mass and heightened sympathetic activation contribute to the blood pressure elevation,[128] and sympathetic hyperactivity might account for the resistance to weight loss of some hypertensive patients.[129] The hyperinsulinemia secondary to insulin resistance activates multiple mechanisms leading to systemic hypertension (increased sodium absorption, stimulation of Na^+-H^+ pump activity, reduction of Na^+-H^+ ATPase activity with increased sensitivity for angiotensin II). Furthermore, insulin activates angiotensinogen secretion from adipose tissue,[130] which leads to a higher plasma renin activity and exerts important cardiovascular effects through the sympathetic nervous system. It has been shown that plasma renin activity is significantly increased in obese individuals.[131,132] This increase is associated with higher levels of angiotensin II, which increase tubular absorption of sodium and contribute to systemic hypertension.[133,134] Although the pathophysiological mechanisms explaining the lowering of blood pressure with weight loss are not clear, numerous factors are probably involved. The reduction in blood pressure could also be attributable to reductions in total circulating and cardiopulmonary blood

volume and in sympathetic nervous system activity.[135] The reductions in plasma catecholamines and plasma renin activity, which are associated with decreased sympathetic activity, also probably play a role.[136]

Studies performed 8 to 10 years after gastric banding are controversial. After a transitory lowering of blood pressure values in the first operative years, a complete relapse of the arterial blood pressure was observed, with final blood pressure values being positively related to follow-up time (aging) and the ongoing weight increase. In the long run, in the Swedish Obese Subjects study, no differences in the incidence of systemic hypertension between patients who had an operation and control subjects were observed.[137,138] On the contrary, highly satisfactory results have been obtained after gastric bypass, with long-term resolution of systemic hypertension in nearly three quarters of the subjects with preoperative systemic hypertension who had an operation.[139] In contrast to gastric banding procedures, the systemic hypertension recovery throughout a 10-year follow-up period was related to the amount of weight lost, whereas the relationships with age and with initial body weight were not significant.[140] Indeed, only 20% of the preoperatively hypertensive subjects still had blood pressure values above normal limits.

Sleep Apnea

Obesity is a potent risk factor for the development and progression of obstructive sleep apnea. Before determining the success of weight loss surgery in ameliorating this disease, we must clearly define how the disability of sleep apnea is measured. Apnea is defined as the cessation of airflow for at least 10 seconds. Hypopnea is present when either[141] there is a 30% drop in airflow from baseline for at least 10 seconds with ≥4% desaturation from baseline,[142] there is ≥50% drop in airflow for at least 10 seconds with ≥3% desaturation, or[143] there is an arousal.[141] The severity of obstructive sleep apnea is described by the number of apnea and hypopnea episodes per hour called the apnea-hypopnea index. Mild sleep apnea includes 5 to 15 events an hour; severe sleep apnea includes >30 events an hour.[142] The extent of the disability of sleep apnea best correlates with the number of events and the degree of hypoxemia encountered.[143,144] Unfortunately, published research in the bariatric surgery literature evaluates surgical success by various parameters. After surgical weight loss, success is often measured only by subjective symptom alleviation. End points that measure improvements in the apnea-hypopnea index and in oxygen saturation would be optimal but cannot be found in the literature to any substantive degree. It is widely accepted that weight loss improves obstructive sleep apnea status and that weight gain (even only 10%) worsens obstructive sleep apnea.[145] Few studies claim true resolution of obstructive sleep apnea by any weight loss modality; bariatric surgery is the most successful treatment. Many small series are limited by small sample size, lack of clear definitions of success, selection bias, follow-up bias, and quality of postoperative data. It was reported in a meta-analysis of bariatric surgery outcomes that obstructive sleep apnea improved by 80%.[24] More recently, it was reported that after surgical weight loss, the average apnea-hypopnea index improved by a reduction of 38.2 events per hour or a combined reduction in the apnea-hypopnea index of 71%.[145] Residual obstructive sleep apnea disease occurred in a substantial number of patients after bariatric surgery. Individuals considered cured of obstructive sleep apnea were less obese and younger than those who continued to have sleep apnea symptoms after bariatric surgery. In logistic regression models, both age and follow-up weight <100 kg independently predicted sleep apnea cure. Because <44% of the patients attained an apnea-hypopnea index of <10 per hour, it is important to emphasize continued compliance with obstructive sleep apnea therapy for the majority of patients after weight loss.[145] Repeated polysomnography after bariatric surgery precisely assesses the presence of obstructive sleep apnea and allows retitration of continuous positive airway pressure treatment, which may translate into improved mask fit and higher continuous positive airway pressure therapy compliance.

Thus, a review of the bariatric surgery literature demonstrates significant overall improvement in obstructive sleep apnea. Results are most often reported as improvements in symptoms associated with sleep apnea such as snoring and daytime sleepiness or with polysomnogram reports at 6 to 18 months after a given surgical procedure. However, significant weight loss after bariatric surgery translates into significant but not complete resolution of obstructive sleep apnea.

Cardiac Function

Excess body fat directly influences heart function. Necropsy studies have demonstrated that there is a relationship between heart weight and body weight.[146,147] Symptoms that may be potentially attributable to cardiac involvement (progressive dyspnea with exertion, lower-extremity edema) are often nonspecific in obesity.[1] Physical examination and electrocardiography of obese patients often underestimate the presence and extent of cardiac dysfunction. Severe obesity causes an increase in total blood volume and cardiac output as a result of increased metabolic demand.[148,149] Ventricular chamber dilatation increases wall stress, which triggers increasing myocardial mass in an attempt to diminish wall stress. This results in left ventricular hypertrophy, characteristically from the concentric to the eccentric type.[150,151] Moreover, as with left ventricular mass, longer durations of obesity are associated with poorer left ventricular systolic function and greater impairment of left ventricular diastolic function.[152] Patients who suffer from heart failure either exclusively or predominantly as a result of their obesity are considered to have obesity cardiomyopathy. However, symptoms and signs of obesity cardiomyopathy occur most commonly with a relative weight of >175% or a BMI ≥40 kg/m^2.[153]

Early atherosclerosis is found in obesity. Besides associated cardiovascular risk factors, lipotoxicity resulting from epicardial fat may play a role in the pathophysiology of atherosclerosis in obesity. Epicardial fat may be considered a visceral cardiac fat depot.[154] Under physiologically adapted circumstances, epicardial fat might function as a buffer to protect the cardiac muscle against exposure to excessively high circulating levels of fatty acids, to be used as a possible

local energy source for myocardium when needed, or to defend the myocardium against hypothermia and trauma.[155] In contrast, under pathological conditions, increased epicardial fat deposit may be involved in the generation of cardiotoxic molecules leading to metabolic dysfunction.[156] From its close proximity to the coronary arteries, it has been proposed that epicardial fat might locally mediate the development of coronary artery disease.[157] In a human pathological study, it was observed that part of the left anterior descending artery with an intramyocardial course was without any intimal atherosclerotic lesion, in contrast to the epicardial segment of the same artery in which atherosclerosis was documented.[158] Accordingly, in an animal model using hypercholesterolemic rabbits, epicardial coronary arteries surrounded by adipose tissue developed atherosclerosis, whereas intramyocardial segments of the same arteries remained unaffected.[159] It is speculated that epicardial fat may have a role in the local production of cytokines in blood vessels such as the vasa vasorum through paracrine signaling. Compared with subcutaneous fat, epicardial adipocyte produces more IL-1β, IL-6, tumor necrosis factor-α, and CD45 and less adiponectin.[160] Hence, the proinflammatory environment of adipose tissue and its close proximity to epicardial coronary arteries could promote a proatherosclerotic environment.

Obesity has been shown to be an independent risk factor for atrial fibrillation with an adjusted 50% risk increase for developing atrial fibrillation.[1] Higher radiation dose during atrial fibrillation ablation procedures has been reported.[161] All of the metabolic syndrome components except elevated triglycerides have been reported to be associated with the development of atrial fibrillation, but elevated blood pressure and obesity contributed the most to the increased risk of new-onset atrial fibrillation.[162] Obesity can cause mechanical atrial stretch and dilatation, resulting in a structural substrate predisposing to atrial fibrillation. The increased incidence of stroke in patients with obesity can be partially explained by its association with asymptomatic or undiagnosed atrial fibrillation.

Weight loss has beneficial impacts on functional and structural cardiac status. Indeed, weight loss as little as 8 kg can be associated with a significant decrease in left ventricular mass.[163] In bariatric surgery, improvements in left ventricular systolic function occur mainly in severely obese individuals whose systolic function was significantly depressed preoperatively. In addition, the best improvement in systolic parameters after surgery occurs in those who have been severely obese for a longer period of time.[164,165] Bariatric surgery also has been associated with clinically significant improvement in left ventricular systolic function in patients with severe cardiomyopathy such as patients awaiting heart transplantation.[166,167] There are several electrocardiographic changes with increasing obesity.[1] A prolonged QT is observed in a relatively high percentage of obese subjects, and the association between abnormal corrected QT and BMI is most evident in the severely obese.[168,169] Caution must therefore be taken because weight loss after obesity surgery has been associated with prolongation of the corrected QT interval, which could be deleterious in some individuals by inducing fatal arrhythmias.[170] Although not statistically significant, there was a numerically higher number of cases of sudden death in the bariatric surgery group reported in the Swedish Obese Subjects study.[67]

Survival Benefit

There is increasing, although not definitive, evidence that bariatric surgery provides a significant survival benefit. Eight studies (observational, case-control, and/or nonrandomized) have demonstrated decreased mortality in patients who have undergone bariatric surgery compared with those who have not[66,67,171–176] that is due specifically to decreases in myocardial infarction, diabetes mellitus, and cancer-related deaths. MacDonald et al[171] found that the mortality in patients who underwent bariatric surgery was 9% (n=154) compared with 28% (n=78) in those who did not. Reasons for not undergoing surgery were inability to obtain insurance coverage or choosing not to proceed with surgery. In a similar study, Sowenimo et al[172] found an 81% reduction in mortality for bariatric surgery patients versus those who did not have surgery when compared at 4 years. Christou et al[173] also noted an 89% reduction in mortality in patients in the province of Québec who underwent surgery for obesity compared with a matched cohort of severely obese patients at 5 years. This study also noted a significant decrease in treatment for cancer in the surgical group. Flum and Dellinger[175] found a significant decrease in mortality at 1 and 15 years postoperatively. Busetto et al[174] found that LAGB patients have a lower risk of death than matched cohorts who did not have surgery. Similar findings were observed by Peeters et al[176] and Sjostrom et al,[67] who noted that LAGB patients had a 72% lower hazard of death than an obese population-based cohort. Adams et al[66] found a 40% decrease in mortality after RYGB in Salt Lake City compared with a matched nonsurgical cohort at 2 years with significant decreases in cancer, diabetes mellitus, and myocardial infarction deaths. The Swedish Obese Subjects study reported a 25% mortality decrease in bariatric surgery patients at 10 years compared with a well-matched control population.[67]

Postoperative Management

Diet

The diet after bariatric surgery is designed to meet nutritional needs, which are variably affected by the type of surgery. The diet after weight loss surgery is based on a strong emphasis on texture.[177,178] The purely restrictive procedures, LABG and the vertical sleeve gastrectomy, do not alter food pathway and affect nutritional status only by virtue of restricting overall caloric intake. Because the bypass procedures alter food pathway, there are significant effects on the absorption of micronutrients, and a lifelong regimen of daily supplementation of vitamins and minerals is essential. The BPD–duodenal switch causes malabsorption of both micronutrients and macronutrients, requiring daily supplementation and increased intake of protein.[7] The amount and type of food that patients can eat and the pace at which they can eat and drink vary widely. At the early follow-up appointments, the dietitian will assess adequacy and tolerance of clear and full liquid intake,

vitamin and mineral supplementation, and potential side effects such as dehydration, nausea, vomiting, dumping syndrome, and/or diarrhea that may be related to lactose intolerance. Most patients will be ready to advance their diet from liquid to a soft food diet by 14 days after surgery.[178] All foods should be low in sugar to prevent dumping syndrome. The diet should be advanced as tolerated, and early emphasis should be placed on meeting nutrient needs, adequate hydration, and vitamin and mineral supplementation. Patients undergoing LAGB may adopt poor eating habits because soft foods and high calorie beverages are easy to consume; therefore, as with all of the bariatric procedures, frequent postoperative nutrition counseling, education, and follow-up are important. Early postoperative nutrition and cognitive-behavioral counseling should focus on adequate nutrition and teaching patients behaviors needed to ensure appropriate, healthy weight loss and maintenance. The restrictive and bypass procedures do not cause malabsorption of the macronutrients, fat, carbohydrates, and protein; however, there are risks of micronutrient deficiencies after the RYGB that increase over time. Therefore, strong emphasis should be placed on the need for lifelong supplementation and yearly monitoring of nutritional laboratory values. The BPD–duodenal switch causes both macronutrient and micronutrient deficiencies, including protein malabsorption, which can lead to severe protein calorie malnutrition; in addition, malabsorption of dietary intake of fat and fat-soluble vitamins after BPD–duodenal switch can lead to night blindness, secondary hyperparathyroidism, and bone demineralization in patients not compliant with supplementation.[7] Anemia is usually seen in the setting of sustained sources of bleeding such as menstruation or stomal ulceration. Deficiency after gastric bypass is most common in menstruating women.[179] Women of childbearing age should be closely monitored for folic acid deficiency, and if found, folic acid should be repleted before pregnancy. Episodes of intractable vomiting, sometimes seen in the early post–bariatric surgery phases, can lead to thiamin deficiencies.[178]

Adequate dietary intake of protein, especially in the early months after bariatric surgery, is important to minimize loss of lean body mass. There are no published evidence-based guidelines for meeting protein needs after bariatric surgery. Because patients experience decreased hunger and early satiety, protein intake is encouraged as the primary food consumed in the period immediately after surgery. RYGB patients may experience dumping syndrome after the consumption of simple sugars. Dumping syndrome produces symptoms such as flushing, palpitations, epigastric pain, fatigue, weakness, vomiting, and/or diarrhea. These symptoms can usually be prevented if patients avoid simple carbohydrates, eat slowly, and include a protein source at each meal or snack. Although patients who have had the LAGB do not experience dumping syndrome, they may develop maladaptive eating behaviors because soft foods, especially ice cream and other soft sweets and liquids, may be the food of choice for these patients. No data address essential fatty acid deficiency in patients undergoing RYGB or LAGB. The nutritional aspects of bariatric surgery and severe obesity have been reviewed in depth elsewhere.[7,178]

LAGB Adjustments

The first fill usually occurs 4 to 6 weeks after band placement. Patients undergoing LAGB should be seen in the surgical center every 4 to 6 weeks for monitoring, education, and support and to assess whether they need a band adjustment. The frequency of visits varies among surgical centers. When patients reach an optimal stable adjustment level where appetite is controlled and early satiety is achieved, they no longer need regular fills/adjustments. However, all patients should be seen at least once per year for monitoring of weight, nutritional status, and comorbidity assessment.

Lifestyle Changes

The perioperative management of weight loss surgery patients is ideally provided by an interdisciplinary team with the surgeon, medical specialist, and registered dietician functioning as the primary postoperative caregivers. Patients may also need regular follow-up with other providers if problems occur. Mental health professionals should be available to patients who struggle postoperatively with psychosocial changes. Patients should be seen early and often after bariatric surgery to monitor progress, to guide diet advancement, and to educate about healthful eating and activity. Diet progression to a full solid food diet may take from 6 to 18 months. During this time, patients will go through various stages of food intolerances, changes in food preferences, and changes in hunger and satiety. When weight is stabilized, patients may need a full re-education about healthful eating, with emphasis on increased consumption of fresh fruits and vegetables, limiting foods high in saturated fats, choosing lean sources of protein, and choosing whole grains. Patients may discover that their old habits of skipping meals, not planning and preparing foods, and ignoring hunger and satiety cues may return. Lifelong support and education programs should be provided by surgical centers. Patients should be informed about the possibility of weight regain and be encouraged to work with their bariatric team to evaluate and address lifestyle issues with the first 5- to 10-kg weight regain rather than waiting until they feel completely out of control and, often, too embarrassed to return.

Physical Activity

Exercise programs can be especially difficult for people suffering from severe obesity because any increment level of physical exertion may be difficult and even unsafe. Overall, walking is one of the best forms of exercise. Nevertheless, severe obesity may impair the ability to walk properly, especially when obesity is of the gynoid form. Gluteal fat increases the friction on clothing and skin, making it even more unpleasant to walk. This common problem is often ignored in clinical practice.[180] Individuals often find the exercise regimen difficult to follow because they get extremely tired while walking at the brisk pace recommended by the clinician. Routine activities such as walking require a higher percentage of total exercise capacity (percent of Vo_2max) in obese compared with normal-weight individuals.

Thus, even walking may represent a difficult exercise modality for obese individuals because they can use as much as 56% Vo_2max (some using between 64% and 98% Vo_2max) to meet the demand of such an activity compared with only ≈35% in normal-weight subjects.[181]

Short bouts of exercise (3 to 5 minutes) may be appropriate soon after bariatric surgery, especially when accumulated throughout the day.[46] The duration of exercise should be increased slowly as tolerated. Patients with impact arthropathy may prefer non–weight-bearing exercise modalities such as the stationary cycle or recumbent ergometer. More recently, the International Association for the Study of Obesity stated: "There is compelling evidence that prevention of weight regain in formerly obese individuals requires 60 to 90 minutes of daily moderate intensity activity or lesser amounts of vigorous activity."[182] To prevent weight regain, resistance training is advocated to preserve and restore fat-free mass while maximizing fat loss.[183,184] This may serve to stabilize or modestly increase the resting or basal metabolic rate, which, in conjunction with the caloric expenditure of aerobic exercise, assists with long-term weight loss maintenance. Indeed, recent studies suggest that more intensive exercise regimens after bariatric surgery may be of benefit in the retention of muscle mass, particularly for those experiencing very rapid rates of weight loss.[185]

It appears that severely obese patients who become active after bariatric surgery achieve greater weight losses and quality-of-life improvements than those who remain inactive.[45,186,187] According to 1 report, exercisers lost an average of 6.1 kg more than inactive patients, suffered less depression and anxiety, and had higher scores in general health.[187] Because severely obese individuals are often completely sedentary,[188] they should be strongly encouraged to engage in regular walking so that they can move out of the least fit, least active, "high-risk" cohort. Surgically induced weight loss can also increase peak heart rate, respiratory exchange ratio, and relative oxygen consumption ($mL \cdot kg^{-1} \cdot min^{-1}$), augmenting the exercise capacity.[189]

General Considerations

Although diet, exercise, and behavior modification are mainstay therapy for overweight and obesity, they are disappointingly unsuccessful for long-term treatment of severe obesity. Secondary to the demographic changes encountered in numerous countries and populations, obesity has become a burden on the health systems of industrialized countries and must now be regarded as a serious public-health issue. Strategies should be developed for better long-term weight management of patients, and bariatric surgery is certainly a viable option today. The treatment goal for severe obesity should be an improvement in health achieved by a durable weight loss that reduces life-threatening risk factors and improves performance of activities of daily living. Severe obesity is a complex disease; many factors (genetic, environmental, metabolic, eating disorders) contribute to its development. Medical experience acquired up to now supports the efficiency and safety of surgery for weight loss in severely obese patients on the basis of metabolic profile, cardiac structure and function, and related disorders.

According to the 1991 National Institutes of Health Consensus Conference statement on gastrointestinal surgery for severe obesity, surgery is indicated for patients with BMI ≥40 kg/m^2 or a BMI ≥35 kg/m^2 in the presence of high-risk comorbid conditions.[190] These patients should have attempted prior conventional methods of diet and exercise, should be free of uncontrolled psychiatric disorders, and should be medically sound enough that the benefits of surgery outweigh the risks.[2,7] With improvements in minimally invasive surgical techniques, anesthetic awareness, and expansion of available operative procedures, bariatric surgery has become safer in the older and younger severely obese patient population. Acceptable risk-benefit outcomes with reduction in comorbidities have been published in patients >60 to 65 years of age.[191–196] Conversely, the severely obese adolescent population continues to grow with no effective sustainable treatment available. Since the 1991 National Institutes of Health consensus statement, more data have become available on the safety and effectiveness of bariatric surgery in adolescents for long-term sustained weight loss and comorbidity resolution, particularly when approached on an interdisciplinary level at experienced centers.[197–201] The consensus statement was revisited and updated in 2005 by the American Society for Bariatric Surgery,[25] expanding the indications to adolescents and possibly to individuals with lower BMI (30 to 34.9 kg/m^2) with associated comorbid conditions. Bariatric surgery for patients with BMI of 30 to 34.9 kg/m^2 and comorbidities is aggressively considered because of the poor results of nonoperative weight loss regimens. In a randomized controlled trial, LAGB resulted in superior weight loss, resolution of metabolic syndrome, and improvement in quality of life compared with medical management at a 2-year follow-up.[85] Safe and effective outcomes in this group have been also shown in observational studies.[202,203] Additional long-term data are necessary before surgery for individuals with BMI <35 kg/m^2 becomes standard practice. Similarly, class 5 obese patients (BMI >60 kg/m^2)[2] have been shown to benefit significantly from bariatric surgery with low complication rates.[204,205] Surgical intervention results in sustained weight loss, improvement in comorbidities, and increased survival. Late complications may arise from both nutritional and behavioral aspects. Care by an interdisciplinary team is imperative for best management. At the moment, bariatric surgery should be reserved for patients who have severe obesity in whom efforts at medical therapy have failed and an acceptable operative risk is present. Patients who have had the procedure and are benefiting from it report improvements in their quality of life, social interactions, psychological well-being, employment opportunities, and economic condition. Lifelong medical surveillance after surgery is necessary.

Relative contraindications to surgery include severe heart failure, end-stage lung disease, active malignancy, cirrhosis with portal hypertension, uncontrolled drug or alcohol dependency, and impaired intellectual capacity whereby the patient

cannot understand the lifestyle changes necessary after surgery. Anatomic limitations such as severe intra-abdominal adhesions, giant ventral hernias, large liver, and physiological intolerance of pneumoperitoneum may make a laparoscopic approach impossible and require traditional open laparotomy for access to surgery. These features associated with a given patient should always be evaluated carefully in experienced centers to evaluate the risk/benefit ratio of a given surgical procedure properly. The bariatric patient is best evaluated and subsequently cared for by an interdisciplinary team approach involving the surgeon, a nurse, a dedicated dietician, office personnel, and other specialists such as an exercise specialist when needed. A complete medical evaluation is necessary.[2,206] In addition, a preoperative discussion or teaching seminar that provides information on the risks of the operations and the postoperative recovery, dietary changes, activity, and clinical outcomes is necessary. Expert anesthesiology support involves knowledge of patient positioning, physiology of the obese patient, airway maintenance, and pharmacokinetics in the severely obese.[2] The facility must have appropriately sized equipment, instruments, furniture, and radiographic ability.[207]

Perspectives

The phenomenon of satiety is multifaceted, and the impact of bariatric surgery on parameters that may regulate appetite such as ghrelin, cholecystokinin, gastrointestinal peptides, and perhaps many other neuroendocrine factors may differ, depending on surgery approaches. Well-designed studies are needed. Presently, the major issue in the field of bariatric surgery is the lack of consensus in terms of the diverse procedures available. It is likely that refinement of bariatric surgery techniques for the treatment of severe obesity will evolve to decrease the risks associated with the procedure. One may think that early and late complications can be markedly reduced with increased quality control coordinated by organizations such as national Centers of Excellence programs in the United States. These programs identify sufficient-volume comprehensive bariatric centers that have an interdisciplinary team dedicated to long-term follow-up. However, available data to date suggest that having procedures performed in Centers of Excellence does not ensure better outcomes and is associated with higher expense.[37] More long-term investigations on different bariatric surgery techniques are necessary, at least to evaluate the impact of the beneficial metabolic and cardiovascular changes associated with weight loss procedures on mortality from noncardiovascular and cardiovascular disease, cardiovascular disease manifestations (peripheral vascular disease, coronary artery disease), and cardiac events (myocardial infarction, stroke, malignant arrhythmias). More concerted research is needed to evaluate the benefits of bariatric surgery in the young. Another area likely to receive much attention is the mechanisms involved in weight regulation with in-depth study of the contribution of neurohormones to increased satiety. It is becoming clear that the pathophysiology of obesity is more complex than simply overeating and not exercising. In studies of bariatric surgery patients, appetite control and satiety appear to be key mechanisms for weight loss in gastric banding and gastric bypass patients, whereas calorie malabsorption plays a role in weight loss maintenance in the intestinal bypass operations. Mechanistic studies about metabolic and clinical outcomes are needed. Regarding obstructive sleep apnea, the long-term impacts of mild sleep apnea in this population are unknown. Future work should pool all data in terms of age, race, sex, initial BMI, change in BMI, lung parameters before and after surgery, comorbid cardiopulmonary conditions, waist circumference, and neck circumference to determine risk stratifications and comorbidity resolution on the basis of the heterogeneity of contributing risk factors. In addition, no data exist to support mandatory psychological evaluation, and no predictive value has been found in a patient's psychological profile in terms of outcomes. However, psychological evaluations have become incorporated into most bariatric surgery practices. Although the focus of these evaluations is often on screening for untreated psychopathology, they should also have a psychoeducational focus and include an assessment of the behavioral and environmental factors that may have contributed to the development of severe obesity, as well as the potential impact of these factors on the patient's ability to make the necessary dietary and behavioral changes to experience an optimal postoperative outcome.[44] More important, the mental health professional will play a greater role in the postoperative care by running support groups or providing individual psychotherapy.

Disclosures

Writing Group Disclosures

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/Honoraria	Ownership Interest	Consultant/ Advisory Board	Other
Paul Poirier	Quebec Heart and Lung Institute/Laval University	None	None	None	None	None	None
Marc-André Cornier	University of Colorado, Denver	None	None	None	None	None	None
Susan Cummings	Harvard/Massachusetts General Hospital	None	None	American Dietetic Association*	None	Blue Cross Blue Shield of Massachusetts Advisory Council (no fees paid)*; Food Sciences Corp*	None
Barry Franklin	William Beaumont Hospital	None	None	None	None	Smart Balance Inc*	None
Samuel Klein	Washington University School of Medicine	Barnes-Jewish Hospital†; DSM Nutritional Products†; Longer Life Foundation†; NIH†; Retirement Research Foundation†	None	Merck*	Aspirations Medical Technologies†	Amylin*; Dannon/Yakult*; Enteromedics†; Ethicon/Johnson & Johnson*; Merck Laboratories*; Sanofi-Aventis*; Solae Co†; Shionogi*; Takeda Pharmaceuticals*	None
Theodore Mazzone	University of Illinois at Chicago	Takeda†	None	Merck*	None	Abbott Laboratories*; GlaxoSmithKline*; Merck*	None
Peter McCullough	William Beaumont Hospital	None	None	None	None	None	None
Christine Ren Fielding	New York University School of Medicine	Allergan†	None	Allergan*	None	Allergan*; Ethicon Endosurgery*	None
Sasha Stiles	Tufts University	Kaiser Permanente Trauma Group*	None	ASMBS Compendium CME Lecturer*; Cine-Med*; Robard*	None	BARI MD*	Editor *SOARD**

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10 000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10 000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest.

†Significant.

Reviewer Disclosures

Reviewer	Employment	Research Grant	Other Research Support	Speakers' Bureau/ Honoraria	Expert Witness	Ownership Interest	Consultant/ Advisory Board	Other
John Kral	Downstate Medical Center	None	None	None	None	None	None	None
Robert Kushner	Northwestern University	None	None	Allergan*	None	None	None	None
Richard Milani	Ochsner Clinic Foundation	None	None	None	None	None	None	None

This table represents the relationships of reviewer that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all reviewers are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10 000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10 000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest.

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KEY WORDS: AHA Scientific Statements ■ bariatric surgery ■ diabetes mellitus ■ hypercholesterolemia ■ hypertension ■ metabolic syndrome ■ metabolism ■ nutrition ■ obesity ■ surgery ■ survival

Bariatric Surgery and Cardiovascular Risk Factors: A Scientific Statement From the American Heart Association

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Circulation. 2011;123:1683-1701; originally published online March 14, 2011;
doi: 10.1161/CIR.0b013e3182149099
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Print ISSN: 0009-7322. Online ISSN: 1524-4539