58 Anesthesia for Bariatric Surgery

GAURAV MALHOTRA and DAVID M. ECKMANN

KEY POINTS

- In the United States more than two out of three people are overweight or obese. Worldwide, more people are obese than are malnourished. Obesity is becoming the largest single preventable cause of death and represents major morbidity and mortality.
- Metabolic syndrome includes abdominal obesity, decreased high-density lipoprotein, insulin resistance, glucose intolerance, and hypertension, and is present in approximately 34% of the adult population in the United States alone.
- The single greatest risk factor for sleep apnea is obesity, with the majority of obese patients having increased oral and pharyngeal tissue. This makes ventilation, intubation, and extubation more challenging.
- Choices for medical management of obesity are limited and efficacious achievement with just medical management is uncommon. Changes in behaviors are important for success.
- Surgery for obesity is recommended at a body mass index (BMI) of 40 kg/m^2 or BMI over 30 kg/m^2 with comorbidities expected to respond to weight loss, secondary to surgical therapy, like hypertension, diabetes, and hypercholesterolemia. In clinical trials, long-term survival is better in the surgically treated group over the medically managed.
- Preoperative evaluation should focus on cardiopulmonary issues and securing an airway, along with other concerns such as diabetes, hypertension, and obstructive sleep apnea.
- Anesthetic drugs should be tailored based on lipid solubility and awareness of lingering respiratory depression effects.
- Patient preparation and positioning are keys to successful airway management. Preoperative pressure support ventilation should be used adjunctively if possible.
- Intraoperative ventilation is assisted by complete paralysis, moderate positive end-expiratory pressure, tidal volumes based on ideal body weight, and recruitment maneuvers as needed.
- Common serious postoperative complications are deep vein thrombosis and staple line issues.
- Obese patients presenting for nonweight-loss surgery benefit from anesthetic approaches similar to that used for bariatric surgery.

Obesity as a Disease

Obesity is firmly established as one of the great epidemics of the 21st century.[1] Worldwide, obesity was considered a rarity until the middle of the previous century,[2] but now there are 1.9 billion overweight adults and over 650 million obese people globally including a significant proportion of the adult population in the United States.[3-5] Obesity is a problem that is affecting the younger population as well since over 340 million children and adolescents aged 5 to 19 were overweight or obese in 2016.[6] Current estimates are that more than 2 out of 3 of the U.S. adult population are overweight or obese. Of these one in three adults has obesity and 1 in 13 has extreme obesity with a body mass index (BMI) greater than 40.[7] Among children and adolescents ages 2 to 19, about 1 in 6 are obese, and about 1 in 17 are considered to have extreme obesity.[8] Obesity and its associated health concerns are now major causes of morbidity and mortality resulting in an enormous impact on healthcare spending. Over 300,000 deaths per annum in the United States and about $270 billion[6] in annual healthcare spending are attributable to obesity, placing it second only to smoking as a preventable cause of death.[9]

Obesity can be defined as a disease since it is a physiologic dysfunction of the human organism with environmental, genetic, and endocrinologic etiologies.[9] Obesity most frequently develops when food caloric intake exceeds energy expenditure over a sustained period of time. Factors influencing obesity involve either energy intake or energy expenditure, and are influenced by genetic, behavioral, cultural, and socioeconomic factors.[10] For example, there are syndromes that are associated with obesity, including leptin deficiency, Prader-Willi syndrome, and Lawrence-Moon-Biedl syndrome.[4] Metabolic factors can influence energy regulation, including hormones, peptides, nutrients, uncoupling proteins, and neural regulatory substances emanating from gut, liver, brain, and fat cells, but most of these are not well understood.

The BMI is the most widely applied classification tool used to assess individual weight status.[11] The BMI is specifically defined as the patient's weight, measured in kilograms, divided by the square of the patient's height, measured

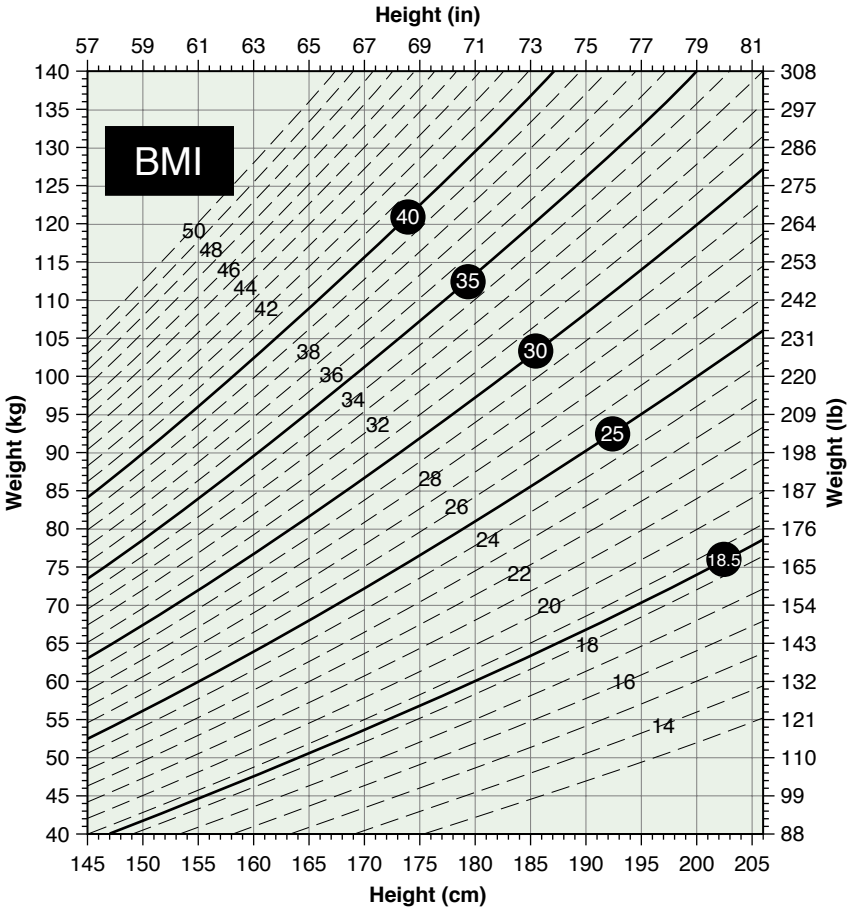

Fig. 58.1 A family of iso-body mass index *(BMI)* curves for BMI ranging from 13 to 50 and mapped on axes for height (in both centimeters and inches) and weight (in both kilograms and pounds).

in meters, yielding a measurement bearing units of kilograms per square meter (kg/m^2). Fig. 58.1 shows a family of iso-BMI curves for BMI ranging from 13 to 50 kg/m^2 and mapped on axes for height (in both inches and centimeters) and weight (in both pounds and kilograms). Most electronic medical record systems are programmed to indicate patient BMI when height and weight inputs are provided. The National Institutes of Health maintains an online BMI calculator and also provides links for downloadable smartphone BMI applications at https://www.nhlbi.nih.gov/health/educational/lose_wt/BMI/bmicalc.htm. Using this system, patients are classified according to BMI and the associated risk of developing health problems is shown in Table 58.1. Patients are considered to be overweight if they have a BMI between 25 and 29.9 kg/m^2, and they are classified as obese with a BMI between 30 and 49.9 kg/m^2. The obese classification is further subdivided into Class 1 (BMI range 30-34.9 kg/m^2), Class 2 (35-39.9 kg/m^2) and Class 3 (40-49.9 kg/m^2). Patients with a BMI of 50 kg/m^2 or greater are classified as superobese. As BMI increases beyond normal weight, the risk of developing serious health problems rises greatly and can be correlated with the individual's waist circumference (Table 58.2). Malnourishment and malnutrition are commonly offered as explanations for the fact that underweight patients are also at increased risk for developing illnesses.

TABLE 58.1 Levels of Risk Associated With Increasing Body Mass Index

Classification	BMI (kg/m^2)	Risk of Developing Health Problems
Underweight	<18.5	Increased
Normal weight	18.5-24.9	Least
Overweight	25.0-29.9	Increased
Obese		
Class 1	30.0-34.9	High
Class 2	35.0-39.9	Very high
Class 3	40.0-49.9	Extremely high
Superobese	≥50	Exceedingly high

BMI, Body mass index.

There are specific diseases commonly associated with obesity, and obesity is often accompanied by multiple, and not single, comorbid states.[7] These frequently include insulin resistance, type 2 diabetes mellitus, obstructive sleep apnea (OSA), asthma, chronic obstructive pulmonary disease, hypoventilation, cardiovascular disease, hypertension, certain malignancies, and osteoarthritis.[1,12-22] Virtually every

TABLE 58.2 Waist Circumference and Risk

Waist	BMI (KG/M²)		
Circumference	**Normal Weight**	**Overweight**	**Obese Class 1**
<102 cm (♂) <88 cm (♀)	Least risk	Increased risk	High risk
≥102 cm (♂) ≥88 cm (♀)	High risk	Very high risk	Increased risk

TABLE 58.3 Health Risks Associated With Increasing Body Mass Index

Metabolic syndrome	30% of middle-aged people in developed countries have features of metabolic syndrome
Type 2 diabetes	90% of type 2 diabetics have a BMI of >23 kg/m²
HTN	5× risk in obesity
	66% of HTN is linked to excess weight
	85% of HTN is associated with a BMI >25 kg/m²
CAD	3.6× risk of CAD for each unit change in BMI
CAD and stroke	Dyslipidemia progressively develops as BMI increases from 21 kg/m² with rise in small particle low-density lipoprotein
	70% of obese women with HTN have left ventricular hypertrophy
	Obesity is a contributing factor to cardiac failure in >10% of patients
	Overweight/obesity plus hypertension is associated with increased risk of ischemic stroke
Respiratory effects (e.g., obstructive sleep apnea)	Neck circumference of >43 cm in men and >40.5 cm in women is associated with obstructive sleep apnea, daytime somnolence, and development of pulmonary hypertension
Cancers	20% of all cancer deaths among nonsmokers are related to obesity (30% of endometrial cancers)
Reproductive function	6% of primary infertility in women is attributable to obesity
	Impotency and infertility are frequently associated with obesity in men
OA	Frequent association in the elderly with increasing body weight—risk of disability attributable to OA equal to heart disease and greater to any other medical disorder of the elderly
Liver and gall bladder disease	Overweight and obesity associated with nonalcoholic fatty liver disease and NASH. 40% of NASH patients are obese; 20% have dyslipidemia
	3× risk of gall bladder disease in women with a BMI of >32 kg/m² 7× risk if BMI of >45 kg/m²

BMI, Body mass index; *CAD*, coronary artery disease; *HTN*, hypertension; *NASH*, nonalcoholic steatohepatitis; *OA*, osteoarthritis;

organ system can be included in the extended list of health risks associated with having an abnormally elevated BMI. A listing of the most common specific disease states along with their obesity-associated risk is detailed in Table 58.3. As a result, obesity is also associated with early death.[11,23]

BOX 58.1 Features Associated With Metabolic Syndrome

Abdominal obesity
Atherogenic dyslipidemia (↑ TGs, ↓ HDL-C, ↑ ApoB, ↑ small LDL particles)
Elevated blood pressure
Insulin resistance ± glucose intolerance
Proinflammatory state (↑ hsCRP)
Prothrombotic state (↑ PAI-1, ↓ FIB)
Other (endothelial dysfunction, microalbuminuria, polycystic ovary syndrome, hypoandrogenism, non-alcoholic fatty liver disease, hyperuricemia)

ApoB, Apolipoprotein-B; *FIB*, fibrinogen; *HDL-C*, high-density lipoprotein cholesterol; *hsCRP*, high-sensitivity C-reactive protein; *LDL*, low-density lipoprotein; *PAI-1*, plasminogen activator inhibitor; *TG's*, triglycerides.

Of all the health risks included in Table 58.3, metabolic syndrome and OSA merit additional attention as they pose special concerns for the anesthetic care of obese patients.

METABOLIC SYNDROME

The clustering of a group of defined metabolic and physical abnormalities is now referred to as the metabolic syndrome.[24] Patients with metabolic syndrome commonly have abdominal obesity, reduced levels of high-density lipoprotein (HDL), hyperinsulinemia, glucose intolerance, hypertension, and other characteristic features[15] as listed in Box 58.1. Specific criteria for diagnosing metabolic syndrome are included in Table 58.4. The diagnosis requires that at least three of the following be present: abdominal obesity, elevated fasting glucose, hypertension, low HDLs, and hypertriglyceridemia.[25] Weight gain with visceral obesity is a major predictor of the metabolic syndrome. The clinical approach uses waist circumference, rather than BMI, to define the adipose mass component contributing to the metabolic syndrome since BMI has been shown to be a relatively insensitive indicator of the risk for obesity-associated metabolic and cardiovascular diseases. Waist circumference, but not BMI, reflects abdominal subcutaneous adipose tissue as well as abdominal visceral adipose tissue and is therefore a better index of central, or truncal, fat mass.

In the United States, approximately 34% of the adult population have metabolic syndrome.[26] Of these, more than 83% meet the criterion of abdominal obesity. The incidence of metabolic syndrome increases with age, with more than 40% of the U.S. population affected by the age of 60 years.[24] Men are affected more commonly than women, and Hispanics and South Asians appear to be particularly susceptible. Its frequency is lower in African American men than in Caucasians. Metabolic syndrome may result from use of some commonly prescribed drugs, including corticosteroid, antidepressant, and antipsychotic agents. Protease inhibitors used to treat human immunodeficiency virus (HIV) infection can induce metabolic syndrome secondary to insulin resistance.

Patients with metabolic syndrome have an increased risk for cardiovascular disease events and are at increased risk for all-cause mortality. Metabolic syndrome increases

TABLE 58.4 Clinical Criteria for Diagnosing Metabolic Syndrome

Central obesity	Waist circumference >102 cm in men Waist circumference >88 cm in women
Plus any two of the following:	
Criteria	**Defining Value**
Triglycerides	150 mg/dL (1.7 mmol/L), or Specific treatment for this lipid abnormality
High-density lipoprotein cholesterol	<40 mg/dL (1.03 mmol/L) in men, or <50 mg/dL (1.29 mmol/L) in women, or Specific treatment for this lipid abnormality
Blood pressure	Systolic blood pressure >130 mm Hg, or Diastolic blood pressure >85 mm Hg, or Treatment of previously diagnosed hypertension
Fasting glucose	110 mg/dL (5.6 mmol/L), or Previously diagnosed type 2 diabetes

the risk of type 2 diabetes, which itself is an important risk factor for atherosclerotic disease and may be considered as a coronary heart disease equivalent.[17,24] Metabolic syndrome is also associated with a variety of other conditions, such as polycystic ovary syndrome, nonalcoholic fatty liver disease, gallstones, sleep disturbances, sexual impotence, and numerous forms of cancer including breast, endometrial, pancreatic, colon, and liver cancer, as detailed in Table 58.3.[27] In multiple trials involving nearly 1900 patients, morbidly obese individuals had much greater weight loss following bariatric surgery than after nonsurgical therapy, with amelioration of most of the diseases associated with morbid obesity in a year's time.[28] Metabolic syndrome is resolved by bariatric surgery in over 95% of patients who achieve the expected weight loss,[29] making it clear that bariatric surgery is a metabolic intervention and not simply a weight management procedure.[30]

Inflammatory processes appear to play an important role in the metabolic syndrome.[20] Adipose tissue has two major functions: storage and release of energy-rich fatty acids and secretion of proteins required for endocrine and autocrine regulation of energy metabolism. Adipocytes exert their metabolic effects by release of free fatty acids, whose release is enhanced by the presence of catecholamines, release of glucocorticoids, increased beta-receptor agonist activity, and reduction of lipid storage mediated by insulin. Visceral adipose tissue has been identified as an important source of proinflammatory cytokines such as tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6), as well as antiinflammatory cytokines such as adiponectin. Increased levels of proinflammatory cytokines likely contribute to the etiology of insulin resistance primarily by obstructing insulin signaling and contributing to downregulation of peroxisomal proliferator-activated receptor-γ, which are fundamentally important regulators of adipocyte differentiation and control. Additionally, insulin resistance may promote inflammation through diminution of insulin's antiinflammatory effects. Lastly, oxidative stress is increased with obesity, primarily as a result of excessive intake of macronutrients and a concomitant increase in metabolic rate. These factors may also contribute to the inflammatory response noted with obesity.[20]

Native immune responses act aberrantly in obese individuals. Natural killer (NK) cell cytotoxic activity is depressed with obesity, as are plasma levels of cytokines such as IL-12, IL-18, and interferon-γ known to regulate NK cell function.[31] Other cytokines (primarily IL-6 and TNF-α) and adipokines (leptin, adiponectin, adipose-derived resistin) are two additional major groups of inflammatory proteins produced and released by adipose and adipose-associated tissue.[20] Both serum and adipose tissue obtained from obese subjects consistently have elevated levels of IL-6 and TNF-α, and circulating levels of IL-6 are consistently increased in individuals having either type 2 diabetes or impaired glucose tolerance. Proteins such as leptin and adiponectin, which are produced primarily by adipocytes, are classified as adipokines. Although leptin is primarily involved in appetite control, its immunologic effects include protection of T lymphocytes from apoptosis and regulation of T-cell activation and proliferation. Reduced leptin levels may increase appetite and slow metabolism, but may also increase susceptibility to the toxicity of proinflammatory stimuli, such as endotoxin and TNF-α. Elevated leptin levels are proinflammatory, and this likely plays an important role in the progression of heart disease and diabetes, especially in obese patients. Serum levels of adiponectin correlate with insulin sensitivity and do not rise in obesity. Significantly reduced adiponectin levels are found in patients having type 2 diabetes. Adiponectin reduces both TNF-α production and activity. It also inhibits IL-6 production. Resistin, an adipokine that induces insulin resistance, is induced by endotoxin and cytokines. Resistin acts at the cellular level to upregulate production of proinflammatory cytokines, most likely through the nuclear factor κB (NFκB) pathway. Resistin appears to present a molecular link among metabolic signaling, inflammatory processes, and the development of cardiovascular disease. Resistin levels have been associated with inflammatory markers apparently independent of BMI in humans.[20]

An understanding of the role of NFκB in insulin resistance is required to fully appreciate the links between obesity and inflammation. Both free fatty acids and TNF-α act via intracellular inflammatory cascade pathways to arrest insulin signaling. This process is mediated by activation of transcription factors present within the cell cytoplasm. Following their translocation to the nucleus, they eventually bind to transcription factors regulating the inflammatory process. The cytoplasm also contains NFκB, another transcription factor whose activation is implicated in a number of diseases, including diabetes. NFκB is also induced by hypoxia, and it increases production of proinflammatory cytokines TNF-α and IL-6, both of which are frequently increased in patients with OSA syndrome.[20] Therefore inflammation provides the common linkage underlying the association between obesity, metabolic syndrome, and OSA.[32]

OBSTRUCTIVE SLEEP APNEA—HYPOPNEA SYNDROME

OSA is a condition characterized by recurrent episodes of partial or complete upper airway collapse occurring during

sleep.[33] An obstructive apneic event is defined universally as the complete cessation of airflow during breathing lasting 10 seconds or longer despite maintenance of neuromuscular ventilatory effort. The definition of an obstructive hypopneic event however may vary depending on the criteria being used for scoring. The Centers for Medicare and Medicaid Services (CMS) defines a hypopneic event as the partial reduction of airflow of 30% or more lasting at least 10 seconds, accompanied by a decrease of at least 4% in the oxygen saturation (SpO_2) as opposed to the American Academy of Sleep Medicine (AASM), which accepts a 3% drop in SpO_2 or a terminal cortical arousal. Additionally, the AASM recommends scoring a third type of respiratory event in which flow limitation is detected and is associated with a cortical arousal. These events are designated respiratory effort related arousals (RERAs).

The diagnosis of OSA can only be made in patients who undergo polysomnography, or a home sleep study.[33] Results of polysomnography are reported as the apnea-hypopnea index (AHI), which is derived from the total number of apneas and hypopneas divided by the total sleep time or the respiratory disturbance index (RDI) which includes RERA. A normal lower limit for AHI has not yet been defined in an epidemiologic study of healthy subjects. Most sleep centers commonly use an AHI between 5 and 10 events per hour as a normal limit. The severity of obstructive sleep apnea/hypopnea syndrome (OSAHS) is arbitrarily defined, but recommendations for disease classification are as follows[33]:

Mild Disease: AHI of 5 to 15 events per hour
Moderate Disease: AHI of 15 to 30 events per hour
Severe Disease: AHI of greater than 30 events per hour

Due to the risks of developing systemic and pulmonary hypertension, left ventricular hypertrophy, cardiac arrhythmias, cognitive impairment, persistent daytime somnolence, and other factors, treatment is recommended for patients with either moderate or severe disease. Treatment partly depends on the severity of the sleep-disordered breathing, but the consensus view is that patients with moderate or severe disease should be treated with continuous positive airway pressure (CPAP) during sleep. Other conservative treatment measures may include weight loss, avoidance of alcohol prior to bedtime, and sleeping on one's side.

Numerous studies have confirmed that obesity is the greatest risk factor for OSAHS, with about 70% of patients (up to 80% of males and up to 50% of females) with OSAHS being obese. Severe sleep apnea disease is more common in men until women reach the age of menopause, and a strong negative correlation between the AHI and minimum SpO_2 has been observed. Importantly, the diagnosis of OSAHS may be missed until the patient presents for surgery. In one study of 170 patients presenting for surgery, only 15% had already been diagnosed with sleep apnea, but on preoperative testing, 76% were found to have OSAHS.[34] A STOP-Bang questionnaire (Box 58.2) can be used to screen patients for OSA, with a score of 5 to 8 identifying patients at risk for moderate to severe disease.[35] We believe it is important for obese patients presenting for bariatric surgery to undergo preoperative polysomnography testing for OSAHS. Preoperative diagnosis and appropriate interventional management can have the following benefits: less postoperative sleep deprivation, improved response to analgesic and anesthetic drugs, and normalization of cardiovascular disturbances.[36]

BOX 58.2 STOP-Bang Questionnaire

1. Snoring: Do you snore loudly (loud enough to be heard through closed doors)?
2. Tired: Do you often feel tired, fatigued, or sleepy during daytime?
3. Observed: Has anyone observed you stop breathing during your sleep?
4. Blood pressure: Do you have or are you being treated for high blood pressure?
5. BMI: BMI more than 35 kg/m²?
6. Age: Age over 50 years old?
7. Neck circumference: Neck circumference >40 cm?
8. Gender: Male?

High risk of OSA: Yes to ≥3 questions.
Low risk of OSA: Yes to <3 questions.
BMI, Body mass index; *OSA*, obstructive sleep apnea.

Anatomically, obese patients with OSAHS typically have increased amounts of adipose tissue deposited into oral and pharyngeal tissues including the uvula, tonsils, tonsillar pillars, tongue, aryepiglottic folds, and lateral pharyngeal walls. An inverse relationship exists between the degree of obesity and pharyngeal area. Deposition of fat in the lateral walls decreases the size of the airway and changes the shape of the oropharynx into an ellipse with a short transverse and long anteroposterior axis.[37-39] This configuration can contribute to both the development and severity of airway obstruction and can also increase the expectation that it will be more difficult to maintain airway patency during mask ventilation and to perform direct laryngoscopy for endotracheal intubation with general anesthesia.[40-41] Neuromuscular blockade should be fully reversed prior to extubation, and low tidal volumes or lung protective ventilation should be employed.

Additionally, airway obstruction following extubation is likely to be complicated by the use of opiate and sedative drugs needed for postoperative pain management because these drugs tend to decrease pharyngeal dilator tone and increase the likelihood of upper airway collapse.[40]

OSA also plays an important role in inflammation and the metabolic syndrome.[20,32] The hypopneic and apneic events that occur in OSAHS are part of the cycle of events that involves both arousal from sleep and oxyhemoglobin desaturation. Sympathetic nervous system activation results as patients with untreated OSA undergo cyclic episodes of hypoxia and reoxygenation. This process leads to elevation of proinflammatory cytokines and may also induce oxidative stress of vascular endothelium, thus inducing an even more heightened state of systemic inflammation in obese patients with OSA.[32] Levels of many different inflammatory mediators, including IL-6, high-sensitivity C-reactive protein (hs-CRP), leptin, TNF-α, IL-1, reactive oxygen species, and adhesion molecules, such as intracellular adhesion molecule-1 (ICAM-1) and vascular cell adhesion molecule-1 (VCAM-1), are increased in patients with OSAHS.[20] Thus obesity, the metabolic syndrome, and

OSAHS are interrelated diseases that significantly alter a patient's inflammatory disease profile and increase multiple health risks, particularly those of cardiovascular and airway origin. Importantly, surgical intervention to cause weight loss has been shown not only to improve obesity-related respiratory disease,[14] but it also can lead to significant and sustained increases in plasma adiponectin levels while decreasing both IL-6 and hs-CRP levels[42] as well as improve NK cell function and increase IL-12, IL-18, and interleukin-γ plasma levels.[31]

Nonsurgical Management of Obesity

The primary goals of nonsurgical management of obesity involve weight loss, the treatment of abnormalities associated with metabolic syndrome, and prevention of type 2 diabetes and cardiovascular disease-related events. Treatment of metabolic syndrome needs to follow an aggressive, multifaceted approach to address multiple underlying metabolic abnormalities and coexistent risk factors simultaneously.[24] An appropriate initial treatment of obesity and metabolic syndrome is therapeutic lifestyle change. This includes dietary modification, weight loss, physical activity, and discontinuation of smoking. The treatment aim is improvement in health, and is the primary reason for advocating weight loss. Modification of energy homeostasis is not easily achieved because a strong brain-gastrointestinal axis drives both food intake and satiety. This axis has hormonal components involving endogenous production of ghrelin, an orexigenic peptide that is produced by the foregut of the stomach and that stimulates appetite.[43] Therefore it is important to monitor the effects of treatment on risk factors and comorbidities at the systems, organ, cell, and molecular levels. Treatment success should be reflected in the decreasing need to treat other coexisting diseases.

The goal for weight loss in therapeutic lifestyle change is not the achievement of normal or ideal body weight (IBW). Even a modest weight loss, in the range of 5% to 10% from the presentation weight, can result in significant initial improvement in the comorbidities of diabetes, dyslipidemia, and hypertension by lowering total cholesterol and triglyceride levels, raising HDL-cholesterol, lowering arterial blood pressure, and lowering blood glucose values while reducing insulin resistance.[24,44] Obesity guidelines stress the need for weight reduction using behavioral change to reduce caloric intake and increase physical activity. A decrease in caloric intake is the most important component in achieving weight loss and increased physical activity is critical in maintaining the lost weight.[45] Reduced-energy diets are more effective and healthier for achieving long-term weight loss. Long-term maintenance of any weight loss achieved is best accomplished with the inclusion of regular exercise as a staple of the weight-reduction regimen. Regular physical exercise improves several risk factors associated with obesity and metabolic syndrome.[46] The standard exercise recommendation is a daily minimum of 30 minutes of moderate-intensity physical activity that is practical to perform. Larger weight loss goals are more appropriate for the more profoundly obese individuals who are contemplating surgical interventions. Even with surgery, ideal weight is hardly ever achieved, and after a number of years at a plateau, weight gain often recurs. In some patients, especially in the presence of severe comorbidities, simple prevention of additional weight gain may be the most reasonable goal.

Beyond the beneficial effects of therapeutic lifestyle change, specific intervention may be required to treat the dyslipidemia and hypertension associated with obesity and metabolic syndrome.[47] Most commonly, patients with metabolic syndrome have elevated triglyceride levels and low HDL cholesterol levels. Many patients receive statin therapy as the treatment of choice when low-density lipoprotein (LDL) cholesterol levels are excessive. Statins reduce cardiovascular disease risk in patients with type 2 diabetes and metabolic syndrome. Ezetimibe, which selectively inhibits intestinal cholesterol absorption, can be combined with statin therapy to further reduce LDL cholesterol by 15% to 20%. Fibrates effectively decrease triglyceride levels while increasing HDL cholesterol. Fibrates lower LDL cholesterol levels mildly, but when combined with statin therapy, fibrates may increase the risk of myopathy. Omega-3 fatty acids decrease triglyceride levels and improve insulin resistance in patients with metabolic syndrome. They are often used in combination therapy with other classes of the hypolipidemic drugs. Nicotinic acid is highly effective in raising HDL cholesterol levels in patients with metabolic syndrome. Nicotinic acid decreases the concentration of small, dense LDL particles and also lowers serum levels of lipoprotein (a).[47]

Dietary salt restriction and therapeutic lifestyle change are the primary means to address hypertension in obesity and metabolic syndrome. According to the 2017 guidelines published by the American College of Cardiology and American Heart Association (ACC/AHA),[48] patients having arterial blood pressure higher than 130/80 mm Hg may require antihypertensive drug therapy. There is no specific antihypertensive drug that is recommended as a first-line treatment in these patients, and generally the goal of antihypertensive therapy requires that polypharmacy be employed. A considerable element of the risk reduction resulting from antihypertensive therapy is decreasing arterial blood pressure.

The treatment of insulin resistance and hyperglycemia in metabolic syndrome, type 2 diabetes, and obesity is usually achieved with oral hypoglycemic drugs.[47] A number of different drug groups (and drugs within each group) that work through various mechanisms of action are available to treat hyperglycemia. These include α-glucosidase inhibitors, sulfonylureas, meglitinides, D-phenylalanine derivatives, biguanides, and thiazolidinediones.[47] Anesthetic implications include the need to assess and treat abnormal blood glucose levels in the perioperative period while being especially careful in the use of insulin in patients who are both insulin resistant and temporarily unable to continue on oral medication. At present, the optimal anesthetic management of patients who are taking metformin is not clear. There is a serious potential for postoperative lactic acidosis that can develop in patients using this drug. This possibility has led some physicians to routinely cancel or delay surgical procedures if metformin has been ingested within 48 hours of the scheduled surgery. Other physicians, however, have their patients continue taking metformin, both before and after surgery, without interruption if possible. Recent evidence indicates that patients taking metformin have a

reduced risk for complications. It appears that metformin may be safely used in the perioperative period.[49]

Patients with metabolic syndrome and obesity may also be prescribed antiplatelet therapy. The AHA recommends that low-dose aspirin be used as a form of primary prevention in patients with metabolic syndrome whose 10-year risk for cardiovascular disease is 10% or greater as determined by Framingham risk scoring.

BEHAVIORAL INTERVENTIONS AND MODIFICATION

Behavioral interventions and behavioral modification are essential for obese patients to change their learned habits related to eating and physical activity in order to produce weight loss and long-term weight reduction.[47] This applies both for nonsurgical and surgical approaches to weight loss. The key features of typical behavioral programs include self-monitoring, goal setting, nutrition and exercise education, stimulus control, problem solving, cognitive restructuring, and relapse prevention. Patients often benefit from referral to multidisciplinary weight loss programs that incorporate diet, physical activity, and behavioral interventions to achieve their weight loss goals because these combined interventions provide the best weight loss and weight maintenance results without pharmacologic or surgical intervention. However, it is essential to identify and treat patients with eating disorders or major psychiatric disorders who require specialized psychiatric and psychological treatment to achieve meaningful weight loss.

PHARMACOTHERAPY FOR WEIGHT LOSS

Recommendations for pharmacotherapy as a treatment of obesity, first and foremost, stress lifestyle and behavioral modifications as the initial approaches to initiate weight loss.[27] Patients who do not reach their established reasonable weight loss goals by a combination of diet and exercise may be directed to pharmacotherapy to increase weight loss. There are weight loss drugs that have been approved by the Food and Drug Administration (FDA) and are currently prescribed for long-term use. These are typically used adjunctively with diet and exercise for patients having a BMI of 30 or greater (≥27 for patients with obesity-related risk factors or comorbid diseases). In current practice, there are only two categories of weight loss drugs: appetite suppressants and lipase inhibitors. Three drugs are currently available for the specific indication of weight loss: phentermine, lorcaserin,[50] and orlistat.[47] Phentermine, an adrenergic reuptake inhibitor, augments adrenergic signaling within the central nervous system and peripheral tissues. Phentermine decreases appetite and food intake and increases resting metabolic rate to promote weight loss. Its side effects include tachycardia and hypertension. Lorcaserin is a selective 5-HT_{2C} receptor agonist that reduces food intake through the activation of pro-opiomelanocortin. Due to its selectiveness for the 5-HT_{2C} receptor, lorcaserin has a better safety profile than the previous serotonin agonists for weight loss that have since been removed from the U.S. market due to increased risk of stroke and acute coronary syndrome. Lorcaserin should not be used in patients on selective serotonin reuptake inhibitors (SSRIs) or monoamine oxidase inhibitors (MAOIs) due to the risk of serotonin syndrome, which can be life threatening. Orlistat, a lipase inhibitor, reversibly binds to lipase and prevents both absorption and digestion of certain dietary fats. Because orlistat also interferes with the absorption of fat-soluble vitamins, patients using this drug need to supplement fat-soluble vitamins A, D, E, and K. It has significant gastrointestinal side effects including diarrhea, steatorrhea, flatulence, fecal incontinence, and oily rectal discharge.

ALTERNATIVE PHARMACOLOGIC OPTIONS

Allison and colleagues [51] reviewed the literature on dietary and herbal medications for weight loss. These agents are marketed as "food supplements," thereby escaping the purview of the FDA. Even though these supplements cannot legally claim to treat a disease, they can claim to reduce the risk of a disease. According to the review, claims for weight loss have been made for multiple products such as chitosan, chromium picolinate, conjugated linoleic acid, ephedra alkaloids (ma huang),[52] and *Garcinia cambogia*. [51] Most of the reports involving these compounds are from poor quality studies without any randomization, control groups, or blinding, thereby placing in question both efficacy and safety of these compounds. The only studies involving herbals that have consistently demonstrated weight loss involve combinations of ephedrine and caffeine.[53-54] Pharmacologically, this is expected because ephedrine, an adrenergic agonist, is known to be an appetite suppressant and a thermogenic agent. For this reason, *ma huang*, a natural source of the ephedra alkaloid, is added to most, if not all, dietary supplements marketed for weight loss. The success of ephedrine as a weight loss agent in combination with caffeine and or aspirin is well established. Unfortunately, multiple cases of cardiac and neurological issues, including hypertension, stroke, seizure, and even death have been reported,[55] possibly related to the inconsistent doses in the preparations and the lack of medical supervision in people consuming these products for weight loss. Consequently, the National Institutes of Health has banned these products from any recommended weight loss regimen.

IMPLANTED ELECTRICAL STIMULATORS

Implantable gastric stimulators are subcutaneously placed devices that resemble cardiac pacemakers and stimulate along the lesser curvature of the stomach. These gastric stimulators have been tried since the early 2000s and have been found to cause modest weight loss with few side effects.[56-57] Numerous clinical trials conducted in the United States and Europe have shown some promising results. Most studies demonstrated weight loss during the first 12 months; however only a few studies had a follow-up period of longer than 1 year.[58] One study documented a 25% excess weight loss, improved response to oral glucose tolerance test, decreases in arterial blood pressure, and an improvement in symptoms of gastroesophageal reflux disease (GERD), along with an increased parasympathetic drive. No serious side effects were noted in the first 65 patients reported on in this study[56] or in the 20 patients followed in another.[57] Ghrelin levels may play a role in the success of these devices.

Surgical Management of Obesity

Adult obesity is clearly rooted in childhood obesity. Unfortunately, pediatric obesity is the most common childhood nutritional disorder in the United States. This is one of the tragedies of the current obesity epidemic: the significant and increasing prevalence of obesity in the young. It has attained a level of controversy that has involved the placement of vending machines in schools and the availability of sweetened snacks and soft drinks in public places as public policy is developed to help control the epidemic of pediatric obesity. The diagnosis of pediatric obesity is commonly defined using criteria developed by the Centers for Disease Control and Prevention (CDC) using BMI-, age-, and sex-specific clinical growth charts.[59] These percentile curves apply to the ages of 2 to 20 years, and because BMI exhibits nonlinear variation during growth, percentile ranges are utilized. The CDC defines children possessing BMIs in the 5th to 85th percentiles as having "healthy weight." Children whose BMI is in the 85th to 95th percentile are "at risk for overweight," while those having a BMI above the 95th percentile are classified as "overweight." These classifications were previously regarded as "overweight" and "obese," respectively. This switch in terminology continues to be a source of confusion. Exceeding the 99th percentile is referred to as extreme pediatric obesity.

The National Health and Nutrition Examination Survey 2011–14 data show that the prevalence of obesity in children for age 2 to 5, 6 to 11, and 12 to 19 years are 9.4%, 17.4%, and 20.6%, respectively.[60] There does not appear to be an influence of gender on the prevalence of obese children above the age of 6 years. While the prevalence of obesity among children of ages 6 to 11 and 12 to 19 has steadily increased since 1988, there has been a slight recent decline in obesity among ages 2 to 5 years. A number of developing countries have childhood prevalence rates higher than that of the United States,[61] indicating that adolescent obesity is becoming pervasive.

Treatment of pediatric obesity is based on the balance of caloric intake and expenditure. These lifestyle changes are most successful with family intervention and support. The three pillars of success in this treatment are: better eating habits, increased physical activity, and decreased sedentary activity.[62] Success involves more than education of child and family; it incorporates goal setting, self-monitoring, incentives, and stimulus control. Lifestyle interventions remain the treatment of choice in pediatric obesity, but concomitant pharmacotherapy may be beneficial in some patients. Orlistat, which is covered elsewhere in this chapter, should be considered as second-line therapy for pediatric obesity.[63]

Although in 2000, less than 1% of all bariatric surgery patients in the United States were younger than 20 years of age,[64] the total number continues to increase. More adolescents are presenting for weight loss surgery, after failing to reduce weight and/or comorbidities in medically supervised and pharmacologically assisted weight loss programs. Even though many adult bariatric surgery programs are well qualified to provide safe and effective perioperative care for these patients, they may not be as well equipped to handle the unique metabolic and psychological needs of teenagers. Because of the shorter duration of obesity and fewer age-related comorbidities, adolescent patients have shorter length of stay and lower immediate postoperative mortality when compared to adults.[64]

Consent is an important issue in this population. Is parental consent to a semi-elective, relatively high-risk procedure appropriate? Does the child really understand the concept of high risk of death in the month after surgery? These and other such questions of ethics in medicine need to be addressed, but are beyond the scope of this chapter.

The rapid increase in the prevalence of both morbid obesity and superobesity, together with the increased risk of early demise within the obese population, has significantly increased the number of bariatric surgical procedures performed annually. The term bariatric surgery refers to surgical alteration of the small intestine or stomach with a view toward producing weight loss. It is estimated that over 216,000 bariatric surgeries are being performed annually in the United States. Care of obese patients is not limited to obesity surgery, however, as these patients present for all types of operations. Nonetheless, the benefits of surgical treatment of obesity now appear to have clear endocrinologic and cardiovascular value for reversing pathophysiologic effects of metabolic syndrome, type 2 diabetes, and other comorbidities of obesity, in addition to providing a mechanical means of enhancing weight loss.[65-71] Several alternative procedures in bariatric surgery have evolved from early procedures based on creating malabsorptive gut pathways to newer procedures aimed at altering hormonal regulation of caloric intake drive and satiety.[72] Primarily the available options can be separated into one of two groups: (1) operative procedures and (2) minimally invasive procedures that combine gastric restriction with induction of nutrient malabsorption.

OPERATIVE PROCEDURES

Surgery for obesity is one of the fastest growing surgeries performed worldwide.[73] The operative group can be divided into gastric restrictive procedures and procedures that combine gastric restriction with induction of nutrient malabsorption.[74-83] These procedures can be performed using an open, laparoscopic, or robot-assisted approach. In general, minimally invasive (laparoscopic, robotic) approaches are preferred over an open procedure as they have a lower complication rate, hospital stay, and morbidity.[84] The rate of open bariatric procedures has continued to decrease steadily to the extent that nearly 90% of surgical bariatric cases are now performed laparoscopically.[85]

RESTRICTIVE PROCEDURES

The surgical goal of restrictive operative procedures is to reduce and limit the patient's food intake capacity.[80] This is most commonly achieved by reducing the stomach's reservoir capacity. The vertical band gastroplasty was a widely practiced restrictive procedure in the previous three decades, but it was replaced by the minimally invasive laparoscopic gastric band procedure (LGB), which was very popular. However, in recent years, the percentage of bariatric procedures represented by LGB surgery performed has also decreased from 35% in 2011 to approximately 3% in 2016.[86] This decline in LGB operations is probably secondary to only modest amount of weight loss and high rates of band revisions and adjustments required. Recently, the sleeve gastrectomy (SG) has become

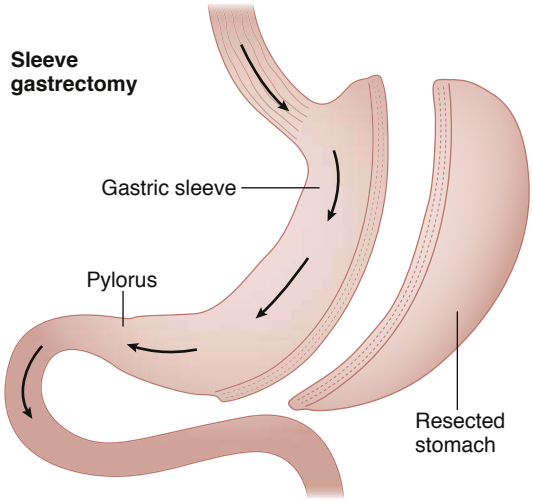

Fig. 58.2 Sleeve gastrectomy: a tubular stomach is created as the fundus and greater curvature of the stomach are removed around a 32F to 40F bougie.

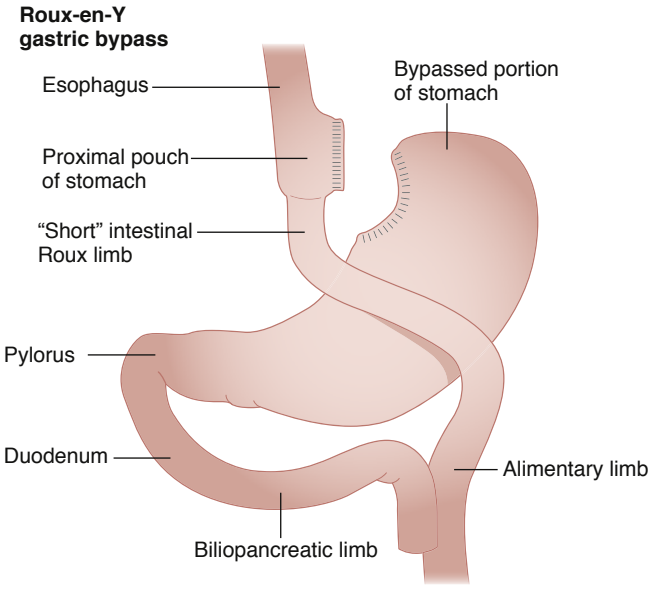

Fig. 58.3 Roux-en-Y gastric bypass: part of the stomach is detached from the rest, creating a small pouch. The pouch is connected to a lower part of the small intestine by a piece of small intestine, resembling a Y. As a result, parts of the stomach and small intestine are bypassed. However, digestive juices (bile acids and pancreatic enzymes) can still mix with the food, enabling the body to absorb vitamins and minerals and reducing the risk of nutritional deficiencies.

the most commonly performed surgery for obesity. As illustrated in Fig. 58.2, in SG surgery, a tubular stomach is created around a 32 to 40 French bougie by removing the greater curvature of the stomach. The SG has been shown to be safe and effective, with a mean excess weight loss of approximately 65%.[87] The weight loss noted after a SG is not only due to the restrictive stomach, but also due to the decrease in hormonal level of ghrelin that regulates appetite and food intake.[88]

MALABSORPTIVE PROCEDURES

The surgical goal of malabsorptive procedures is to cause weight loss both by gastric restriction and nutrient malabsorption. Historically, early surgical procedures involved creation of a long jejunoileal bypass. Patients achieved significant weight reduction but this was commonly accompanied by an unacceptable incidence of serious vitamin and protein malabsorption, osteoporosis, and hepatic failure.

Today, gastric bypass (GBP), which is illustrated in Fig. 58.3, and biliary pancreatic diversion (BPD) are the two most commonly performed malabsorptive operations, and both have achieved success in safety and efficacy.[80] GBP is more commonly performed than BPD, and it is considered the safer of the two malabsorption procedures. GBP surgery involves the creation of a small gastric pouch by stapling or banding the stomach. This results in an element of gastric restriction. GBP also involves the creation of a Roux-en-Y anastomosis, in which the small gastric pouch is directly connected to the middle portion of the jejunum.[89] The OrVil device, shown along with nontapered and tapered bougies in Fig. 58.4, may be used by the surgeon to assist with creation of the anastomosis. The orogastric portion of the OrVil device is passed through the oropharynx and then pulled through by the surgeon via a gastrostomy, until the anvil portion of the OrVil device reaches the stomach. It is then attached to a stapler to create the anastomosis. The path for food through the gastrointestinal tract subsequently bypasses the gastric remnant and the upper portion of the duodenum. This procedure can be performed with a laparotomy incision or by using laparoscopic techniques.[90-91]

Fig. 58.4 32F tapered bougie *(top)*, 40F nontapered bougie *(middle)*, and OrVil device *(bottom)* commonly used during bariatric surgery.

Although this is considered to be a safe operation, the associated perioperative mortality is higher than that of restrictive procedures.[77] Additionally, there are some important long-term complications such as vitamin B_{12} deficiency, anemia, incisional hernia, cholelithiasis, and staple line failure that occur with significant frequency. Often a cholecystectomy is performed concurrently to prevent future cholelithiasis.[92] BPD is also a form of Roux-en-Y GBP reconstructive alimentary anatomy in which the bypassed intestinal segment includes the portion of duodenum at the point of entry of biliary and pancreatic secretions. This allows the bypassed intestine to be exposed to bile and pancreatic juice. The antrum is removed to avoid peptic ulceration, and food intake is only affected to a limited degree. The essential element of this operation is division of the small intestine about midlength, with the distal end of the alimentary limb being anastomosed to a generous gastric remnant. The more proximal biliopancreatic limb is anastomosed to the alimentary limb about 50 cm from the ileocecal valve. Following either the GBP or BPD form of operation, patients require life-long follow-up and may need micronutrient replacement long term.

MINIMALLY INVASIVE PROCEDURES

There have recently been a few devices approved in the United States for weight loss. One such device is the intragastric balloon that acts in a restrictive manner.[93] The intragastric balloon is placed in the patient's stomach endoscopically and is filled with saline to provide a sensation of fullness and satiety. The device can be kept in the patient's stomach for a maximum of 6 months and has been shown to decrease weight by an average of 6.8%. Most common complications of the intragastric balloon are related to gastrointestinal symptoms such as nausea, vomiting, halitosis, GERD, and abdominal discomfort; however serious complications such as balloon rupture, migration, and intestinal obstruction are possible.[94]

Another device that has been approved for use in the United States is an endoscopically placed percutaneous gastrostomy tube to facilitate stomach emptying. The device can be used for draining ingested foods and in preliminary studies has been shown to decrease total body weight (TBW) by approximately 12%.[95] The most common side effects were abdominal discomfort and peristomal irritation; however serious complications such as abdominal pain requiring hospitalization and peritonitis were noted infrequently.

HEALTH BENEFITS OF BARIATRIC SURGERY

The two most significant outcome measures of bariatric surgical success are weight loss and resolution of comorbidities. Major effects of bariatric operations have been nicely summarized in extensive meta-analyses[67,96-97] as well as in a major literature review.[98] Patients undergoing bariatric surgery with either SG or GBP have lost 52% to 68% of their excess weight.[99-100] This magnitude of weight loss is far greater than what is commonly achievable by nonsurgical methods, and it is considerably closer to patients' desired and expected weight loss. Long-term maintenance of weight loss of this magnitude can also be maintained for well over a decade.

Improvement in obesity-related and metabolic syndrome-related comorbidities have also been evaluated.[67,97-98,101] One clinical study demonstrated that at 1 year, type 2 diabetes remission rate with bariatric surgery was approximately 40% compared to 12% with medical treatment.[102] Another study showed remission rate at 2 years of approximately 85% compared to 0% with medical treatment.[103] This benefit seems to persist long-term as well, as at 6 years 62% of surgical patients experienced remission in their type 2 diabetes.[104] Resolution of diabetes following surgery is inversely related to the preoperative duration of the disease and occurs more in those patients having diabetes controlled using oral hypoglycemic agents. This clearly distinguishes surgical treatment of obesity as an important endocrinologic intervention, especially since the modest improvement in diabetes management that is produced by nonsurgical weight loss is accompanied by relapsing disease by nearly 100% of patients within 5 years.

Surgical effects on hypertension and hyperlipidemia are similarly impressive. A study of almost 1900 patients demonstrated that at a 6.5 year follow-up, 32% of surgically treated patients had remission of their hypertension versus 12.5% in medically managed patients.[105] A significant decrease in triglyceride levels, approximately 30% to 40% postbariatric surgery versus 8% from medical therapy; and increase in HDL cholesterol levels, 30% postsurgery versus 7% from medical therapy, have also been noted at 5 years.[106] Resolution of OSAHS seems to be independent of the specific bariatric surgical procedure employed, with resolution occurring overall in 85.7% of patients.[107] Postsurgical improvement in other comorbidities has also been demonstrated, with improvement in fatty infiltration of the liver, GERD symptoms, in respiratory function and asthmatic symptoms, reversal of the cardiomyopathy of obesity, and improvements in joint pain and mobility all having been reported.[71,108-112] The reduction in obesity-related comorbidities has been shown to endure with 5-year follow-up.[113] Although bariatric surgery has been shown to provide greater weight loss when compared with nonsurgical lifestyle interventions, comorbidities do resolve and risk factors do improve similarly with successful conservative treatments.[101]

Anesthetic Management of the Bariatric Surgical Patient

Patients undergoing bariatric surgery are receiving what is now the best long-term treatment for morbid obesity. However, their presurgical state is an agglomeration of abnormal physiologic conditions involving multiple organ systems. For that reason, there are considerable risks of mortality and morbidity associated with all types of bariatric surgery. Preoperative risk stratification can be used to identify patients at highest risk for significant morbid or fatal events when having bariatric surgery[114]; however detailed planning of the anesthesia care for the perioperative, intraoperative, and postoperative phases must be pursued for purposes of patient safety.

PREOPERATIVE EVALUATION

The preoperative assessment for anesthesia should include consideration of hypertension, diabetes, heart failure, and obesity hypoventilation syndrome. Results of the sleep study that the patient underwent are important. An AHI score greater than 30, implying severe sleep apnea, is a warning sign and a predictor for rapid and severe desaturation at induction. CPAP levels greater than 10 imply a patient with the potential for difficult mask ventilation.

Another useful piece of information in the preoperative evaluation is gleaned by examining the history of prior surgeries, their anesthetic challenges (i.e., ease or difficulty in securing the airway, intravenous access), need for intensive care unit admission, surgical outcomes, and the weight of the patient at that time. This information may either help ease some concerns or allow for better preparation for the upcoming anesthetic care. Recommended preoperative laboratory evaluations include fasting blood glucose, lipid profile, and serum chemistries (evaluating renal and hepatic function), complete blood count, ferritin, vitamin B_{12}, thyrotropin, and 25-hydroxyvitamin D.

If appropriately evaluated, ideally prepared, and optimally managed perioperatively, even patients with well-known

coronary artery disease (CAD) have comparable morbidity and mortality as those without CAD.[115] The effect of OSA on perioperative risk remains controversial,[116] but most patients are screened for OSA using overnight oximetry or polysomnography, or both, if appropriate. If identified with OSA and recommended for CPAP, then patients are encouraged to initiate therapy at home and it should be continued throughout the perioperative period.

Liver function abnormalities are common in this patient population, especially nonalcoholic fatty liver disease, with the extent of disease a determining factor for perioperative risk and postoperative outcomes. Cirrhotic liver disease with portal hypertension may be considered a contraindication to bariatric surgery.[116] Gastrointestinal symptoms of dyspepsia may indicate the presence of *Helicobacter pylori*, which requires treatment by standard medical regimen.

The factors that may be considered contraindications for bariatric surgery include unstable CAD, uncontrolled severe OSA, uncontrolled psychiatric disorder, intellectual disability (IQ <60), inability to understand the surgery, perceived inability to adhere to postoperative restrictions, continued drug abuse, and malignancy with a poor 5-year survival prognosis. In most situations preoperative management of the medical morbidities can help optimize risk and convert high-risk patients to ones of acceptable risk.

The anesthetic care of patients with OSA is complicated by the side effects of anesthetic drugs on an already compromised respiratory system. The commonly presented comorbidities in this patient population only accentuate the problem.[117] The change in perioperative risk based on the anesthetic agent utilized is only deduced from anecdotal reports of different cases. There is a paucity of trials to definitively determine an optimal anesthetic regimen. Nevertheless, the American Society of Anesthesiologists and the Society for Anesthesia and Sleep Medicine have both published statements based on existing literature and consensus of expert opinion addressing the perioperative identification and management of patients who carry a formal diagnosis of OSA as well as for those who are at risk for sleep apnea but remain undiagnosed. Additional questions commonly arise and cannot yet be answered on the basis of high-grade scientific evidence: is it safe to perform procedures in patients with sleep apnea and treat them as outpatients or day surgery patients? What procedures pose sufficient risk to the patient to recommend an overnight hospital stay? Does narcotic use impact this decision? Would the increased role of nonsteroidal antiinflammatory drugs (NSAIDs) change these recommendations? Factors such as neck size and open versus laparoscopic surgery are being studied as to their impact on outcomes. Further research on these and other patient safety related questions is continuing.

INTRAOPERATIVE CARE

Obese patients present special intraoperative challenges for the anesthesiologist in airway management, positioning, monitoring, choice of anesthetic technique and anesthetic drugs, pain control, and fluid management. Many of these issues are equally important in their postoperative care. The most significant and best studied are airway management, including endotracheal intubation, pulmonary physiology, and techniques of maintaining adequate blood oxygenation and lung volume. Specific interventions, techniques, and approaches employed by the anesthesia care team providing anesthesia for obese patients are important determinants of outcomes.

Patient Positioning

Even though there are no evidence-based studies that demonstrate that obese patients have more frequent complications from positioning, it seems fairly intuitive that morbidly obese patients do require extra care in positioning (see also Chapter 34). Even in the supine position, rhabdomyolysis from pressure on gluteal muscles leading to renal failure[118] and death[119] have been reported. For obese patients placed in the prone position, cushioning gel pads or other weight-bearing rolls may have excessive weight placed on them. Pressure points must be checked carefully, and even though pressure sites may be carefully padded, skin breakdown can still occur. This may cause tissue necrosis and infections, especially in lengthy surgery.[120] It is difficult to protect the dependent hip from pressure effects with patients placed in lateral decubitus position. Obese patients have excess axillary tissue that may make it impossible or unnecessary to place a traditional axillary roll in this position. In the lithotomy position the challenge may be in supporting the weight of the patient's legs using regular, and not oversized, stirrups. To minimize the risk of tissue compression injury or development of compartment syndrome, the duration during which legs are held in stirrups should be held as short as possible.[121]

Airway Management

No positioning issue is more important than that regarding positioning of the obese individual for laryngoscopy and endotracheal intubation. These are often considered to be much more difficult to perform in obese patients than patients having a normal BMI. Difficulty with laryngoscopy and intubation is usually thought to result from the obese patient having a short, thick neck, large tongue, and significant redundant pharyngeal soft tissue. However, the correlation between morbid obesity and difficult laryngoscopy and intubation is not universally observed in clinical practice. This is likely to result from a simple, but important approach to clinical care, with careful attention being paid to patient positioning prior to induction of general anesthesia. Appropriate positioning plays an important role in providing optimal conditions for successful placement of the endotracheal tube under direct vision.

A number of studies have been conducted to determine the incidence of difficult laryngoscopy or intubation in the obese population, with mixed findings. One study found an association between oropharyngeal Mallampati classification and BMI as predictors of difficult laryngoscopy.[122] During laryngoscopy, the patients' heads were placed in optimum sniffing position, regardless of BMI. In a study conducted exclusively with obese patients, BMI was not found to be associated with intubation difficulties.[41] A high Mallampati score was identified as a predictor of "potential intubation problems," but intubation by direct laryngoscopy was successful in 99 of 100 patients studied. All patients were positioned with pillows or towels under their shoulders, with the head elevated and neck extended.

Another group studied both lean and obese patients and found a Mallampati score of III or IV to be the only independent risk factor for difficult intubation in the obese study group.[123] The authors demonstrated that the Mallampati score had low specificity and low positive predictive values (62% and 29%, respectively) for difficult intubation. They concluded that intubation was more difficult in the obese patients. During intubation, patients were placed in a semirecumbent position (30-degree elevation) with the head in the sniffing position. In another study ultrasound was used to quantify the amount of soft tissue between the skin and the anterior aspect of the trachea at the level of vocal cords.[124] The authors also assessed the airway by measurement of thyromental distance, mouth opening, degree of neck mobility, Mallampati score, neck circumference, and presence of OSA. Only an abundance of pretracheal soft tissue measured ultrasonically and neck circumferences were found to be positive predictors of difficult intubation with laryngoscopy performed with patients in the sniffing position. A meta-analysis of 35 studies was conducted to determine the diagnostic accuracy of preinduction tests for predicting difficult intubation in patients having no airway pathology.[125] The incidence of difficult intubation in obese patients was three times the incidence compared to the nonobese population. This may have resulted from suboptimal patient positioning, which was not clearly described in any of the preceding studies, including ramped positioning or elevating the upper body and head of morbidly obese patients to align the ear with the sternum horizontally, as has been shown to improve laryngoscopic view.[126] In this study, morbidly obese patients were assigned either to be in sniffing position or ramped position for airway management. The study demonstrated a statistically significant difference in laryngeal view, with ramped position providing the superior view. In one study, morbidly obese patients in a ramped position with a Mallampati score of III or IV and male gender were shown to predict possible difficult intubation, while no relationship was noted between difficult direct laryngoscopy and the presence of OSA, patient neck circumference, or BMI.[127]

Based on the evidence from randomized controlled trials and other literature on airway management of obese patients, patients should be readily intubated by direct laryngoscopy if placed carefully in ramped position. This can be achieved using commercially available positioning devices[128] or by building a ramp from blankets or sheets to achieve the desired placement of the patient's head relative to the thorax.[129] Obese patients must be examined for the common objective signs of potential difficult intubation, which include small mouth opening, large protuberant teeth, limited neck mobility, and retrognathia. Alternative airway management techniques include the use of a video laryngoscope for intubating obese patients.[130] Compared to direct laryngoscopy, the use of video laryngoscopy in patients undergoing bariatric surgery has shown to provide a better glottic view and decreased time required to successfully intubate the trachea.[131] Performance of an awake, topicalized direct laryngoscopy with modest sedation can also be utilized to assess laryngoscopic view in deciding whether to proceed with induction of general anesthesia or awake, sedated fiberoptic intubation. Of course, the equipment for emergency airway management including laryngeal masks and a fiberoptic bronchoscope should be immediately available.

Another area that requires specific attention during the perioperative period is the obese patient's pulmonary physiology. It is especially important to appreciate techniques to maintain oxygenation and lung volume in caring for the obese patient. First, obese patients have multiple pulmonary abnormalities, including decreased vital capacity, inspiratory capacity, expiratory reserve volume, and functional residual capacity. Second, closing capacity in obese individuals is close to or may fall within tidal breathing, particularly in the supine or recumbent position. Moreover, both lung and respiratory system compliance are low with obesity because patients breathe at lung volumes that are abnormally low.[132] As a result of the underlying physiology, the obese patient is likely to undergo rapid oxygen desaturation, particularly during periods of apnea such as occurs during induction of general anesthesia. However, the presence of OSA by itself does not independently increase the risk of the patient desaturating during induction of general anesthesia as long as appropriate precautions are instituted.[133] However, after induction and intubation have occurred, patients may continue to derecruit gas exchange units throughout the anesthetic course.[134] A variety of maneuvers have been studied to preserve oxygenation and maintain lung volume specifically in the obese population.

In one study of the rate of development of hypoxemia in patients during apnea, patients received 100% oxygen by facemask to achieve denitrogenation before induction of general anesthesia.[134] The apneic period was continued after induction until the SpO_2 fell to 90%. Obese patients reached the end point in less than 3 minutes, whereas it took 6 minutes in patients having a normal BMI. Efforts to prevent atelectasis formation and desaturation during induction of general anesthesia in obese individuals include application of CPAP during preoxygenation,[135-137] and mask application of positive end-expiratory pressure (PEEP), and mechanical ventilation following induction.[137] Use of 10 cm H_2O CPAP during preoxygenation in the supine position resulted in a higher partial pressure of oxygen (PaO_2) after intubation and decreased the amount of atelectasis that developed.[81] The combination of CPAP during preoxygenation and PEEP/mechanical ventilation after induction significantly increased the nonhypoxemic apnea duration from 2 minutes, found in controls not receiving CPAP or PEEP, to 3 minutes. Use of 7.5 cm H_2O CPAP during 3 minutes of preoxygenation while supine, however, did not change the time required for obese patients to desaturate to an SpO_2 of 90%.[136] Preoxygenation using 25-degree head-up (back inclined) as opposed to supine positioning without positive airway pressure did increase the elapsed time needed for anesthetized, apneic, obese individuals to desaturate to an SpO_2 of 92%.[138] The patients in head-up position had a significantly higher PaO_2 after preoxygenation, just prior to induction. The obesity-associated gas exchange defect depended on the waist-to-hip ratio, an index of the distribution of adipose tissue surrounding the thorax.[139] This study further demonstrated that morbidly obese men are more likely to have poorer pulmonary gas exchange than morbidly obese women. In a study conducted to assess the relationship between patient positioning and development of hypoxemia in obese patients during apnea after anesthetic

induction and intubation, patients were ventilated with a 50% oxygen/50% air mixture for 5 minutes before the ventilator circuit was disconnected.[140] Apnea was continued until the SpO_2 fell to 92%, at which time ventilation was resumed. Patients in the supine position reached the end point in 2 minutes, but it took 30 seconds longer if supine position with the back elevated 30-degrees was used and 1 minute longer if 30-degree reverse Trendelenburg position was used. Use of 30-degree reverse Trendelenburg position in obese patients undergoing bariatric surgery was also shown to reduce the alveolar-to-arterial oxygen difference, as well as increase total ventilatory compliance and reduce peak and plateau airway pressures when compared to supine position.[141] Vital capacity has also been shown to decrease to a greater extent under general anesthesia in obese patients compared to normal weight patients.[142]

A variety of intraoperative maneuvers to maintain lung volume and oxygenation have also been studied. Increasing tidal volume incrementally from 13 to 22 mL/kg in obese patients ventilated under general anesthesia did not improve the gas exchange defect but did increase airway pressures.[143] Use of 10 cm H_2O PEEP has been demonstrated to have a greater effect in obese patients compared to normal subjects on improving ventilatory mechanics, increasing PaO_2, and decreasing alveolar-to-arterial oxygen difference during general anesthesia with neuromuscular blockade.[144] In addition to PEEP alone, use of a recruitment maneuver such as sustained lung inflation to 55 cm H_2O for 10 seconds followed by application of PEEP has been demonstrated to prevent atelectasis from developing and to improve oxygenation, whereas neither PEEP nor a recruitment maneuver alone achieved the same degree of maintenance of pulmonary function.[145]

Pneumoperitoneum during laparoscopic procedures increases pulmonary resistance and decreases dynamic lung compliance.[146] During pneumoperitoneum, alterations in body position, tidal volume, and respiratory rate had no effect on the alveolar-to-arterial oxygen difference in obese patients.[147] During pneumoperitoneum for laparoscopic bariatric surgery, alveolar recruitment by repeated sustained lung inflation to 50 cm H_2O followed by mechanical ventilation with 12 cm H_2O PEEP has been shown to increase PaO_2 intraoperatively, however at the expense of causing hypotension that required vasopressor use.[148] In an attempt to optimize PEEP in obese patients undergoing laparoscopic surgery, a normal functional residual capacity was maintained with 15 ± 1 cm H_2O PEEP. Infusion of intravascular volume expanders was required to prevent PEEP-induced hemodynamic compromise.[149]

In summary, a back-up position with the use of PEEP during preoxygenation at induction of anesthesia and intraoperatively has been shown to decrease the A-a gradient in an obese patient.[150] Furthermore, the application of noninvasive modes of ventilation including pressure support and bilevel delivered by mask for preoxygenation, induction, and maintenance of anesthesia to maintain oxygenation and ventilatory mechanics in obese patients has been shown to be beneficial. Ideal patient positioning, use of PEEP, and special modes of ventilation just prior to emergence and extubation may help maintain pulmonary function and gas exchange postextubation. Currently there are no published guidelines to address the issues of maintenance of oxygenation and ventilatory mechanics in obese patients undergoing general anesthesia. Considering both the airway management issues detailed previously as well as the oxygenation, lung volume, and ventilatory mechanics issues described above for obese individuals, anesthesia care providers should position patients to achieve the combined goals of providing a superior laryngoscopic view for ease of endotracheal intubation while establishing optimal conditions for oxygenation and preservation of pulmonary mechanical function.

It is the practice at our institution that obese patients are initially placed in a ramped position and then into reverse Trendelenburg, if needed, to achieve a 25- to 30-degree incline of the thorax prior to preoxygenation. Patients are then preoxygenated for 3 to 5 minutes using 100% oxygen delivered under positive pressure. For those patients receiving CPAP at home for OSA, we use CPAP or pressure support ventilation by facemask at a pressure level identical to the patient's home CPAP setting. Otherwise, CPAP of 8 to 10 cm H_2O is appropriate. Following induction, it is reasonable to maintain 8 to 10 cm H_2O PEEP intraoperatively, but care must be taken to treat any hypotension that may occur. Finally, if the patient's position must be changed intraoperatively, one must return the patient to head-up position prior to emergence and extubation.

In preparation for emergence from anesthesia, neuromuscular blockade must be fully reversed before the patient is extubated. Given the advent of a pressure support ventilation mode on many newer models of anesthesia machines, the bariatric patient can be maintained on pressure support during emergence once spontaneous ventilation has resumed. When adequate muscle strength has returned, as demonstrated by sustained tetanus using the nerve stimulator and performance of a 5-second head lift, the awake patient who is following commands can be extubated. Pressure support or CPAP can be delivered immediately by mask applied to the face as is done during preoxygenation prior to induction of anesthesia. The use of PEEP and noninvasive modes of ventilation just after emergence and extubation has been shown to improve pulmonary function in patients up to 24 hours later.[151] We utilize CPAP during recovery, especially in those patients already prescribed CPAP for their OSA. The basic premise that must be respected with regard to airway management and its integral relationship to pulmonary function is that morbid obesity incurs significant derangements of lung function and pulmonary mechanics. These factors must be managed carefully in order to minimize intraoperative and postoperative pulmonary complications.[152,153]

Anesthetic Drugs and Dosing

Anesthetic drugs such as opioids, propofol, and benzodiazepines are all well known to have exaggerated responses in patients with OSA. They may decrease pharyngeal musculature tone, which is essential in maintaining airway patency.[40,154] In the setting of OSA, volatile agents are known to diminish ventilatory response to carbon dioxide, especially in children with tonsillar hypertrophy. Another pediatric study showed that spontaneously breathing intubated children with a history of OSA have depressed ventilation and half have apnea with 0.5 μg/kg of intravenous fentanyl. Although these data are from the pediatric

literature, it would be prudent to apply the same principles in obese adult patients until proven otherwise. It therefore becomes attractive to use short-acting drugs and nondepressors of ventilation like the α_2-agonist dexmedetomidine. This should, at least in theory, speed up the return to baseline respiratory function.[120]

Commonly used anesthetic drugs can be dosed on TBW or IBW based on lipid solubility. In the past, IBW was interpreted to mean fat-free weight, implying that it could be used as a substitute for lean body weight or more correctly, lean body mass (LBM), usually approximated as 120% of IBW. LBM is a good weight approximation to use when dosing hydrophilic medications. As expected, volume of distribution (V_D) is changed in obese patients with regard to lipophilic drugs. This is especially true of benzodiazepines and barbiturates, among the commonly used anesthetic drugs. Two exceptions to this rule are procainamide[155] and remifentanil,[156] which even though highly lipophilic, have no relationship between properties of the drug and their V_D.[157] Consequently, commonly used anesthetic drug dosing is based on IBW for propofol, vecuronium, rocuronium, sugammadex, and remifentanil. In contrast, midazolam, succinylcholine, cisatracurium, fentanyl, and sufentanil should be dosed on the basis of TBW. Another caveat to this recommendation is that maintenance doses of propofol should be based on TBW and conversely for sufentanil on IBW.[157] This implies that one can use, based on patient weight, larger amounts of benzodiazepines, fentanyl, or sufentanil, although these are best titrated to desired clinical effect. Conversely, based on real body weight, smaller amounts of propofol are needed to anesthetize the patient.

With vecuronium or rocuronium, the initial dose should be based on IBW with additional doses based on the closely followed state of neuromuscular blockade. Complete blockade in the morbidly obese patient is necessary not just for the surgeon's convenience, but also to facilitate mechanical ventilation. The drug chosen is not as important as the depth of the state of paralysis. The pharmacokinetic profile of sugammadex is similar to that of rocuronium, and its dosing should be based on IBW.[158]

Volatile anesthetics are chosen based on physical characteristics of tissue solubility which are expressed as blood-gas partition coefficients and fat-blood partition coefficients. There is some evidence to suggest that desflurane may be the anesthetic of choice based on consistent and rapid recovery profile versus sevoflurane and propofol.[159,160] Some studies suggest that the differences in immediate recovery between sevoflurane and desflurane are not clinically significant.[161]

Even though nitrous oxide provides some analgesic effect and is rapidly eliminated, we prefer to avoid it based on the high oxygen demands in the obese. Its entry into air spaces in short intraabdominal surgeries may not be a significant factor, but in bariatric surgery, especially when done laparoscopically or robotically, any increase in bowel gas volume could make a challenging surgical procedure even more difficult for the surgeon.

Induction of Anesthesia

There has been considerable debate regarding obesity, the risk of aspiration of gastric contents, and the need to provide aspiration prophylaxis.[162] Abdominal bloating or fullness and female sex are associated with delayed gastric emptying of solids and liquids in diabetic patients.[163] Although many obese patients have type 2 diabetes, separate studies of gastroesophageal reflux during anesthesia have not demonstrated body habitus to be a predictor of reflux rates.[164] Gastric fluid volume and pH have been shown to be the same in obese patients who are fasting or who have oral intake of up to 300 mL of clear liquid 2 hours prior to being anesthetized.[165] Obesity itself does not increase aspiration risk. However, acid aspiration prophylaxis including H_2–receptor agonists or proton pump inhibitors must be considered in patients having identifiable aspiration risks. Rapid sequence induction or awake fiberoptic intubation may also be considered in such patients.[40]

Based on experience from obstetric practice, there is a body of evidence to show that regional anesthesia, especially epidural and spinal, are safe and feasible in patients with large body habitus.[120] However, regional anesthesia is technically more difficult due to the physical challenge of placing the catheters and the tendency of these catheters to migrate out of the epidural space. Special equipment, including longer needles or special ultrasound probes, may be needed for the correct placement of catheters in these patients. Care should be exercised in dosing these catheters because of the increased cephalad spread of the drug and the block due to the smaller epidural space compared to normal-weight patients.[166,167] The degree of respiratory compromise suffered by obese patients will also be of a larger magnitude than normal-weight patients with a high regional block.

There is little evidence to suggest that epidural pain management improves overall outcomes. Since the trend of laparoscopic surgery is increasing compared to open laparotomies, this becomes less of an issue. In the morbidly obese patient who is having an open laparotomy, use of a thoracic epidural catheter to control pain has the most important benefit of reducing the attenuation of vital capacity postoperatively.[168]

Obesity per se does not require invasive monitoring, so GBP surgery can be performed safely with routine monitoring. The indications for invasive monitoring stem from the comorbidities present in these patients. Since these tend to run together in the patients needing surgery, the incidence of invasive monitoring in these patients thereby increases.[169] Morbidly obese patients having serious comorbidities such as obesity-hypoventilation syndrome, who present with pulmonary hypertension and cor pulmonale, may require a pulmonary artery catheter or intraoperative evaluation utilizing transesophageal echocardiography. The rationale for central venous access may stem from difficulties in peripheral access rather than any other indication. Many patients receive an inferior vena cava filter prophylactically due to the high risk of deep vein thrombosis and pulmonary embolism associated with obesity and bariatric surgery.[170] It is recommended that a central line inserted at the time of surgery should be placed under ultrasound guidance to decrease complications as well as to increase ease of placement. Similarly, difficulty in noninvasive blood pressure measurements, secondary to body habitus–related difficulty in appropriate cuff placement, may be an indication for arterial catheter placement. Obtaining arterial blood gases may help to guide intraoperative ventilation and extubation.

Postoperative pain management in weight loss surgery patients can involve intravenous analgesia employing patient-controlled analgesia (PCA) or thoracic epidural analgesia. There is no clear data proving the superiority of one technique over the other; therefore in many instances, surgical technique, open versus laparoscopic, may help guide that decision. In our practice, we tend to reserve epidural anesthesia for patients undergoing open GBP. Even with these drawbacks, we have approximately an 80% success rate with superobese patients. It may be prudent to test the effectiveness of the epidural prior to induction of anesthesia. An opioid-based PCA with local anesthetic wound infiltration and adjunct non-narcotics is a reasonable alternative approach for most patients. Injection of local anesthetic in the incision site prior to making the incision may result in preemptive analgesia. Adjunct analgesia with non-narcotic medications such as oral and intravenous acetaminophen, NSAIDs, low dose ketamine, and dexmedetomidine, will decrease opioid requirements and thereby opioid-induced side effects as well.

Intraoperatively, utilizing the appropriate operating room table for the patient's weight is critical since the consequences of having a table not rated to the patient's weight can have serious consequences for both patient and the operating room personnel. It may be useful to keep the patient strapped throughout the period of sedation and sleep. Along with a safety strap, it might also be useful to apply a bean bag under the patient to keep the patient from sliding off the operating room table. Thermal management in the operating room is best accomplished by forced-air warmers. Arm boards may need extra padding to keep the patient from having the arm and shoulder out of an anatomical position. If the arms are to be tucked by the side of the patient, then wide, well padded sleds may be useful.

Fluid requirements for the obese patient are larger than predicted for those of normal BMI and even during a relatively short, 2- to 3-hour case these patients may need 3 to 4 L of crystalloid fluid to prevent acute tubular necrosis (ATN) in the kidneys. Hypovolemia, which can cause a protracted prerenal state and contribute to the development of ATN, can be prevented by appropriate hydration. Retrospective data from the University of Pittsburgh Medical Center suggests that primary acute renal failure after weight loss surgery occurs in approximately 2% of patients. Other predisposing factors include BMI greater than 50 kg/m^2, prolonged surgical time, prior history of renal disease, and intraoperative hypotension.[171]

POSTOPERATIVE MANAGEMENT

In our practice, we prefer to have the postbariatric surgery patients stay at the same location consistently. This allows skilled nursing and ancillary care to be provided to patients on a consistent basis. These patients are kept on their CPAP or bilevel positive airway pressure machines as much as possible with monitoring of end-tidal carbon dioxide and SpO_2 by pulse oximetry recommended. Monitoring capnometry in the postoperative period is critical for assessing a morbidly obese patient's respiratory function since the administration of supplemental oxygen may delay the diagnosis of hypoventilation. At the Hospital of the University of Pennsylvania, patients identified as having difficult airways are distinguished with armbands, and with visible signs on their beds, their hospital charts, and on the electronic medical records for the remainder of their hospital stay. Additionally, a note by the attending anesthesiologist explaining the difficulty in intubation as well as the means utilized to secure the airway in the operating room is available in the room. In case of an unexpected emergency intubation, for whatever reason, we feel that this extra information is extremely useful to the resuscitation team.

Postoperative nausea and vomiting (PONV) is a common reason for delayed discharge from the postanesthesia care unit (PACU). Intraabdominal surgery is a known risk factor for PONV.[172] Patients undergoing bariatric surgery have significant manipulation of their stomach, which may increase their risk of PONV. It is recommended to include a multimodal approach to PONV prophylaxis, including ondansetron, dexamethasone, and placement of a scopolamine patch when not contraindicated in order to minimize patient dissatisfaction and serious complications such as aspiration or wound and anastomotic dehiscence.

Enhanced recovery after surgery (ERAS) protocols are being used widely in surgical patients to decrease morbidity and decrease in-hospital stay. One study looking at patients undergoing an SG demonstrated a median hospital stay of 1 day in the ERAS group compared to 2 days in the control group, without any difference in postoperative complications or readmissions.[173] The ERAS protocol included standardized preoperative patient education, shortened preoperative fasting, multimodal analgesia and antiemetic therapy, avoidance of fluid overload, early ambulation and feeding, and incentive spirometry.

MANAGEMENT OF COMPLICATIONS

Bariatric surgery is considered to be very safe, but not without potential complications, which are increasingly predictable.[174] In-hospital mortality rates for patients undergoing laparoscopic and open bariatric surgery are 0.17% and 0.79%, respectively.[175] Mortality is associated with a need for reoperation during the same hospitalization,[176] with intestinal leakage being a serious complication accounting for a large number of patient deaths.[177] Risk factors also include older age, excessive obesity, poor functional status, and comorbid conditions of congestive heart failure or renal failure.

Morbidity occurring during the immediate postoperative in-hospital period typically falls into one of four categories; wound, gastrointestinal, pulmonary, and cardiovascular complications. The complication rates are significantly lower in each category for patients undergoing laparoscopic rather than open procedures. The American College of Surgeons National Surgical Quality Improvement Program database showed that patients who had an open GBP compared with a laparoscopic GBP had a morbidity rate of 7.4% versus 3.4%, return visits to the operating room rate of 4.9% versus 3.6%, and median postoperative length of stay of 3 versus 2 days, respectively. Box 58.3 provides a categorical listing of complications of bariatric surgery.

The most common complications requiring reoperation include postoperative intraabdominal bleeding, anastomotic leakage, suture line dehiscence, small bowel obstruction, and deep wound infection,[177-182] all of which may

BOX 58.3 Complications of Bariatric Surgery

Early	Bleeding
	Infection
	Dehydration
	Peritonitis
	Leak from anastomotic site
	Bowel obstruction
	Perforation
	Pneumonia
	DVT/PE
	Death
Late	Anorexia
	Cholelithiasis/cholecystitis
	Pouch dilation or stricture
	Gastroesophageal reflux disease/dysphagia
	Herniation at surgical site
	Small bowel obstruction
	Marginal ulcers
	Pancreatitis
	Nutritional issues
	Fat-soluble vitamin deficiencies, especially vitamin B_{12}

require a general anesthetic for laparotomy. Despite deep vein thrombosis prophylaxis therapy in the perioperative period, patients can also present postoperatively with deep vein thrombosis or pulmonary embolism and require an anesthetic for placement of an inferior vena cava filter device. As mentioned earlier, prophylactic filters are often placed prior to bariatric surgery.[170] The risk of venous thromboembolism is higher in open bariatric procedures, men, and patients with preexisting lower extremity edema and pulmonary hypertension.[183]

In all cases requiring reoperation shortly after the original bariatric procedure it is prudent to review the anesthesia record. Specific attention should be paid to the documentation of patient position and technique employed for airway management in the prior anesthetic. Patients may be hypovolemic from blood loss, inadequate hydration, vasodilatation, and insensible fluid losses associated with fever and infection. It is especially important to consider additional or new risks of aspiration of gastric contents. This may be due to the presence of a postoperative ileus, small bowel obstruction, and surgical creation of a Roux-en-Y GBP limb that excludes the pylorus as an element of protection from reflux of intestinal contents. Decompression of the gastric pouch in patients going for surgery to relieve a small bowel obstruction can be achieved with careful introduction of a nasogastric or orogastric tube just prior to induction of general anesthesia. Although this may increase the risk of violating a fresh, competent anastomotic suture line, communication between anesthesiologist and surgeon can be pursued to determine the risks and benefits of performing this maneuver. During the ensuing laparotomy, any perforation of a fresh suture line resulting from the attempt to decompress the gastrointestinal tract can be repaired immediately and the nasogastric or orogastric tube can subsequently be left in place for continued postoperative drainage.

Depending on the extent of reoperation, requirement for volume resuscitation, blood transfusion, degree of peritonitis with anastomotic leak, presence of sepsis, or other significant continued risks to health, patients undergoing reoperation may require prolonged postoperative ventilation. Requirements for postoperative pain management may also be considerably different than those associated with the initial bariatric procedure. In patients who are sufficiently hemodynamically stable immediately prior to reoperation, an epidural catheter can be placed prior to induction for pain management as part of the postoperative care. This is especially valuable in obese patients undergoing laparotomy, as is described elsewhere in this chapter.

There are also a number of potential major complications requiring surgical intervention weeks, months, or even years after a bariatric surgical operation has been performed. Patients may develop anastomotic strictures or ulcers, ventral hernias, gastrogastric fistulae, and severe reflux disorders requiring additional surgery.[92] Small bowel obstruction may appear weeks after surgery.[181] Patients may desire cosmetic operations to remove excess skin or liposuction procedures to sculpt dysmorphic body areas following significant weight loss. Patients may also require adjustment of a gastric band or pursue band removal. The anesthetic considerations for such patients should include a review of the prior anesthetic record to glean information regarding airway and pain management. The degree of weight loss achieved and the resolution of comorbidities including diabetes, hypertension, and OSA may significantly alter the approach used in the bariatric operation.

A fraction of patients develop significant neurologic complications following GBP surgery.[184-186] These are known to include polyneuropathy, polyradiculoneuropathy, myelopathy, encephalopathy, and optic neuropathy. Myelopathy occurs most frequently but does not present until about 10 years after surgery.[184] These patients have demonstrable nutritional deficiencies, but except for the vitamin B_{12} and copper deficiencies found in patients with myelopathy, no specific nutritional profile has been found to correlate with neurological complications. Although neurologic symptoms accompanying weight loss surgery are not likely to result in additional surgery, they represent new or additional comorbidities that should be fully considered by the anesthesiologist caring for patients having previously undergone bariatric surgery.

The nutritional and metabolic complications of bariatric surgery also include protein and protein-calorie malnutrition. Patients may have excessive weight loss occurring either too rapidly or beyond the predetermined goals, steatorrhea or severe diarrhea, hypoalbuminemia, marasmus, edema, and hyperphagia.[92,187-189] In cases of severe malnutrition, patients may require enteral or parenteral nutrition therapy. Surgical revision may be required to correct excessive weight loss and hypoalbuminemia. Under such circumstances, an anesthetic regimen accounting for decreased drug binding effects of a low serum albumin must be considered.

Considerations for Management of the Obese Patient Presenting for Nonbariatric Surgery

Few studies have evaluated the importance of morbidity when performing common nonbariatric surgical

procedures on the obese patient. A study by Dindo and coworkers[190] of 6336 patients did not find any difference in the incidence or severity of complications after elective general surgery, except surgical site infections. Other studies have assessed the effect of obesity on wound infections and have shown that they are increased.[191-192] Multiple studies have demonstrated that obese patients are at higher risk after gynecological, orthopedic, cardiovascular, urological, and transplantation surgery, while other studies have not shown there to be obesity-related risk differences.[193-194]

From the data of Dindo and associates[190] regarding complications and the type of surgical procedure, operative severity and open surgery were independent risk factors for postoperative complications. They found no differences in the type of postoperative complications in obese and nonobese groups. This study provides data to decrease the prejudice that patients with obesity are implied to have a higher incidence of postoperative complications. This preconception may pertain more to the perception of the medical team as a result of technical difficulties on the anesthetic and surgical end. Surgeries may last longer, about 25% more time for laparoscopic than for open cholecystectomy in the morbidly obese. Significantly, the wound infection rate was much better after laparoscopic than after open surgery, which supports the practice of performing laparoscopic surgery in obese patients rather than the alternative.

Complete references available online at expertconsult.com.

References

1. Kaidar-Person O, et al. *Obes Surg*. 2011;21(11):1792–1797.
2. Haslam D. *Obes Rev*. 2007;8(suppl 1):31–36.
3. Baskin ML, et al. *Obes Rev*. 2005;6(1):5–7.
4. Hensrud DD, Klein S. *Mayo Clin Proc*. 2006;81(suppl 10):S5–10.
5. Canoy D, Buchan I. *Obes Rev*. 2007;8(suppl 1):1–11.
6. Obesity and Overweight Fact Sheet. www.who.int/mediacentre/factsheets/fs311/en.
7. Fryar CD, Carroll MD, Ogden CL. *Prevalence of overweight, obesity, and extreme obesity among adults aged 20 and over: United States, 1960–1962 Through 2011–2014*. National Center for Health Statistics Data; 2016.
8. Ogden CL, et al. *JAMA*. 1988-1994 Through 2013-2014. 2016; 315(21):2292–2299.
9. Conway B, et al. *Obes Rev*. 2004;5(3):145–151.
10. Zabena C, et al. *Obes Surg*. 2009;19(1):87–95.
11. Katzmarzyk PT, et al. *Obes Rev*. 2003;4(4):257–290.
12. Kopelman P. *Obes Rev*. 2007;8(suppl 1):13–17.
13. Nguyen NT, et al. *Obes Surg*. 2011;21(5):351–355.
14. Zammit C, et al. *Int. J. Gen. Med*. 2010;3:335–343.
15. Apovian CM, et al. *Circulation*. 2012;125(9):1178–1182.
16. Wearing SC, et al. *Obes Rev*. 2006;7(3):239–250.
17. Scopinaro N, et al. *Obes Surg*. 2007;17(2):185–192.
18. Guidone C, et al. *Diabetes*. 2006;55(7):2025–2031.
19. Pinkney J, et al. *Obes Rev*. 2004;5(1):69–78.
20. Alam I, et al. *Obes Rev*. 2007;8(2):119–127.
21. Machado M, et al. *Curr Opin Clin Nutr Metab Care*. 2006;9(5):637–642.
22. Valera-Mora ME, et al. *Am J Clin Nutr*. 2005;81(6):1292–1297.
23. Poirier P, et al. *Circulation*. 2011;123:1683–1701.
24. Liberopoulos EN, et al. *Obes Rev*. 2005;6(4):283–296.
25. Levin PD, et al. *Med Clin North Am*. 2009;93(5):1049–1063.
26. Ervin RB, et al. *Natl Vital Stat Rep*. 2009;(13):1–7.
27. O'Neill S, et al. *Obes Rev*. 2015;16(1):1–12.
28. Buchwald H, et al. *JAMA*. 2004;292(14):1724–1737.
29. Frezza EE, et al. *Obes Surg*. 2011;21(3):379–385.
30. Buchwald H, et al. *Obes Surg*. 2009;19(12):1605–1611.
31. Moulin CM, et al. *Obes Surg*. 2011;21(1):112–118.
32. Selmi C, et al. *Exp Biol Med*. 2007;232(11):1409–1413.
33. Patil SP, et al. *Chest*. 2007;132(1):325–337.
34. O'Keefe T, et al. *Obes Surg*. 2004;14:23–26.
35. Chung F, et al. *Br J Anaesth*. 2012;108(5):768–775.
36. Tung A, et al. *Curr Opin Anaesthesiol*. 2001;14:671–678.
37. Biro P, et al. *J Clin Anesth*. 1995;7(5):417–421.
38. Pierce RJ, et al. *Clin Exp Pharmacol P*. 1999;26(1):1–10.
39. Mayer P, et al. *Eur Respir J*. 1996;9(9):1801–1809.
40. Benumof JL, et al. *Anesthesiol Clin*. 2002;20(4):789–811.
41. Brodsky JB, et al. *Anesth Analg*. 2002;94(3):732–736.
42. Gomez-Illan F, et al. *Obes Surg*. 2012;22:950–955.
43. Roth CL, et al. *Obes Surg*. 2009;(1):29–35.
44. National Cholesterol Education Program (NCEP). *Circulation*. 2002;106(25):3143–3421.
45. Kushner RF, et al. *Prog Cardiovasc Dis*. 2014;56(4):465–472.
46. Grundy SM, et al. *Circulation*. 2004;109(3):433–438.
47. Low AK, et al. *Am J Med Sci*. 2006;331(4):175–182.
48. Whelton PK, et al. *Hypertension (Dallas, Tex. : 1979)*. 2017.
49. Martinez EA, et al. *Anesth Analg*. 2007;104(1):4–6.
50. Bray GA, et al. *Best Pract Res Clin Gastroenterol*. 2014;28(4):665–684.
51. Allison DB, et al. *Crit Rev Food Sci Nutr*. 2001;41(1):1–28.
52. Boozer CN, et al. *Int J Obes Relat Metab Disord*. 2001;25(3):316–324.
53. Boozer CN, et al. *Int J Obes Relat Metab Disord*. 2002;26(5):593–604.
54. Astrup A, et al. *Int J Obes Relat Metab Disord*. 1992;16(4):269–277.
55. Haller CA, et al. *N Engl J Med*. 2000;343(25):1833–1838.
56. Cigaina V. *Obes Surg*. 2004;14(suppl 1):S14–S22.
57. Favretti F, et al. *Obes Surg*. 2004;14(5):666–670.
58. Cha R, et al. *World J Gastrointest Endosc*. 2014;6(9):419–431.
59. Dietz WH, et al. *N Engl J Med*. 2005;352(20):2100–2109.
60. Fryar CD, Carroll MD, Ogden CL. *Prevalence of Overweight and Obesity Among Children and Adolescents Aged 2–19 Years: United States, 1963–1965 Through 2013–2014. Surveys, D. o. H. a. N. E., Ed*; 2016.
61. de Onis M, et al. *Am J Clin Nutr*. 2000;72(4):1032–1039.
62. Kirk S, et al. *J Am Diet Assoc*. 2005;105(suppl 5):44–51.
63. Boland CL, et al. *Ann Pharmacother*. 2015;49(2):220–232.
64. Tsai WS, et al. *Arch Pediatr Adolesc Med*. 2007;161(3):217–221.
65. Christou NV, et al. *Ann of Surg*. 2004;240(3):416–423.
66. Pereira JA, et al. *Obes Res*. 2003;11(12):1495–1501.
67. Buchwald H, et al. *J Am Coll Surg*. 2005;200(4):593–604.
68. Shaffer EA, et al. *J Clin Gastroenterol*. 2006;40(3 suppl 1):S44–S50.
69. Encinosa WE, et al. *Med Care*. 2006;44(8):706–712.
70. Flum DR, et al. *J Am Coll Surg*. 2004;199(4):543–551.
71. Lynch RJ, et al. *J Clin Gastroenterol*. 2006;40(8):659–668.
72. Akkary E, et al. *Obes Surg*. 2012;22(5):827–831.
73. Buchwald H, et al. *Obes Surg*. 2013;23(4):427–436.
74. Salameh JR, et al. *Am J Med Sci*. 2006;331(4):194–200.
75. Poulose BK, et al. *J Am Coll Surg*. 2005;201(1):77–84.
76. Santry HP, et al. *Ann of Surg*. 2007;245(1):59–67.
77. Weber M, et al. *Ann of Surg*. 2004;240(6):975–982.
78. Deitel M, et al. *J Am Coll Nutr*. 2002;21(5):365–371.
79. Galvani C, et al. *Surg Endosc*. 2006;20(6):934–941.
80. Kendrick ML, et al. *Mayo Clin Proc*. 2006;81(suppl 10):S18–S24.
81. Deitel M. *Obes Rev*. 2007;17(6):707–710.
82. Eisenberg D, et al. *World J Gastroenterol*. 2006;12(20):3196–3203.
83. Montgomery K, et al. *Obes Surg*. 2007;17:711–716.
84. Weller WE, et al. *Ann Surg*. 2008;248(1):10–15.
85. Nguyen NT, et al. *J Am Coll Surg*. 2011;213(2):261–266.
86. Estimate of Bariatric Surgery Numbers, 2011-2016. https://asmbs.org/resources/estimate-of-bariatric-surgery-numbers.
87. van Rutte PW, et al. *Br J Surg*. 2014;101(6):661–668.
88. Ramon JM, et al. *J Gastrointest Surg*. 2012;16(6):1116–1122.
89. Schauer PR, et al. *Ann of Surg*. 2000;232(4):515–529.
90. Gentileschi P, et al. *Surg Endosc*. 2002;16(5):736–744.
91. Huang CK, et al. *Obes Surg*. 2011;21(3):391–396.
92. Malinowski SS. *Am J Med Sci*. 2006;331(4):219–225.
93. Mathus-Vliegen EM, et al. *Gastrointest endosc*. 2005;61(1):19–27.
94. Gaur S, et al. *Gastrointest endosc*. 2015;81(6):1330–1336.
95. Thompson CC, et al. *Am J Gastroenterol*. 2017;112(3):447–457.
96. Garb J, et al. *Obes Surg*. 2009;19(10):1447–1455.
97. Buchwald H. *J Am Coll Surg*. 2002;194(3):367–375.
98. Franco JV, et al. *Obes Surg*. 2011;21(9):1458–1468.
99. Salminen P, et al. *JAMA*. 2018;319(3):241–254.
100. Peterli R, et al. *Ann Surg*. 2013;258(5):690–694. discussion 695.
101. Martins C, et al. *Obes Surg*. 2011;21(7):841–849.
102. Schauer PR, et al. *N Engl J Med*. 2012;366(17):1567–1576.

103. Mingrone G, et al. *N Engl J Med*. 2012;366(17):1577–1585.
104. Adams TD, et al. *JAMA*. 2012;308(11):1122–1131.
105. Jakobsen GS, et al. *JAMA*. 2018;319(3):291–301.
106. Schauer PR, et al. *N Engl J Med*. 2017;376(7):641–651.
107. Sarkhosh K, et al. *Obes Surg*. 2013;23(3):414–423.
108. Kushner RF, et al. *Mayo Clin Proc*. 2006;81(suppl 10):S46–S51.
109. Ikonomidis I, et al. *J Hypertens*. 2007;25(2):439–447.
110. Dixon JB, et al. *Obes Res*. 2002;10(9):903–910.
111. Bouldin MJ, et al. *Am J Med Sci*. 2006;331(4):183–193.
112. Champault A, et al. *Surg Laparosc Endosc Percutan Tech*. 2006;16(3):131–136.
113. Bolen SD, et al. *Obes Surg*. 2012;22(5):749–763.
114. Thomas H, et al. *Obes Surg*. 2012;22:1135–1140.
115. Lopez-Jimenez F, et al. *Mayo Clin Proc*. 2005;80(9):1157–1162.
116. Collazo-Clavell ML, et al. *Mayo Clin Proc*. 2006;81(suppl 10):S11–S17.
117. Hillman DR, et al. *Sleep Med Rev*. 2004;8(6):459–471.
118. Bostanjian D, et al. *Obes Surg*. 2003;13(2):302–305.
119. Collier B, et al. *Obes Surg*. 2003;13(6):941–943.
120. Passannante AN, et al. *Anesthesiol Clin*. 2005;23(3):479–491.
121. Mathews PV, et al. *J Orthop Trauma*. 2001;15(8):580–583.
122. Voyagis GS, et al. *Eur J Anaesthesiol*. 1998;15(3):330–334.
123. Juvin P, et al. *Anesth Analg*. 2003;97(2):595–600.
124. Ezri T, et al. *Anaesth*. 2003;58(11):1111–1114.
125. Shiga T, et al. *Anesthesiol*. 2005;103(2):429–437.
126. Collins JS, et al. *Obes Surg*. 2004;14(9):1171–1175.
127. Neligan PJ, et al. *Anesth Analg*. 2009;109(4):1182–1186.
128. Cattano D, et al. *Obes Surg*. 2010;20(10):1436–1441.
129. Kristensen MS. *Eur J Anaesthesiol*. 2010;27(11):923–927.
130. Schumann R. *Best Pract Res Clin Anaesthesiol*. 2011;25(1):83–93.
131. Yumul R, et al. *J Clin Anesth*. 2016;31:71–77.
132. Behazin N, et al. *Eur J Appl Physiol*. 2010;108(1):212–218.
133. Eikermann M, et al. *Open Respir Med J*. 2010;4:58–62.
134. Jense HG, et al. *Anesth Analg*. 1991;72(1):89–93.
135. Coussa M, et al. *Anesth Analg*. 2004;98(5):1491–1495.
136. Cressey DM, et al. *Anaesth*. 2001;56(7):680–684.
137. Gander S, et al. *Anesth Analg*. 2005;100(2):580–584.
138. Dixon BJ, et al. *Anesthesiol*. 2005;102(6):1110–1115.
139. Zavorsky GS, et al. *Chest*. 2007;131(2):362–367.
140. Boyce JR, et al. *Obes Surg*. 2003;13(1):4–9.
141. Perilli V, et al. *Anesth Analg*. 2000;91(6):1520–1525.
142. Ungern-Sternberg BS, et al. *Br J Anaesth*. 2004;92(2):202–207.
143. Bardoczky GI, et al. *Anesth Analg*. 1995;81(2):385–388.
144. Pelosi P, et al. *Anesthesiol*. 1999;91(5):1221–1231.
145. Reinius H, et al. *Anesthesiol*. 2009;111(5):979–987.
146. El Dawlatly AA, et al. *Obes Surg*. 2004;14(2):212–215.
147. Sprung J, et al. *Anesth Analg*. 2003;97(1):268–274.
148. Whalen FX, et al. *Anesth Analg*. 2006;102(1):298–305.
149. Erlandsson K, et al. *Acta Anaesthesiol Scand*. 2006;50(7):833–839.
150. Harbut P, et al. *Acta Anaesthesiol Scand*. 2014;58(6):675–680.
151. Neligan PJ, et al. *Anesthesiol*. 2009;110(4):878–884.
152. Pelosi P, et al. *Best Pract Res Clin Anaesthesiol*. 2010;24:211–225.
153. Candiotti K, et al. *Br J Anaesth*. 2009;103(suppl 1):i23–i30.
154. Dhonneur G, et al. *Anesth Analg*. 1999;89(3):762–767.
155. Christoff PB, et al. *Ann Pharmacother*. 1983;17(7):516–522.
156. Egan TD, et al. *Anesthesiol*. 1998;89(3):562–573.
157. Ogunnaike BO, et al. *Anesth Analg*. 2002;95(6):1793–1805.
158. Carron M, et al. *Anaesth*. 2012;67(3):298–299.
159. Juvin P, et al. *Anesth Analg*. 2000;91(3):714–719.
160. De Baerdemaeker L, et al. *Br J Anaesth*. 2003;91(5):638–650.
161. De Baerdemaeker L, et al. *Obes Surg*. 2006;16(6):728–733.
162. Kalinowski CP, et al. *Best Pract Res Clin Anaesthesiol*. 2004;18(4):719–737.
163. Jones KL, et al. *Diabetes Care*. 2001;24(7):1264–1269.
164. Illing L, et al. *Can J Anaesth*. 1992;39(5 Pt 1):466–470.
165. Maltby JR, et al. *Can J Anaesth*. 2004;51(2):111–115.
166. Hodgkinson R, et al. *Anesth Analg*. 1980;59(2):89–92.
167. Hodgkinson R, et al. *Anesth Analg*. 1981;60(6):421–424.
168. Ungern-Sternberg BS, et al. *Br J Anaesth*. 2005;94(1):121–127.
169. Capella J, et al. *Obes Surg*. 1996;6(1):50–53.
170. Vaziri K, et al. *Obes Surg*. 2011;21(10):1580–1584.
171. Ricciardi R, et al. *Surg Laparosc Endosc Percutan Tech*. 2006;16(5):317–320.
172. Gan TJ. *Anesth Analg*. 2006;102(6):1884–1898.
173. Lemanu DP, et al. *Br J Surg*. 2013;100(4):482–489.
174. Turner PL, et al. *Obes Surg*. 2011;21(5):655–662.
175. Lancaster RT, et al. *Surg Endosc*. 2008;22(12):2554–2563.
176. Poulose BK, et al. *J Surg Res*. 2005;127(1):1–7.
177. Fernandez AZ, et al. *Surg Endosc*. 2004;18(2):193–197.
178. Podnos YD, et al. *Arch Surg*. 2003;138(9):957–961.
179. Derzie AJ, et al. *J Am Coll Surg*. 2000;191(3):238–243.
180. Gonzalez R, et al. *J Am Coll Surg*. 2007;204(1):47–55.
181. Hwang RF, et al. *Surg Endosc*. 2004;18(11):1631–1635.
182. Livingston EH, et al. *J Am Coll Surg*. 2006;203(5):625–633.
183. Winegar DA, et al. *Surg Obes Relat Dis*. 2011;7(2):181–188.
184. Juhasz-Pocsine K, et al. *Neurol*. 2007;68(21):1843–1850.
185. Singh S, et al. *Mayo Clin Proc*. 2005;80(1):136–137.
186. Thaisetthawatkul P, et al. *Neurol*. 2004;63(8):1462–1470.
187. Parkes E. *Am J Med Sci*. 2006;331(4):207–213.
188. Poitou BC, et al. *Diabetes Metab*. 2007;33(1):13–24.
189. Xanthakos SA, et al. *Curr Opin Clin Nutr Metab Care*. 2006;9(4):489–496.
190. Dindo D, et al. *Lancet*. 2003;361(9374):2032–2035.
191. Jin R, et al. *Circulation*. 2005;111(25):3359–3365.
192. Shapiro M, et al. *N Engl J Med*. 1982;307(27):1661–1666.
193. Brandt M, et al. *Eur J Cardiothorac Surg*. 2001;19(5):662–666.
194. Engelman DT, et al. *J Thorac Cardiovasc Surg*. 1999;118(5):866–873.

References

1. Kaidar-Person O, Bar-Sela G, Person B. The two major epidemics of the twenty-first century: obesity and cancer. *Obes Surg*. 2011;21(11):1792–1797.
2. Haslam D. Obesity: a medical history. *Obes Rev*. 2007;8(suppl 1):31–36.
3. Baskin ML, Ard J, Franklin F, Allison DB. Prevalence of obesity in the United States. *Obes Rev*. 2005;6(1):5–7.
4. Hensrud DD, Klein S. Extreme obesity: a new medical crisis in the United States. *Mayo Clin Proc*. 2006;81(suppl 10):S5–10.
5. Canoy D, Buchan I. Challenges in obesity epidemiology. *Obes Rev*. 2007;8(suppl 1):1–11.
6. Obesity and Overweight Fact Sheet. www.who.int/mediacentre/factsheets/fs311/en.
7. Fryar CD, Carroll MD, Ogden CL. Prevalence of overweight, obesity, and extreme obesity among adults aged 20 and over: United States, 1960–1962 Through 2011–2014. National Center for Health Statistics Data; 2016.
8. Ogden CL, Carroll MD, Lawman HG, et al. Trends in obesity prevalence among children and adolescents in the United States, 1988-1994 through 2013-2014. *JAMA*. 2016;315(21):2292–2299.
9. Conway B, Rene A. Obesity as a disease: no lightweight matter. *Obes Rev*. 2004;5(3):145–151.
10. Zabena C, Gonzalez-Sanchez JL, Martinez-Larrad MT, et al. The FTO obesity gene. Genotyping and gene expression analysis in morbidly obese patients. *Obes Surg*. 2009;19(1):87–95.
11. Katzmarzyk PT, Janssen I, Ardern CI. Physical inactivity, excess adiposity and premature mortality. *Obes Rev*. 2003;4(4):257–290.
12. Kopelman P. Health risks associated with overweight and obesity. *Obes Rev*. 2007;8(suppl 1):13–17.
13. Nguyen NT, Nguyen XM, Lane J, Wang P. Relationship between obesity and diabetes in a US adult population: findings from the National Health and Nutrition Examination Survey, 1999-2006. *Obes Surg*. 2011;21(5):351–355.
14. Zammit C, Liddicoat H, Moonsie I, Makker H. Obesity and respiratory diseases. *Int. J. Gen. Med*. 2010;3:335–343.
15. Apovian CM, Gokce N. Obesity and cardiovascular disease. *Circulation*. 2012;125(9):1178–1182.
16. Wearing SC, Hennig EM, Byrne NM, Steele JR, Hills AP. Musculoskeletal disorders associated with obesity: a biomechanical perspective. *Obes Rev*. 2006;7(3):239–250.
17. Scopinaro N, Papadia F, Marinari G, Camerini G, Adami G. Long-term control of type 2 diabetes mellitus and the other major components of the metabolic syndrome after biliopancreatic diversion in patients with BMI < 35 kg/m^2. *Obes Surg*. 2007;17(2):185–192.
18. Guidone C, Manco M, Valera-Mora E, et al. Mechanisms of recovery from type 2 diabetes after malabsorptive bariatric surgery. *Diabetes*. 2006;55(7):2025–2031.
19. Pinkney J, Kerrigan D. Current status of bariatric surgery in the treatment of type 2 diabetes. *Obes Rev*. 2004;5(1):69–78.
20. Alam I, Lewis K, Stephens JW, Baxter JN. Obesity, metabolic syndrome and sleep apnoea: all pro-inflammatory states. *Obes Rev*. 2007;8(2):119–127.
21. Machado M, Cortez-Pinto H. Non-alcoholic steatohepatitis and metabolic syndrome. *Curr Opin Clin Nutr Metab Care*. 2006;9(5):637–642.
22. Valera-Mora ME, Simeoni B, Gagliardi L, et al. Predictors of weight loss and reversal of comorbidities in malabsorptive bariatric surgery. *Am J Clin Nutr*. 2005;81(6):1292–1297.
23. Poirier P, Cornier AM, Mazzone T, et al. Bariatric surgery and cardiovascular risk fractors. A scientific statement from the American Heart Association. *Circulation*. 2011;123:1683–1701.
24. Liberopoulos EN, Mikhailidis DP, Elisaf MS. Diagnosis and management of the metabolic syndrome in obesity. *Obes Rev*. 2005;6(4):283–296.
25. Levin PD, Weissman C. Obesity, metabolic syndrome, and the surgical patient. *Med Clin North Am*. 2009;93(5):1049–1063.
26. Ervin RB. Prevalence of metabolic syndrome among adults 20 years of age and over, by sex, age, race and ethnicity, and body mass index: United States, 2003-2006. *Natl Vital Stat Rep*. 2009;(13):1–7.
27. O'Neill S, O'Driscoll L. Metabolic syndrome: a closer look at the growing epidemic and its associated pathologies. *Obes Rev*. 2015;16(1):1–12.
28. Buchwald H, Avidor Y, Braunwald E, et al. Bariatric surgery: a systematic review and meta-analysis. *JAMA*. 2004;292(14):1724–1737.
29. Frezza EE, Wachtel M. Metabolic syndrome: a new multidisciplinary service line. *Obes Surg*. 2011;21(3):379–385.
30. Buchwald H, Oien DM. Metabolic/bariatric surgery worldwide 2008. *Obes Surg*. 2009;19(12):1605–1611.
31. Moulin CM, Marguti I, Peron JP, Halpern A, Rizzo LV. Bariatric surgery reverses natural killer (NK) cell activity and NK-related cytokine synthesis impairment induced by morbid obesity. *Obes Surg*. 2011;21(1):112–118.
32. Selmi C, Montano N, Furlan R, Keen CL, Gershwin ME. Inflammation and oxidative stress in obstructive sleep apnea syndrome. *Exp Biol Med*. 2007;232(11):1409–1413.
33. Patil SP, Schneider H, Schwartz AR, Smith PL. Adult obstructive sleep apnea - pathophysiology and diagnosis. *Chest*. 2007;132(1):325–337.
34. O'Keefe T, Patterson EJ. Evidence supporting routine polysomnography before bariatric surgery. *Obes Surg*. 2004;14:23–26.
35. Chung F, Subramanyam R, Liao P, Sasaki E, Shapiro C, Sun Y. High STOP-Bang score indicates a high probability of obstructive sleep apnoea. *Br J Anaesth*. 2012;108(5):768–775.
36. Tung A, Rock P. Perioperative concerns in sleep apnea. *Curr Opin Anaesthesiol*. 2001;14:671–678.
37. Biro P, Kaplan V, Bloch KE. Anesthetic management of a patient with obstructive sleep apnea syndrome and difficult airway access. *J Clin Anesth*. 1995;7(5):417–421.
38. Pierce RJ, Worsnop CJ. Upper airway function and dysfunction in respiration. *Clin Exp Pharmacol P*. 1999;26(1):1–10.
39. Mayer P, Pepin JL, Bettega G, et al. Relationship between body mass index, age and upper airway measurements in snorers and sleep apnoea patients. *Eur Respir J*. 1996;9(9):1801–1809.
40. Benumof JL. Obstructive sleep apnea in the adult obese patient: implications for airway management. *Anesthesiol Clin*. 2002;20(4):789–811.
41. Brodsky JB, Lemmens HJM, Brock-Utne JG, Vierra M, Saidman LJ. Morbid obesity and tracheal intubation. *Anesth Analg*. 2002;94(3):732–736.
42. Gomez-Illan F, Ortega-Gonzalves M, Soler-Orea I, et al. Obesity and inflammation: change in cdiponectin, C-reactive protein, tumour necrosis factor-Alpha and Interleukin-6 after bariatric surgery. *Obes Surg*. 2012;22:950–955.
43. Roth CL, Reinehr T, Schernthaner GH, Kopp HP, Kriwanek S, Schernthaner G. Ghrelin and obestatin levels in severely obese women before and after weight loss after Roux-en-Y gastric bypass surgery. *Obes Surg*. 2009;(1):29–35.
44. National Cholesterol Education Program (NCEP). Third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III) final report. *Circulation*. 2002;106(25):3143–3421.
45. Kushner RF. Weight loss strategies for treatment of obesity. *Prog Cardiovasc Dis*. 2014;56(4):465–472.
46. Grundy SM, Brewer HB, Cleeman JI, Smith SC, Lenfant C, Participants ftC. Definition of metabolic syndrome. *Circulation*. 2004;109(3):433–438.
47. Low AK, Bouldin MJ, Sumrall CD, Loustalot FV, Land KK. A clinician's approach to medical management of obesity. *Am J Med Sci*. 2006;331(4):175–182.
48. Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: executive summary: a report of the American College of Cardiology/American Heart Association task force on clinical practice guidelines. *Hypertension*. 2017.
49. Martinez EA, Williams KA, Pronovost PJ. Thinking like a pancreas: perioperative glycemic control. *Anesth Analg*. 2007;104(1):4–6.
50. Bray GA. Medical treatment of obesity: the past, the present and the future. *Best Pract Res Clin Gastroenterol*. 2014;28(4):665–684.
51. Allison DB, Fontaine KR, Heshka S, Mentore JL, Heymsfield SB. Alternative treatments for weight loss: a critical review. *Crit Rev Food Sci Nutr*. 2001;41(1):1–28.
52. Boozer CN, Nasser JA, Heymsfield SB, Wang V, Chen G, Solomon JL. An herbal supplement containing ma huang-guarana for weight loss: a randomized, double-blind trial. *Int J Obes Relat Metab Disord*. 2001;25(3):316–324.
53. Boozer CN, Daly PA, Homel P, et al. Herbal ephedra/caffeine for weight loss: a 6-month randomized safety and efficacy trial. *Int J Obes Relat Metab Disord*. 2002;26(5):593–604.

54. Astrup A, Breum L, Toubro S, Hein P, Quaade F. The effect and safety of an ephedrine/caffeine compound compared to ephedrine, caffeine and placebo in obese subjects on an energy restricted diet. A double blind trial. *Int J Obes Relat Metab Disord*. 1992;16(4):269–277.
55. Haller CA, Benowitz NL. Adverse cardiovascular and central nervous system events associated with dietary supplements containing ephedra alkaloids. *N Engl J Med*. 2000;343(25):1833–1838.
56. Cigaina V. Long-term follow-up of gastric stimulation for obesity: the Mestre 8-year experience. *Obes Surg*. 2004;14(suppl 1):S14–S22.
57. Favretti F, De Luca M, Segato G, et al. Treatment of morbid obesity with the Transcend Implantable Gastric Stimulator (IGS): a prospective survey. *Obes Surg*. 2004;14(5):666–670.
58. Cha R, Marescaux J, Diana M. Updates on gastric electrical stimulation to treat obesity: systematic review and future perspectives. *World J Gastrointest Endosc*. 2014;6(9):419–431.
59. Dietz WH, Robinson TN. Overweight children and adolescents. *N Engl J Med*. 2005;352(20):2100–2109.
60. Fryar CD, Carroll MD, Ogden CL. Prevalence of overweight and obesity among children and adolescents aged 2–19 years: United States, 1963–1965 through 2013–2014. Surveys, D. o. H. a. N. E., Ed; 2016.
61. de Onis M, Blossner M. Prevalence and trends of overweight among preschool children in developing countries. *Am J Clin Nutr*. 2000;72(4):1032–1039.
62. Kirk S, Scott BJ, Daniels SR. Pediatric obesity epidemic: treatment options. *J Am Diet Assoc*. 2005;105(suppl 5):44–51.
63. Boland CL, Harris JB, Harris KB. Pharmacological management of obesity in pediatric patients. *Ann Pharmacother*. 2015;49(2):220–232.
64. Tsai WS, Inge TH, Burd RS. Bariatric surgery in adolescents: recent national trends in use and in-hospital outcome. *Arch Pediatr Adolesc Med*. 2007;161(3):217–221.
65. Christou NV, Sampalis JS, Liberman M, et al. Surgery decreases long-term mortality, morbidity, and health care use in morbidly obese patients. *Ann of Surg*. 2004;240(3):416–423.
66. Pereira JA, Lazarin MA, Pareja JC, de Souza A, Muscelli E. Insulin resistance in nondiabetic morbidly obese patients: effect of bariatric surgery. *Obes Res*. 2003;11(12):1495–1501.
67. Buchwald H, Conference C. Bariatric surgery for morbid obesity: health implications for patients, health professionals, and third-party payers. *J Am Coll Surg*. 2005;200(4):593–604.
68. Shaffer EA. Bariatric surgery: a promising solution for nonalcoholic steatohepatitis in the very obese. *J Clin Gastroenterol*. 2006;40(3 suppl 1):S44–S50.
69. Encinosa WE, Bernard DM, Chen CC, Steiner CA. Healthcare utilization and outcomes after bariatric surgery. *Med Care*. 2006;44(8):706–712.
70. Flum DR, Dellinger EP. Impact of gastric bypass operation on survival: a population-based analysis. *J Am Coll Surg*. 2004;199(4):543–551.
71. Lynch RJ, Eisenberg D, Bell RL. Metabolic consequences of bariatric surgery. *J Clin Gastroenterol*. 2006;40(8):659–668.
72. Akkary E. Bariatric surgery evolution from the malabsorptive to the hormonal era. *Obes Surg*. 2012;22(5):827–831.
73. Buchwald H, Oien DM. Metabolic/bariatric surgery worldwide 2011. *Obes Surg*. 2013;23(4):427–436.
74. Salameh JR. Bariatric surgery: past and present. *Am J Med Sci*. 2006;331(4):194–200.
75. Poulose BK, Holzman MD, Zhu Y, et al. National variations in morbid obesity and bariatric surgery use. *J Am Coll Surg*. 2005;201(1):77–84.
76. Santry HP, Lauderdale DS, Cagney KA, Rathouz PJ, Alverdy JC, Chin MH. Predictors of patient selection in bariatric surgery. *Ann of Surg*. 2007;245(1):59–67.
77. Weber M, Muller MK, Bucher T, et al. Laparoscopic gastric bypass is superior to laparoscopic gastric banding for treatment of morbid obesity. *Ann of Surg*. 2004;240(6):975–982.
78. Deitel M, Shikora SA. The development of the surgical treatment of morbid obesity. *J Am Coll Nutr*. 2002;21(5):365–371.
79. Galvani C, Gorodner M, Moser F, et al. Laparoscopic adjustable gastric band versus laparoscopic Roux-en-Y gastric bypass: ends justify the means? *Surg Endosc*. 2006;20(6):934–941.
80. Kendrick ML, Dakin GF. Surgical approaches to obesity. *Mayo Clin Proc*. 2006;81(suppl 10):S18–S24.
81. Deitel M. A synopsis of the development of bariatric operations. *Obes Rev*. 2007;17(6):707–710.
82. Eisenberg D, Duffy AJ, Bell RL. Update on obesity surgery. *World J Gastroenterol*. 2006;12(20):3196–3203.
83. Montgomery K, Watkins B, Ahroni J, et al. Outpatient laparoscopic adjustable gastric banding in super-obese patients. *Obes Surg*. 2007;17:711–716.
84. Weller WE, Rosati C. Comparing outcomes of laparoscopic versus open bariatric surgery. *Ann Surg*. 2008;248(1):10–15.
85. Nguyen NT, Masoomi H, Magno CP, Nguyen XM, Laugenour K, Lane J. Trends in use of bariatric surgery, 2003-2008. *J Am Coll Surg*. 2011;213(2):261–266.
86. Estimate of Bariatric Surgery Numbers, 2011-2016. https://asmbs.org/resources/estimate-of-bariatric-surgery-numbers.
87. van Rutte PW, Smulders JF, de Zoete JP, Nienhuijs SW. Outcome of sleeve gastrectomy as a primary bariatric procedure. *Br J Surg*. 2014;101(6):661–668.
88. Ramon JM, Salvans S, Crous X, et al. Effect of Roux-en-Y gastric bypass vs sleeve gastrectomy on glucose and gut hormones: a prospective randomised trial. *J Gastrointest Surg*. 2012;16(6):1116–1122.
89. Schauer PR, Ikramuddin S, Gourash W, Ramanathan R, Luketich J. Outcomes after laparoscopic Roux-en-Y gastric bypass for morbid obesity. *Ann of Surg*. 2000;232(4):515–529.
90. Gentileschi P, Kini S, Catarci M, Gagner M. Evidence-based medicine: open and laparoscopic bariatric surgery. *Surg Endosc*. 2002;16(5):736–744.
91. Huang CK, Tsai JC, Lo CH, et al. Preliminary surgical results of single-incision transumbilical laparoscopic bariatric surgery. *Obes Surg*. 2011;21(3):391–396.
92. Malinowski SS. Nutritional and metabolic complications of bariatric surgery. *Am J Med Sci*. 2006;331(4):219–225.
93. Mathus-Vliegen EM, Tytgat GN. Intragastric balloon for treatment-resistant obesity: safety, tolerance, and efficacy of 1-year balloon treatment followed by a 1-year balloon-free follow-up. *Gastrointest endosc*. 2005;61(1):19–27.
94. Gaur S, Levy S, Mathus-Vliegen L, Chuttani R. Balancing risk and reward: a critical review of the intragastric balloon for weight loss. *Gastrointest Endosc*. 2015;81(6):1330–1336.
95. Thompson CC, Abu Dayyeh BK, Kushner R, et al. Percutaneous gastrostomy device for the treatment of class ii and class iii obesity: results of a randomized controlled trial. *Am J Gastroenterol*. 2017;112(3):447–457.
96. Garb J, Welch G, Zagarins S, Kuhn J, Romanelli J. Bariatric surgery for the treatment of morbid obesity: a meta-analysis of weight loss outcomes for laparoscopic adjustable gastric banding and laparoscopic gastric bypass. *Obes Surg*. 2009;19(10):1447–1455.
97. Buchwald H. Overview of bariatric surgery. *J Am Coll Surg*. 2002;194(3):367–375.
98. Franco JV, Ruiz PA, Palermo M, Gagner M. A review of studies comparing three laparoscopic procedures in bariatric surgery: sleeve gastrectomy, Roux-en-Y gastric bypass and adjustable gastric banding. *Obes Surg*. 2011;21(9):1458–1468.
99. Salminen P, Helmio M, Ovaska J, et al. Effect of laparoscopic sleeve gastrectomy vs laparoscopic Roux-en-Y gastric bypass on weight loss at 5 years among patients with morbid obesity: the SLEEVEPASS randomized clinical trial. *JAMA*. 2018;319(3):241–254.
100. Peterli R, Borbely Y, Kern B, et al. Early results of the Swiss Multicentre Bypass or Sleeve Study (SM-BOSS): a prospective randomized trial comparing laparoscopic sleeve gastrectomy and Roux-en-Y gastric bypass. *Ann Surg*. 2013;258(5):690–694. discussion 695.
101. Martins C, Strommen M, Stavne OA, Nossum R, Marvik R, Kulseng B. Bariatric surgery versus lifestyle interventions for morbid obesity--changes in body weight, risk factors and comorbidities at 1 year. *Obes Surg*. 2011;21(7):841–849.
102. Schauer PR, Kashyap SR, Wolski K, et al. Bariatric surgery versus intensive medical therapy in obese patients with diabetes. *N Engl J Med*. 2012;366(17):1567–1576.
103. Mingrone G, Panunzi S, De Gaetano A, et al. Bariatric surgery versus conventional medical therapy for type 2 diabetes. *N Engl J Med*. 2012;366(17):1577–1585.
104. Adams TD, Davidson LE, Litwin SE, et al. Health benefits of gastric bypass surgery after 6 years. *JAMA*. 2012;308(11):1122–1131.
105. Jakobsen GS, Smastuen MC, Sandbu R, et al. Association of bariatric surgery vs medical obesity treatment with long-term medical complications and obesity-related comorbidities. *JAMA*. 2018;319(3):291–301.

106. Schauer PR, Bhatt DL, Kirwan JP, et al. Bariatric surgery versus intensive medical therapy for diabetes - 5-Year Outcomes. *N Engl J Med*. 2017;376(7):641–651.
107. Sarkhosh K, Switzer NJ, El-Hadi M, Birch DW, Shi X, Karmali S. The impact of bariatric surgery on obstructive sleep apnea: a systematic review. *Obes Surg*. 2013;23(3):414–423.
108. Kushner RF, Noble CA. Long-term outcome of bariatric surgery: an interim analysis. *Mayo Clin Proc*. 2006;81(suppl 10):S46–S51.
109. Ikonomidis I, Mazarakis A, Papadopoulos C, et al. Weight loss after bariatric surgery improves aortic elastic properties and left ventricular function in individuals with morbid obesity: a 3-year follow-up study. *J Hypertens*. 2007;25(2):439–447.
110. Dixon JB, O'Brien PE. Lipid profile in the severely obese: changes with weight loss after lap-band surgery. *Obes Res*. 2002;10(9):903–910.
111. Bouldin MJ, Ross LA, Sumrall CD, Loustalot FV, Low AK, Land KK. The effect of obesity surgery on obesity comorbidity. *Am J Med Sci*. 2006;331(4):183–193.
112. Champault A, Duwat O, Polliand C, Rizk N, Champault GG. Quality of life after laparoscopic gastric banding: prospective study (152 cases) with a follow-up of 2 years. *Surg Laparosc Endosc Percutan Tech*. 2006;16(3):131–136.
113. Bolen SD, Chang HY, Weiner JP, et al. Clinical outcomes after bariatric surgery: a five-year matched cohort analysis in seven US states. *Obes Surg*. 2012;22(5):749–763.
114. Thomas H, Agrawal S. Systematic review of obesity mortality risk score-preoperative risk stratification in bariatric surgery. *Obes Surg*. 2012;22:1135–1140.
115. Lopez-Jimenez F, Bhatia S, Collazo-Clavell ML, Sarr MG, Somers VK. Safety and efficacy of bariatric surgery in patients with coronary artery disease. *Mayo Clin Proc*. 2005;80(9):1157–1162.
116. Collazo-Clavell ML, Clark MM, McAlpine DE, Jensen MD. Assessment and preparation of patients for bariatric surgery. *Mayo Clin Proc*. 2006;81(suppl 10):S11–S17.
117. Hillman DR, Loadsman JA, Platt PR, Eastwood PR. Obstructive sleep apnoea and anaesthesia. *Sleep Med Rev*. 2004;8(6):459–471.
118. Bostanjian D, Anthone G, Hamoui N, Crookes P. Rhabdomyolysis of gluteal muscles leading to renal failure: a potentially fatal complication of surgery in the morbidly obese. *Obes Surg*. 2003;13(2):302–305.
119. Collier B, Goreja M, Duke B. Postoperative rhabdomyolysis with bariatric surgery. *Obes Surg*. 2003;13(6):941–943.
120. Passannante AN, Rock P. Anesthetic management of patients with obesity and sleep apnea. *Anesthesiol Clin*. 2005;23(3):479–491.
121. Mathews PV, Perry JJ, Murray PC. Compartment syndrome of the well leg as a result of the hemilithotomy position: a report of two cases and review of literature. *J Orthop Trauma*. 2001;15(8):580–583.
122. Voyagis GS, Kyriakis KP, Dimitriou V, Vrettou I. Value of oropharyngeal Mallampati classification in predicting difficult laryngoscopy among obese patients. *Eur J Anaesthesiol*. 1998;15(3):330–334.
123. Juvin P, Lavaut E, Dupont H, et al. Difficult tracheal intubation is more common in obese than in lean patients. *Anesth Analg*. 2003;97(2):595–600.
124. Ezri T, Gewurtz G, Sessler DI, et al. Prediction of difficult laryngoscopy in obese patients by ultrasound quantification of anterior neck soft tissue. *Anaesth*. 2003;58(11):1111–1114.
125. Shiga T, Wajima Z, Inoue T, Sakamoto A. Predicting difficult intubation in apparently normal patients - a meta-analysis of bedside screening test performance. *Anesthesiol*. 2005;103(2):429–437.
126. Collins JS, Lemmens HJM, Brodsky JB, Brock-Utne JG, Levitan RM. Laryngoscopy and morbid obesity: a comparison of the "sniff" and "ramped" positions. *Obes Surg*. 2004;14(9):1171–1175.
127. Neligan PJ, Porter S, Max B, Malhotra G, Greenblatt EP, Ochroch EA. Obstructive sleep apnea is not a risk factor for difficult intubation in morbidly obese patients. *Anesth Analg*. 2009;109(4):1182–1186.
128. Cattano D, Melnikov V, Khalil Y, Sridhar S, Hagberg CA. An evaluation of the rapid airway management positioner in obese patients undergoing gastric bypass or laparoscopic gastric banding surgery. *Obes Surg*. 2010;20(10):1436–1441.
129. Kristensen MS. Airway management and morbid obesity. *Eur J Anaesthesiol*. 2010;27(11):923–927.
130. Schumann R. Anaesthesia for bariatric surgery. *Best Pract Res Clin Anaesthesiol*. 2011;25(1):83–93.
131. Yumul R, Elvir-Lazo OL, White PF, et al. Comparison of three video laryngoscopy devices to direct laryngoscopy for intubating obese patients: a randomized controlled trial. *J Clin Anesth*. 2016;31:71–77.
132. Behazin N, Jones SB, Cohen RI, Loring SH. Respiratory restriction and elevated pleural and esophageal pressures in morbid obesity. *Eur J Appl Physiol*. 2010;108(1):212–218.
133. Eikermann M, Serrano-Garzon J, Kwo J, Sundrup-Grosse M, Schmidt U, Bigatello L. Do patients with obstructive sleep apnea have an increased risk of desaturation during induction of anesthesia for weight loss surgery? *Open Respir Med J*. 2010;4:58–62.
134. Jense HG, Dubin SA, Silverstein PI, O'Leary-Escolas U. Effect of obesity on safe duration of apnea in anesthetized humans. *Anesth Analg*. 1991;72(1):89–93.
135. Coussa M, Proietti S, Schnyder P, et al. Prevention of atelectasis formation during the induction of general anesthesia in morbidly obese patients. *Anesth Analg*. 2004;98(5):1491–1495.
136. Cressey DM, Berthoud MC, Reilly CS. Effectiveness of continuous positive airway pressure to enhance pre-oxygenation in morbidly obese women. *Anaesth*. 2001;56(7):680–684.
137. Gander S, Frascarolo P, Suter M, Spahn DR, Magnusson L. Positive end-expiratory pressure during induction of general anesthesia increases duration of nonhypoxic apnea in morbidly obese patients. *Anesth Analg*. 2005;100(2):580–584.
138. Dixon BJ, Dixon JB, Carden JR, et al. Preoxygenation is more effective in the 25 degrees head-up position than in the supine position in severely obese patients: a randomized controlled study. *Anesthesiol*. 2005;102(6):1110–1115.
139. Zavorsky GS, Murias JM, Kim dJ, Gow J, Sylvestre JL, Christou NV. Waist-to-hip ratio is associated with pulmonary gas exchange in the morbidly obese. *Chest*. 2007;131(2):362–367.
140. Boyce JR, Ness T, Castroman P, Gleysteen JJ. A preliminary study of the optimal anesthesia positioning for the morbidly obese patient. *Obes Surg*. 2003;13(1):4–9.
141. Perilli V, Sollazzi L, Bozza P, et al. The effects of the reverse trendelenburg position on respiratory mechanics and blood gases in morbidly obese patients during bariatric surgery. *Anesth Analg*. 2000;91(6):1520–1525.
142. Ungern-Sternberg BS, Regli A, Schneider MC, Kunz F, Reber A. Effect of obesity and site of surgery on perioperative lung volumes. *Br J Anaesth*. 2004;92(2):202–207.
143. Bardoczky GI, Yernault JC, Houben JJ, d'Hollander AA. Large tidal volume ventilation does not improve oxygenation in morbidly obese patients during anesthesia. *Anesth Analg*. 1995;81(2):385–388.
144. Pelosi P, Ravagnan I, Giurati G, et al. Positive end-expiratory pressure improves respiratory function in obese but not in normal subjects during anesthesia and paralysis. *Anesthesiol*. 1999;91(5):1221–1231.
145. Reinius H, Jonsson L, Gustafsson S, et al. Prevention of atelectasis in morbidly obese patients during general anesthesia and paralysis: a computerized tomography study. *Anesthesiol*. 2009;111(5):979–987.
146. El Dawlatly AA, Al Dohayan A, Abdel-Meguid ME, El Bakry A, Manaa EM. The effects of pneumoperitoneum on respiratory mechanics during general anesthesia for bariatric surgery. *Obes Surg*. 2004;14(2):212–215.
147. Sprung J, Whalley DG, Falcone T, Wilks W, Navratil JE, Bourke DL. The effects of tidal volume and respiratory rate on oxygenation and respiratory mechanics during laparoscopy in morbidly obese patients. *Anesth Analg*. 2003;97(1):268–274.
148. Whalen FX, Gajic O, Thompson GB, et al. The effects of the alveolar recruitment maneuver and positive end-expiratory pressure on arterial oxygenation during laparoscopic bariatric surgery. *Anesth Analg*. 2006;102(1):298–305.
149. Erlandsson K, Odenstedt H, Lundin S, Stenqvist O. Positive end-expiratory pressure optimization using electric impedance tomography in morbidly obese patients during laparoscopic gastric bypass surgery. *Acta Anaesthesiol Scand*. 2006;50(7):833–839.
150. Harbut P, Gozdzik W, Stjernfalt E, Marsk R, Hesselvik JF. Continuous positive airway pressure/pressure support pre-oxygenation of morbidly obese patients. *Acta Anaesthesiol Scand*. 2014;58(6):675–680.
151. Neligan PJ, Malhotra G, Fraser M, et al. Continuous positive airway pressure via the Boussignac system immediately after extubation improves lung function in morbidly obese patients with obstructive sleep apnea undergoing laparoscopic bariatric surgery. *Anesthesiol*. 2009;110(4):878–884.
152. Pelosi P, Gregoretti C. Perioperative management of obese patients. *Best Pract Res Clin Anaesthesiol*. 2010;24:211–225.
153. Candiotti K, Sharma S, Shankar R. Obesity, obstructive sleep apnoea, and diabetes mellitus: anaesthetic implications. *Br J Anaesth*. 2009;103(suppl 1):i23–i30.

154. Dhonneur G, Combes X, Leroux B, Duvaldestin P. Postoperative obstructive apnea. *Anesth Analg*. 1999;89(3):762–767.
155. Christoff PB, Conti DR, Naylor C, Jusko WJ. Procainamide disposition in obesity. *Ann Pharmacother*. 1983;17(7):516–522.
156. Egan TD, Huizinga B, Gupta SK, et al. Remifentanil pharmacokinetics in obese versus lean patients. *Anesthesiol*. 1998;89(3):562–573.
157. Ogunnaike BO, Jones SB, Jones DB, Provost D, Whitten CW. Anesthetic considerations for bariatric surgery. *Anesth Analg*. 2002;95(6):1793–1805.
158. Carron M, Freo U, Parotto E, Ori C. The correct dosing regimen for sugammadex in morbidly obese patients. *Anaesth*. 2012;67(3):298–299.
159. Juvin P, Vadam C, Malek L, Dupont H, Marmuse JP, Desmonts JM. Postoperative recovery after desflurane, propofol, or isoflurane anesthesia among morbidly obese patients: a prospective, randomized study. *Anesth Analg*. 2000;91(3):714–719.
160. De Baerdemaeker L, Struys M, Jacobs S, et al. Optimization of desflurane administration in morbidly obese patients: a comparison with sevoflurane using an "inhalation bolus" technique. *Br J Anaesth*. 2003;91(5):638–650.
161. De Baerdemaeker L, Jacobs S, Den Blauwen N, et al. Postoperative results after desflurane or sevoflurane combined with remifentanil in morbidly obese patients. *Obes Surg*. 2006;16(6):728–733.
162. Kalinowski CP, Kirsch JR. Strategies for prophylaxis and treatment for aspiration. *Best Pract Res Clin Anaesthesiol*. 2004;18(4):719–737.
163. Jones KL, Russo A, Stevens JE, Wishart JM, Berry MK, Horowitz M. Predictors of delayed gastric emptying in diabetes. *Diabetes Care*. 2001;24(7):1264–1269.
164. Illing L, Duncan PG, Yip R. Gastroesophageal reflux during anaesthesia. *Can J Anaesth*. 1992;39(5 Pt 1):466–470.
165. Maltby JR, Pytka S, Watson NC, Cowan RA, Fick GH. Drinking 300 mL of clear fluid two hours before surgery has no effect on gastric fluid volume and pH in fasting and non-fasting obese patients. *Can J Anaesth*. 2004;51(2):111–115.
166. Hodgkinson R, Husain FJ. Obesity and the cephalad spread of analgesia following epidural administration of bupivacaine for cesarean section. *Anesth Analg*. 1980;59(2):89–92.
167. Hodgkinson R, Husain FJ. Obesity, gravity, and spread of epidural anesthesia. *Anesth Analg*. 1981;60(6):421–424.
168. Ungern-Sternberg BS, Regli A, Reber A, Schneider MC. Effect of obesity and thoracic epidural analgesia on perioperative spirometry. *Br J Anaesth*. 2005;94(1):121–127.
169. Capella J, Capella R. Is routine invasive monitoring indicated in surgery for the morbidly obese? *Obes Surg*. 1996;6(1):50–53.
170. Vaziri K, Watson JD, Harper AP, et al. Prophylactic inferior vena cava filters in high-risk bariatric surgery. *Obes Surg*. 2011;21(10):1580–1584.
171. Ricciardi R, Town RJ, Kellogg TA, Ikramuddin S, Baxter NN. Outcomes after open versus laparoscopic gastric bypass. *Surg Laparosc Endosc Percutan Tech*. 2006;16(5):317–320.
172. Gan TJ. Risk factors for postoperative nausea and vomiting. *Anesth Analg*. 2006;102(6):1884–1898.
173. Lemanu DP, Singh PP, Berridge K, et al. Randomized clinical trial of enhanced recovery versus standard care after laparoscopic sleeve gastrectomy. *Br J Surg*. 2013;100(4):482–489.
174. Turner PL, Saager L, Dalton J, et al. A nomogram for predicting surgical complications in bariatric surgery patients. *Obes Surg*. 2011;21(5):655–662.
175. Lancaster RT, Hutter MM. Bands and bypasses: 30-day morbidity and mortality of bariatric surgical procedures as assessed by prospective, multi-center, risk-adjusted ACS-NSQIP data. *Surg Endosc*. 2008;22(12):2554–2563.
176. Poulose BK, Griffin MR, Moore DE, et al. Risk factors for post-operative mortality in bariatric surgery. *J Surg Res*. 2005;127(1):1–7.
177. Fernandez AZ, DeMaria EJ, Tichansky DS, et al. Experience with over 3,000 open and laparoscopic bariatric procedures: multivariate analysis of factors related to leak and resultant mortality. *Surg Endosc*. 2004;18(2):193–197.
178. Podnos YD, Jimenez JC, Wilson SE, Stevens CM, Nguyen NT. Complications after laparoscopic gastric bypass: a review of 3464 cases. *Arch Surg*. 2003;138(9):957–961.
179. Derzie AJ, Silvestri F, Liriano E, Benotti P. Wound closure technique and acute wound complications in gastric surgery for morbid obesity: a prospective randomized trial. *J Am Coll Surg*. 2000;191(3):238–243.
180. Gonzalez R, Sarr MG, Smith CD, et al. Diagnosis and contemporary management of anastomotic leaks after gastric bypass for obesity. *J Am Coll Surg*. 2007;204(1):47–55.
181. Hwang RF, Swartz DE, Felix EL. Causes of small bowel obstruction after laparoscopic gastric bypass. *Surg Endosc*. 2004;18(11):1631–1635.
182. Livingston EH, Arterburn D, Schifftner TL, Henderson WG, DePalma RG. National Surgical Quality Improvement Program analysis of bariatric operations: modifiable risk factors contribute to bariatric surgical adverse outcomes. *J Am Coll Surg*. 2006;203(5):625–633.
183. Winegar DA, Sherif B, Pate V, DeMaria EJ. Venous thromboembolism after bariatric surgery performed by Bariatric Surgery Center of Excellence Participants: analysis of the Bariatric Outcomes Longitudinal Database. *Surg Obes Relat Dis*. 2011;7(2):181–188.
184. Juhasz-Pocsine K, Rudnicki SA, Archer RL, Harik SI. Neurologic complications of gastric bypass surgery for morbid obesity. *Neurol*. 2007;68(21):1843–1850.
185. Singh S, Nautiyal A. Neurologic complications of bariatric surgery. *Mayo Clin Proc*. 2005;80(1):136–137.
186. Thaisetthawatkul P, Collazo-Clavell ML, Sarr MG, Norell JE, Dyck PJ. A controlled study of peripheral neuropathy after bariatric surgery. *Neurol*. 2004;63(8):1462–1470.
187. Parkes E. Nutritional management of patients after bariatric surgery. *Am J Med Sci*. 2006;331(4):207–213.
188. Poitou BC, Ciangura C, Coupaye M, Czernichow S, Bouillot JL, Basdevant A. Nutritional deficiency after gastric bypass: diagnosis, prevention and treatment. *Diabetes Metab*. 2007;33(1):13–24.
189. Xanthakos SA, Inge TH. Nutritional consequences of bariatric surgery. *Curr Opin Clin Nutr Metab Care*. 2006;9(4):489–496.
190. Dindo D, Muller MK, Weber M, Clavien PA. Obesity in general elective surgery. *Lancet*. 2003;361(9374):2032–2035.
191. Jin R, Grunkemeier GL, Furnary AP, Handy JR. Is obesity a risk factor for mortality in coronary artery bypass surgery? *Circulation*. 2005;111(25):3359–3365.
192. Shapiro M, Munoz A, Tager IB, Schoenbaum SC, Polk BF. Risk factors for infection at the operative site after abdominal or vaginal hysterectomy. *N Engl J Med*. 1982;307(27):1661–1666.
193. Brandt M, Harder K, Walluscheck KP, et al. Severe obesity does not adversely affect perioperative mortality and morbidity in coronary artery bypass surgery. *Eur J Cardiothorac Surg*. 2001;19(5):662–666.
194. Engelman DT, Adams DH, Byrne JG, et al. Impact of body mass index and albumin on morbidity and mortality after cardiac surgery. *J Thorac Cardiovasc Surg*. 1999;118(5):866–873.