CHAPTER 102 ■ THE OBESE SURGICAL PATIENT IN THE CRITICAL CARE UNIT

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Throughout the economically developed world, the incidence of obesity is rising at epidemic proportions. University medical centers are reporting that 25% of routine surgical patients are obese, and at least 10% of all patients are morbidly obese. Given the increasing prevalence of obesity in the general population, it is not surprising that many morbidly obese patients undergoing surgery are treated in intensive care units (ICU). Nevertheless, the real prevalence of critically ill morbidly obese patients, even in the United States, is not known. A retrospective study has allowed researchers to estimate that the incidence rate of morbidly obese patients requiring nonsurgical intensive care treatment approaches 14 cases per 1,000 ICU admissions annually. Bariatric surgical procedures alone have increased in the United States from 37,000 cases in 2000 up to 200,000 in 2006 as reported by the American Society for Bariatric Surgery (1).

The critically ill morbidly obese surgical patient presents the critical care team with many unique problems (2). As a result, every health care provider eventually involved in surgical procedures must be familiar with the management of morbidly obese patients, not only for bariatric procedures, but also for all types of surgery (3,4). Morbidly obese patients have an eight-fold higher mortality rate after blunt trauma than nonobese patients presenting with the same diagnosis (5). Retrospective reviews of morbidly obese patients—hospitalized with or without ICU requirements—have shown significant increases in length of ICU stay, mortality, and duration of mechanical ventilation (6–9).

The pathophysiologic consequences of obesity involve all major organ systems (10). Conditions such as diabetes mellitus and hyperlipidemia are associated with obesity and contribute to chronic morbidity in the obese. However, the main concerns for the intensivist, anesthesiologist, and surgeon have been the same for over three decades: the derangements of the cardiopulmonary system (11,12).

The perioperative care of these patients must be understood as a continuous, indivisible, and dynamic process that requires multidisciplinary involvement from surgeons, anesthesiologists, internists, and intensivists. Collaborative and coordinated activity within the surgical team is vital in these scenarios involving the morbidly obese (13). In this chapter, we will discuss cardiac and respiratory diseases in the morbidly obese patient undergoing a surgical procedure, following the above-mentioned focus, from preoperative assessment to postoperative care.

SPECIFIC SURGICAL ISSUES

Obese patients undergoing emergency general surgery are particularly challenging for the team managing the patient before and after surgery. These patients occasionally present with more advanced disease than otherwise might be expected due to the obesity causing delays in diagnosis.

The first decision to be made is whether or not the procedure can be performed laparoscopically, or if an open procedure is required. Where possible, it has been our practice to perform emergency surgery laparoscopically in obese patients; there is evidence to support this approach for laparoscopic appendectomy and cholecystectomy. However, these patients require an additional level of expertise in the operating theater, not only from the surgeon and anesthesiologist, but also from the equipment handlers and the assistants. Colon cases and other cases involving more complicated visceral dissection may be detrimental if performed laparoscopically.

Obese patients are at higher risk for wound infection. The worst-case scenario develops when an obese patient eviscerates in the postoperative period. Emergent management will require closure, but this may not be technically feasible. These patients may require leaving the abdomen “open” with packing, and the patient will generally be intubated and paralyzed until the abdominal contents can be reduced or sufficient granulation develops that allows the construct to stabilize.

CARDIOVASCULAR CONSIDERATIONS IN THE MORBIDLY OBESE

Cardiovascular diseases are common in obese individuals, and manifest as ischemic heart disease, hypertension, and cardiac failure. Cardiovascular disease is reported in 37% of adults with a body mass index (BMI) greater than 30 kg/m², 21% with a BMI of 25 to 30 kg/m², and only 10% in those with a BMI less than 25 kg/m². Obesity—defined as a BMI of greater than or equal to 30 kg/m²—has been observed to be an independent risk factor for the development of hypertension. The Framingham Heart Study suggests that 65% of the risk for hypertension in women and 78% of the risk in men can be related to obesity (14). Interestingly, mortality rates were reported to
be 3.9 times greater in the overweight group versus the normal-weight group participating in the Framingham study (15).

The relationship between the increase in blood pressure and the risk of cardiovascular disease is considered to be independent of other risk factors. The chances of myocardial infarction, heart failure, stroke, and kidney disease are all greater as a patient’s blood pressure increases (16). Obesity is also well recognized as a risk factor for ischemic heart disease. Many obese individuals also suffer from “metabolic syndrome,” which has a strong association as being a precursor in the development of diabetes, cardiovascular disease, and increased mortality rates from cardiovascular disorders. There is also a 5% increased risk of heart failure for men and 7% for women associated with each unit of increase in body mass (15).

### Preoperative Considerations

#### Pathophysiology

In morbidly obese patients, blood volume, cardiac output, systemic and pulmonary artery pressures, and left and right ventricular pressures are all elevated (17,18). These changes manifest clinically as arterial hypertension and, with advancing age, one may note ischemic heart disease and right-left heart failure (19). The incidence of pre-existing, often severe, cardiovascular disease in morbidly obese patients scheduled for elective bariatric surgery is reported to be as great as 20% (20,21). It is the complex interaction of hypertension, ischemic heart disease, and pulmonary hypertension that contribute to the development of global cardiac dysfunction and exacerbates congestive heart failure. This clinical situation is referred to as “obesity cardiomyopathy.”

### Arterial Hypertension

The pathogenesis of obesity-related hypertension is complex. There is a continuous relationship between body mass index and systolic/diastolic blood pressures (22,23). Blood pressure is normally regulated by a series of feedback loops (baroreceptors) and by the secretion of vasoactive hormones—renin, angiotensin, aldosterone, and catecholamines. A derangement in any of these feedback loops may lead to hypertension. Many factors act together to promote vasoconstriction, sodium retention, and volume overload in obesity, and are noted in Table 102.1 (24–28). In the long term, these changes cause glomerular injury, ultimately leading to glomerulosclerosis. A review of 7,000 renal biopsies between the years 1990 and 2000 showed a 10-fold increase in obesity-related glomerulopathy (glomerulomegaly and glomerulosclerosis). Prolonged obesity may lead to a gradual loss of nephron function that contributes to a pressure overload of the heart, as well as an expansion of extracellular and blood volume combining to create a volume overload. Other variables that may also lead to hypertension in obese patients include leptin, free fatty acids, and insulin, which stimulate sympathetic activity and vasoconstriction (24). Furthermore, obesity-induced insulin resistance and endothelial dysfunction may act as amplifiers of the vasoconstrictor response. Obstructive sleep apnea (OSA), which also is more prevalent in obese patients, leads to periods of apnea and hypoxia, triggering a chemoreceptor response, which causes sympathetic activation (24,30,31).

#### Ischemic Heart Disease

Obesity is a recognized risk factor for ischemic heart disease (19,32). The risk is proportional to the duration of obesity and distribution of fat. A habitually overweight individual is less likely to be at risk than individuals who exhibit continuous weight gain, and individuals with a central distribution of fat are more at risk than individuals with a peripheral distribution. Additionally, hypertension, diabetes, hypercholesterolemia, and increased levels of low-density lipoproteins (LDLs), which are common in obese patients, further increase the risk of coronary stenosis. Nevertheless, more than 40% of obese patients with angina do not have significant coronary artery disease (30,33,34). Angina would then be attributable to the oxygen supply/demand imbalance due to cardiac hypertrophy and other factors. In the morbidly obese, myocardial oxygen consumption is higher than in normal-weight adults. The ventricular cavity dimension is enlarged due to a chronically augmented preload. An enhanced sympathetic activity and subsequent arterial hypertension and/or increased heart rate promote higher wall tension and increased ventricular systolic stress. In addition, the ventricular wall is commonly hypertrophic (35–39). Patients suffering chronic hypoxemia (pickwickian syndrome, obesity hypoventilation syndrome, and

### TABLE 102.1

<table>
<thead>
<tr>
<th>Factors that Act Together to Promote Vascular Constriction, Sodium Retention, and Volume Overload in the Obese Patient</th>
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<tbody>
<tr>
<td>► Elevated glomerular filtration rate, an elevated renal blood flow, and exhibiting delayed urinary sodium excretion in response to the saline load</td>
</tr>
<tr>
<td>► Increased renal sympathetic nervous activity, which directly promotes tubular reabsorption of sodium at the proximal and distal tubules</td>
</tr>
<tr>
<td>► The renin-angiotensin-aldosterone system is activated, which contributes to sodium retention and an increase in extracellular volume, despite an elevated blood pressure</td>
</tr>
<tr>
<td>► Natriuretic peptide levels are low, both at basal levels and also in response to salt loading</td>
</tr>
<tr>
<td>► Hyperinsulinemia directly promotes the tubular absorption of sodium</td>
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Compensatory mechanisms in the obese for overcoming increased sodium reabsorption include renal vasodilation, an increased glomerular filtration rate, and a higher blood pressure.

Data from:

- Dillona GR, Kepp VU. Neural control of renal function. Physiol Rev. 1997;77:75–149.
obstructive sleep apnea syndrome) frequently develop secondary polycythemia and, subsequently, elevated blood viscosity. In these patients, due to the higher blood viscosity, the contractility is augmented, which consequently increases myocardial oxygen requirements (40).

It may be assumed that morbidly obese patients are at a higher risk of myocardial ischemia. In these subjects, reducing myocardial oxygen consumption and ensuring the maximum possible oxygen delivery to the heart must be a major therapeutic target.

Cardiac Failure
There is an increased risk of heart failure of 5% for men and 7% for women per each unit of increase in body mass (15). There is a linear relationship between body weight and cardiac weight gain attributed to concentric and eccentric hypertrophy. This event is secondary to pressure overload, which is due to arterial hypertension and possibly increased blood viscosity. In obese patients, circulating blood volume, plasma volume, and cardiac output increase proportionately with rising weight. For a patient with a fat mass of 50 kg, blood flow to the fat mass accounts for an extra cardiac output of 1.5 to 2.0 L/min, resulting in both ventricular enlargement and an increase in stroke volume. The hypertrophy that ensues subsequently contributes to a reduction in cardiac compliance and left ventricular diastolic function, which leads to increased left ventricular end-diastolic pressure and possible pulmonary edema (41).

In long-standing obesity, systolic function might be reduced if hypertrophy is unable to keep pace with the increasing demand. A decrease in midwall fiber shortening and a decrease in ejection fraction may thus become evident in developing “obesity cardiomyopathy.” The right ventricle can also exhibit hypertrophy secondary to pulmonary hypertension due to obstructive sleep apnea and subsequent chronic hypoxemia and hypercapnia.

Preoperative Evaluation and Optimization for Surgery
A significant percentage of obese patients who present for intermediate- to high-risk noncardiac surgery is likely to have cardiovascular disease. It is imperative to know if any related impairment actually exists. If so, assessment of its severity, decisions on whether or not any therapeutic measures can be taken prior to surgery, and consideration of any extra intraoperative or postoperative monitoring are of utmost importance for effective treatment of the obese patient.

Arterial Hypertension
The preoperative assessment, optimization, and treatment of arterial hypertension in the obese patient are guided by the same principles as in the nonobese patient. Urgent and emergent surgeries should be evaluated on a case-by-case basis, and aggressive control of blood pressure in the perioperative period is vital.

Acute hypertensive episodes and hypertensive emergencies should also be approached as they would be in the nonobese, but with the strong recommendation for meticulous and adequate monitoring. Careful drug titration is required due to the particular hemodynamic liability of this population, coupled with the fact that for obvious reasons, pharmacokinetics and dynamics might be altered.

In current clinical practice, measurement of blood pressure may be difficult, even to the point of deciding where to make the measurement. Obese individuals—especially women—tend to have a conical shape of the upper arm, termed “glycoidal obesity,” and accurate measurement of blood pressure is difficult with conventional cuffs. As an alternative, the cuff can be placed around the forearm for more predictable cuff pressures. An increasing arm circumference is associated with miscalculation of blood pressure if standard-length cuffs are used. An appropriate-sized cuff that encompasses at least 80% of the arm should be used to ensure accurate measurement of blood pressure in an obese patient (42). If these maneuvers fail to result in adequate and reliable measurements, invasive blood pressure monitoring should then be considered as an alternative.

Ischemic Heart Disease and Cardiac Failure
To date, no specific cardiac risk index has been proposed for obese patients. Preoperative cardiac assessment of obese patients follows the same sequence as for lean patients, and for that reason, the American College of Cardiology/American Heart Association (ACC/AHA) guidelines are thought to be valid for this population (43). However, the guidelines do not take into account the presence of multiple, intermediate, or minor risk factors that are frequently observed in the obese. The presence of multiple cardiac risk factors has been shown to increase the incidence of perioperative cardiac morbidity (44).

In addition, surgical risk categories may be modified based on institutional expertise, which is highly dependent on clinical experience, surgical skills, anesthetic care, and nursing quality (45,46). In this regard, there is evidence to suggest significant differences between care and outcomes at different institutions for the same surgical procedure (46–49). In addition to careful appraisal of arterial hypertension and its consequences, a comprehensive cardiac evaluation in obese patients should focus on assessing both cardiac function and the presence and severity of ischemic heart disease. Evaluation of cardiac function by clinical signs can be extremely difficult in the obese and, for that reason, objective evaluation of ejection fraction and cardiac function by echocardiography and/or left ventriculography is usually necessary.

The ACC/AHA recommendation for preoperative noninvasive evaluation of left ventricular function includes patients with current or poorly controlled heart failure, patients with a history of heart failure, and patients with dyspnea of unknown origin—an extremely frequent finding within this population. Despite the fact that no randomized study has been performed in obese patients to determine the utility of routine evaluation of right and left ventricular function, it is very probable that such evaluation, guided by symptoms prior to intermediate- and high-risk surgery, will be helpful in guiding intraoperative and postoperative management of obese patients.

Evaluation for ischemic heart disease requires stress testing, although there is no consensus on which type of stress test is optimal. The choice among the various noninvasive tests must be made based on local preferences. The patient’s weight must be taken into account not only for logistical reasons—as many diagnostic devices have significant weight limitations—but also because the effect of the patient’s body habitus has
Optimized medical management (typically with revascularization by percutaneous coronary intervention or surgery (coronary artery bypass grafting [CABG]). Patients who have positive stress test results will require coronary angiography. If cardiac catheterization is indicated, patient weight has to be taken into account because some tables used to perform this study have weight limits as low as 300 lbs. Despite the concerns raised by the size of the patients, cardiac catheterization is considered safe in these patients. Fifty-five percent of the time, cardiac catheterization results in negative findings; when positive, medical management, interventional cardiology therapy, or even cardiac bypass surgery may be indicated (52).

Once ischemic heart disease has been identified and its severity quantified in the morbidly obese patient, three therapeutic options are available prior to elective noncardiac surgery:

- Revascularization by surgery (coronary artery bypass grafting [CABG])
- Revascularization by percutaneous coronary intervention (PCI)
- Optimized medical management (typically with β-blockers or α2-agonists)

There is no irrefutable evidence that indications for preoperative cardiac revascularization are any different for obese patients than for nonobese patients.

Coronary Artery Bypass Grafting. Still a controversial topic, some studies suggest that moderately and morbidly obese patients have a higher rate of deep sternal wound infection, renal failure, prolonged postoperative hospital stay, and operative mortality after coronary artery bypass surgery (53–55). Coronary revascularization is guided by the patient’s cardiac condition—that is, is there unstable angina, left main coronary artery disease (CAD), three-vessel disease, decreased left ventricular (LV) function, and/or left anterior descending artery disease—as well as by the added risk of the coronary intervention and the potential consequences of delaying the noncardiac surgery for recovery after the cardiac intervention (56). It has been demonstrated that when indicated, patients undergoing coronary revascularization prior to major-risk noncardiac surgery did better postoperatively. Comparing this population of preoperatively revascularized patients with those medically managed suggests that the latter patient group had a mortality rate two times higher than the former (57,58).

Percutaneous Coronary Intervention. Evidence suggests that patients who underwent angioplasty prior to elective noncardiac surgery had better outcomes (59–62). However, angioplasty is now often accompanied by stenting, with postprocedural ant platelet therapy to prevent acute coronary thrombosis and maintain long-term patency of the intervened vessel. It is strongly suggested that elective noncardiac surgery should be delayed for 4 to 6 weeks after PCI with stenting to allow for complete endothelialization of the stent and completion of aggressive ant platelet therapy with glycoprotein (GP) IIb/IIIa inhibitors (63).

The introduction of drug-eluting stents may obviate the need for such prolonged systemic anticoagulation, thus allowing patients to undergo noncardiac surgery sooner. The complication rate of PCI in obese patients has not been reported to be different than in nonobese individuals, and similar precautions should also be taken in morbidly obese patients (64).

Medical Management. Perioperative use of β-blockers has been shown to be efficacious in reducing perioperative morbidity and mortality (65–68). The ACC/AHA guidelines recommend initiating β-blockers as early as possible prior to high-risk surgery and titrating the patient’s heart rate to 60 bpm (43). Perioperative β-blocker use is recommended for patients with one or more Revised Cardiac Index risk factors despite a negative stress test and for patients with two minor risk factors, even with a good functional status and/or a negative noninvasive stress test (43,67).

Many morbidly obese patients are already receiving β-blocker therapy when they present for their preoperative assessment. β-Blockers have been used intraoperatively to control hemodynamics, intraoperative ischemia, and cardiac arrhythmias (65). Some studies investigating their prophylactic role have demonstrated decreased intraoperative ischemia (69).

The general consensus appears to be that if β-blockers are indicated perioperatively, they should be given not only intraoperatively but, more appropriately, they should be initiated during the preoperative period and—except in the presence of significant contraindications—should be continued through the postoperative period. The pharmacokinetics of β-blockers are affected by obesity, and there exists significant pharmacodynamic variability. The dosage of β-blockers should be initiated based on lean body mass and then titrated until the desired clinical effect is achieved (70,71).

Intraoperative Considerations

Mechanics

Surgical beds are now available that accommodate patients weighing as much as 500 kg. However, these patients require tremendous preparation on behalf of the operative staff. Even with appropriate beds, the obese patient can be at high risk for falling during sudden motion. This situation can be extremely dangerous to the patient and the supporting staff alike. Institution of a lift team and the availability of both “bean” bags and bed extensions in order to keep the folds of pannus stable are of the utmost importance to the surgical team, and have already become widely used in accredited surgical facilities. Finally, institutional investments in lifting equipment such as ceiling-mounted lifts and beds that oscillate and/or transform into chairs may be necessary to fully deploy the necessary mechanical advantage to care for the obese patient.

Rhabdomyolysis

Rhabdomyolysis is often described in the obese patient. This scenario generally follows prolonged operations and presents as dark urine, representing muscle necrosis from groups on the flanks or buttock. Also termed pressure-induced myoglobinuria, this is more commonly noted among patients with diabetes mellitus (72,73). It is generally attributed to lying on a hard surface, and has been frequently associated with an...
TABLE 102.2

MOST FREQUENT AND PROMINENT RISKS OF THE MORBIDLY OBESE PATIENT UNDERGOING SURGERY

- Pulmonary aspiration of gastric contents
- Difficult mask ventilation and tracheal intubation
- Rapid development of hypoxemia after apnea
- Pulmonary atelectasis
- Hemodynamic instability
- Decreased ability to deal with the physiologic responses to stressful situations (i.e., hyperglycemia, hypertension, cardiac failure, arrhythmias, and myocardial ischemia)
- Delayed recovery
- Postoperative respiratory dysfunction
- Deep venous thrombosis

The classic, most frequent and prominent risks of the morbidly obese patient undergoing surgery are listed in Table 102.2. What must be absolutely clear are the pathophysiologic alterations and subsequent risks presented in each individual case. This should guide the physician to define the intraoperative goals. Considering only the potential impairments of the cardiovascular and respiratory systems, we must highlight the potential risks and recommended anesthetic goals.

Exaggerated lithotomy position in the operating theater (74). To handle this situation, attending staff should initiate aggressive hydration and monitor creatine phosphokinase (CPK). If CPK exceeds 5,000 IU/L, staff must initiate diuresis with mannitol and alkalinate the urine with sodium bicarbonate (75,76). Acute renal failure may develop, but recovery of renal function is generally expected.

Postoperative Considerations

Logistic and Technical Issues

The main diagnostic and therapeutic principles related to cardiovascular diseases and/or complications commonly observed in the ICU setting—such as arrhythmias, cardiac failure, hyper-

tensive or ischemic episodes, and so forth—do not differ significantly when comparing the morbidly obese with lean patients. Therefore, a detailed discussion is not warranted. Nevertheless, we will highlight the few—but in our eyes important—differences to consider when presented with a morbidly obese surgical patient in the ICU setting.

Pathophysiologic Principles for a Rational Therapeutic Approach

Obesity has been likened to “exercise,” that is, a constant state of “exercise.” The morbidly obese patient’s cardiovascular system is continuously overdemanded, even at rest, mainly because of chronic intravascular volume overload, blood viscosity, and sympathetic hyperactivity. These are components of a “dysfunctional compensating mechanism,” which tries to satisfy the increased metabolic rate imposed by the excessive adipose tissue. The resulting eccentric left ventricular hypertrophy (LVH) is associated with a reduced LV compliance, causing elevation of LV filling pressure in many morbidly obese persons (18,82).

The additional increase in cardiac output promoted by any perioperative stress may markedly increase LV filling pressure, often exceeding the threshold for pulmonary edema. Respiratory disease, especially obstructive sleep apnea syndrome (OSAS) and the obesity hypoventilation syndrome (OHS), acting on the pulmonary circulation may affect the right heart cavities as well. The heart of a morbidly obese patient may have less tolerance to any kind of cardiovascular stress—hypovolemia, hypervolemia, hypertension, hypotension, and so forth—and is at a higher risk of organ failure. Consequently, the appropriate diagnostic and therapeutic measures should be applied as soon and as accurately as possible to avoid systemic hypoperfusion and inadequate oxygen delivery, which may predispose to multiple organ system failure.

Importance of Coupled Cardiorespiratory Function

It is vital to maintain the best possible ventilation/perfusion (V/Q) balance, since V/Q mismatch is a prominent mechanism that can trigger respiratory and subsequent cardiac dysfunction in the morbidly obese surgical patient. In mechanically ventilated, morbidly obese patients, airway pressure may be elevated. Additionally, morbid obesity is associated with volume and pressure overload. Volume load conditions may fluctuate according to patient positioning. For example, changing position from the “physiologically ideal” reverse Trendelenburg to the supine position can significantly increase venous blood return to the heart and, as a result, augment cardiac output, pulmonary capillary wedge pressure, and mean pulmonary artery pressure, potentially increasing the risk of acute heart failure (83); one would expect this maneuver to increase airway pressure as well due to the increased weight of the chest.

Compression of the inferior vena cava may reduce venous return to the heart, and is thus a possible mechanism of hypotension. This can be avoided by tilting the operating room table or ICU bed by placing a wedge under the patient. These maneuvers are similar to those performed during caesarean section to reduce the pressure of the gravid uterus on the inferior vena cava (84). Considering that the reverse Trendelenburg position significantly improves cardiac and respiratory performance, it should be maintained during the entire perioperative period unless there is a particular contraindication.
Drug Dosing
The distribution, metabolism, protein binding, and clearance of many drugs are altered by the physiologic changes asso-
ciated with obesity (85–87). In addition, the patient’s under-
lying disease may substantially influence the pharmacokinetic properties of a drug (88). The net pharmacologic alteration in any patient is, therefore, often uncertain, especially in those suffering from morbid obesity. Nevertheless, for a number of drugs used in the ICU—most notably digoxin, aminophylline, aminoglycosides, and cyclosporine—drug toxicity may occur if the patients are dosed based on their actual, rather than ideal or adjusted, body weight (70,85,87,89–91). For drug dosing, with few exceptions, it is advisable to base drug calculations on ideal body weight (IBW) rather than real weight, and then adjust doses through meticulous monitoring (92).

RESPIRATORY CONSIDERATIONS IN THE MORBIDLY OBESE

The higher morbidity and mortality of hospitalized obese pa-
tients may be related to the increased pulmonary complications with which morbidly obese patients present (93). In the postop-
erative state, obese individuals are at increased risk of develop-
ing atelectasis, aspiration, ventilatory failure, and pulmonary embolism (93).

Clinicians caring for morbidly obese patients must be aware of the significant physiologic changes associated with their obe-
sity, such as reduced lung volumes, increased work of breath-
ing, and alterations in control of breathing and gas exchange.
Many factors are involved including, but not limited to, BMI, patient’s age, duration of obesity, fat distribution (central or pe-
eripheral), and the strong association of certain disorders such as OSAS, OHS, and pickwickian syndrome. In addition, obe-
sity itself has a major detrimental impact on the respiratory system (94).

Preoperative Considerations

Respiratory Disorders

Obstructive Sleep Apnea Syndrome. Morbid obesity is the most common and major risk factor for OSAS (95). While its prevalence in the general U.S. population is 2% to 4%, this increases to 40% to 78% in the morbidly obese (95–99). Notwithstanding these facts, it is thought that 80% to 90% of American sleep apnea sufferers are undiagnosed (100,101).

The detection of OSAS among obese surgical patients is vital for several reasons:

- Obese patients are more sensitive to the depressant effects of narcotics and opioids (102). Perioperative administra-
tion may lead to life-threatening respiratory complications (103,104), especially in face of pre-existing OSAS.
- OSAS is associated with difficult laryngoscopy and mask ventilation (104–107).
- Obese patients have, in general, a diminished expiratory reserve volume (ERV) with, consequently, reduced oxygen stores; this promotes faster development of desaturation af-
ter apex (108).

If OSAS is present, these effects become exaggerated. The combination of these factors sets the stage for an airway cata-
trophe, not only during induction of general anesthesia, but also during tracheal extubation, and especially if an emergent intubation becomes necessary in the ICU or during intra- or interhospital transfer.

Obesity Hypoventilation Syndrome. Some obese patients suf-
fer from a disorder characterized by chronic daytime hypoventi-
tilation, also known as obesity hypoventilation syndrome (109). These individuals are typically extremely obese, with a BMI greater than 40 kg/m², and the likelihood of OHS in-
creases as the BMI increases. OHS is associated with chronic daytime hypoxemia—with a PaO₂ less than 65 mm Hg—and hypercapnia (107,108,110). It is essential to find out if the obese patient suffers from chronic daytime hypoxemia, as this is a better predictor of pulmonary hypertension and cor pul-
malone than the presence and/or severity of OAS (111–113).

Pickwickian Syndrome. Patients suffering OHS who addition-
ally have signs and symptoms of cor pulmonale are termed pickwickian—from the Charles Dickens novel, The Pickwick Papers—and they have an increased perioperative morbidity and mortality (93).

Respiratory Insufficiency. Obesity per se is not a common cause for chronic respiratory insufficiency (109). Significant respiratory dysfunction is more common when chronic ob-
structive pulmonary disease (COPD) and obesity coexist. When respiratory insufficiency is present, impairment of gas exchange is greater than expected from a simple summation of the alter-
cations caused by each pathophysiologic process (114).

Simple Obesity

Obese patients with minimal or no coexisting pulmonary con-
ditions are classified as “simple” obesity patients. The patho-
physiology of simple obesity consists of alterations in daytime gas exchange and pulmonary function, and may result from compression and restriction of the chest wall and diaphragm by excess adipose tissue (115). The ERV and functional resid-
ual capacity (FRC) are particularly affected, being reduced to 60% to 80% of normal, respectively.

If ERV decreases below the alveolar closing volume, then airway closure occurs during normal tidal breathing, and de-
pendent alveoli are relatively or completely underventilated. As a consequence, V/Q mismatch, pulmonary shunt, and daytime hypoxemia results. One may see, in formerly obese patients after massive weight loss, a marked improvement in the PaO₂ and alveolar–arterial oxygen gradient; thus, this improvement is directly proportional to the increase in the ERV (116,117).

Other mechanisms may further impair respiratory function. Sleep apnea in the obese is usually obstructive, secondary to air-
way narrowing from abundant peripharyngeal adipose tissue, and an abnormal decrease of upper airway muscle tone during rapid eye movement (REM) sleep (93). Hypopneic and apneic events lead to arousal from REM sleep, oxyhemoglobin desat-
uration, and symptomatic nervous system activation in response to hypoxemia. This may explain the strong association between OSA and systemic hypertension (118). The precise pathophys-
ologic mechanism of OHS is unclear (93,109).

Of most importance is that vital capacity, reduced to 90% of normal in simple obesity, decreases to 60% of normal in
OHS. This reflects a profound and important decrement in lung volumes in OHS, as compared with simple obesity patients. Thus, one may see

- A marked increase in distal airway resistance
- A more profound abnormality in V/Q matching
- A more significant impact on the PaO₂
- A larger A/a gradient in patients with OHS

Supine positioning further reduces lung volume and, as a result, increases the magnitude of all these alterations (107,115,119).

Diaphragmatic function is also affected due to overstretching and cephalic displacement resulting from increased intraabdominal pressure. All of these factors combined may lead to chronic respiratory muscle fatigue and the chronic hypventilation characteristic of OHS (93).

Venous Thromboembolism

Perioperative venous thromboembolism (VTE) occurs in 0.2% to 2.4% of bariatric patients receiving thromboprophylaxis (120). According to the Chest Consensus Statement, obese patients in the ICU will generally fall into either the high or highest risk categories in which, if left untreated, the risk of deep venous thrombosis (DVT) ranges from 20% to 80%. The risk of clinical pulmonary embolism (PE) ranges from 2% to 10%, and fatal PE occurs in 0.4% to 5% of patients (121).

The obese population in the surgical ICU requires thromboprophylaxis; however, the best regimen is not clear. Multiple variables are worthy of mention in regard to this matter. Venous stasis ulcers are more common in the obese, and in turn, are associated with DVT. Prophylactic inferior vena cava filters can be considered, but may also be technically difficult in the heaviest patients. Sequential compression devices (SCDs) are generally recognized as a useful adjunct but, again, may be limited by the patient size. The adequacy of pedal pumps is not clear. Unfractionated or low-molecular-weight heparins are both viable options, though precise dosing regimens and duration of dosage have emerged largely from uncontrolled trials. There are reports in the bariatric literature that 40 mg of enoxaparin every 12 hours may provide better thromboprophylaxis than 30 mg every 12 hours; however, this recommendation came from a retrospective report that coincided because of chronic daytime hypoxemia—PaO₂ less than 65 mm Hg, but especially sustained hypercapnia—a PaCO₂ greater than 45 mm Hg—in the morbidly obese patient without significant obstructive pulmonary disease is diagnostic for this syndrome.

One must differentiate whether morbid obesity coexists with either OHS or COPD. These combinations often result in chronic daytime hypoxemia and increase the chances for pulmonary hypertension, right ventricular hypertrophy, and/or right ventricular failure. Assessment of these pickwickian patients may require extensive testing to guide preoperative medical optimization and postoperative management, given that their morbidity and mortality rate is increased (93,102,126).

It is unclear if it is appropriate to delay bariatric surgery for aggressive optimization of airway status and oxygenation with continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BiPAP) therapy. Rennotte et al. observed no major postoperative respiratory complications in 14 patients treated with nasal CPAP for up to 3 weeks prior to surgery (127). Two to three weeks may be necessary not only to maximize medical benefits, but also to allow sufficient time for patients unfamiliar with CPAP or BiPAP therapy to acclimate to the nocturnal use of the device. Three weeks of nightly CPAP treatment prior to bariatric surgery improved left ventricular ejection fraction and afterload in patients with concomitant heart failure (128). Eight weeks of preoperative nasal CPAP therapy may be required to treat hypertension secondary to OSA (129).

Intraoperative Considerations

Airway Management

It is still a debatable issue whether or not morbid obesity should be considered a risk factor for difficult airway management. Brodsky et al. observed that neither absolute obesity nor BMI was associated with problematic intubation in morbidly obese patients. They also concluded that only a large neck circumference and a Mallampati score of 3 or more were significantly correlated with a high probability of problematic intubation (130). In our opinion, preoperative airway assessments should be similar in both morbidly obese and lean patients.

Whether or not morbid obesity is considered a risk factor for intubation, issues related specifically to the patient’s BMI that impact airway management include:

- Preoxygenation
- Positioning
- Immediate availability to adequate resources, both technical: special laryngoscopes, blades, tracheal tubes, orotracheal or nasal airways, cannulas, introducing laryngeal airway (LMA), LMA Fastrach, Combiblade, etc.; human: personnel sufficient in both number and expertise
In the perioperative setting, one never knows when an emergent tracheal intubation or reintubation may be necessary, or when an unpredicted difficult mask ventilation and difficult tracheal intubation combination may appear. Indeed, the latter is a common situation in morbidly obese patients suffering from OSAS or OHS. The presence of any of these urgent/emergent events—taking into account the reduced time to hypoxemia after apnea and possible increased risk for gastric aspiration in morbidly obese individuals—will likely result in a life-threatening, although preventable/treatable, respiratory misadventure.

In our opinion, the same American Society of Anesthesiologists algorithm for difficult airway management and indications for conscious tracheal intubation should be considered for both obese and lean patients. If conscious intubation is indicated, it must be remembered that most episodes of gastroesophageal reflux, and the greatest potential for pulmonary aspiration of gastric contents, occur from and during “backing” on an endotracheal tube; consequently, appropriate preparation of the patient is crucial ([131,132]).

Meticulous patient explanation, a low infusion rate of remifentanil (0.05 mcg/kg/minute) to avoid loss of response to verbally required active ventilation, and/or local anesthesia—such as bilateral blockade of the internal branch of the glossopharyngeal nerve—have shown to be the most effective and safest induction alternative ([77]). Finally, although somewhat controversial, it is prudent to treat morbidly obese individuals with prophylactic measures against gastric aspiration, such as cimetidine, ranitidine, Bicitra, and/or metoclopramide ([133]); the timing of administration of these agents is, of course, of great significance.

**Preoxygenation and Position**

Hypoxemia during induction of general anesthesia in obese individuals is, and must be, a real concern for the anesthesiologist and/or intensivist. These patients may experience rapid arterial oxygen desaturation after apnea ([134]). Compared with supine-positioned obese patients, preoxygenation in the 25- to 30-degree head-up position with 100% oxygen for 3 minutes achieves higher oxygen tensions—in other words, more time and better oxygenation for intubation and airway control, given that as BMI increases, it has been shown that the amount of time for desaturation in the patient decreases ([135]). Preoxygenation in the 30-degree reverse-Trendelenburg position provides a longer, safer apnea period than the 30-degree semi-Fowler and supine positions. Consequently, the Trendelenburg position has been recommended as the optimal position for induction of general anesthesia in obese patients ([136]). The head-up position results in an unloading of the intra-abdominal contents from the diaphragm; thus, pulmonary compliance and FRC increase, and oxygenation returns toward baseline values, as compared to the same patients who were placed in the supine position ([137]).

Prior to induction of general anesthesia in any setting—whether the operating room, the ICU, or on the hospital floor—the obese patient should be positioned with pillows under the shoulders, with the head and upper body elevated in a semirecumbent or reverse Trendelenburg position. This “ramped” position is strongly recommended in the morbidly obese, as it improves pulmonary function, oxygenation, cardiovascular function, conditions for mask ventilation, laryngoscopic view, and tracheal intubation ([138]). Extremely obese patients should never be allowed to lie completely flat. Their upper body should be constantly elevated at 25 to 30 degrees in the perioperative period.

**Atelectasis**

In the perioperative setting, reduction of chest wall and diaphragmatic muscle tone following the induction of general anesthesia and skeletal muscle relaxation impairs oxygenation. In simple obesity, the net effect may reduce ERV and FRC to less than 30% of preinduction values, excluding even more alveoli from effective gas exchange ([115]). As reduction of ERV and FRC increases exponentially with increasing BMI, the combination of these factors predisposes the morbidly obese patient to suffer atelectasis not only during anesthesia and surgery, but also in the postoperative period. The importance of this topic will be developed in the postoperative section.

**Mechanical Ventilation: Invasive Positive Pressure Ventilation**

Respiratory physiology must be taken into account when considering mechanical ventilation. Oxygen consumption and carbon dioxide production increase due to a higher metabolic rate promoted by excessive fat and an augmented workload on the respiratory system ([138]). Normocapnia is maintained by increased minute ventilation. Regarding mechanics, total compliance of the respiratory system declines exponentially with increasing BMI, as do FRC, ERV, and total lung capacity ([139]). Clinical correlates of these changes are increased work of breathing, small airway closure, ventilation/perfusion mismatch, pulmonary shunt, and hypoxemia. Sedation, anesthesia, and positioning supine further reduce FRC in the obese as compared to nonobese subjects, and consequently worsen respiratory performance ([140]). Considering these factors, the initial tidal volume should be based on ideal body weight rather than actual body weight, and adjustments made according to airway pressures and appropriate respiratory monitoring ([141]). As lung volumes are reduced and airway resistance is increased, a tidal volume based on the patients’ actual body weight would probably result in high airway pressures, alveolar overdistention, and barotrauma. Some data suggest, as a lung-protective strategy, the use of smaller tidal volumes and adequate positive end-expiratory pressure (PEEP)/CPAP ([142]) to prevent airway closure ([143]). Although this technique may result in decreased cardiac output, fluid loading will correct the problem. Additionally, in an attempt to improve ventilator-patient synchrony and reduce airway pressure, the patient’s spontaneous respiratory effort should be maintained and assisted with pressure support ventilation as needed ([144]).

**Tracheal Extubation and Intrahospital Transfer**

As a result of the increased work of breathing and impaired respiratory mechanics, morbid obesity has been associated with prolonged mechanical ventilation, extended weaning periods, and longer ICU and hospital lengths of stay ([6]). Strategies suggested for facilitating the weaning process include positioning of the patient in a 45-degree reverse Trendelenburg position—thus optimizing lung mechanics, increasing tidal volume, and reducing respiratory rate ([145])—and BiPAP post extubation ([146]).

If hemodynamic stability has been achieved, the trachea should be extubated with the patient’s upper body elevated.
between 30 and 45 degrees. The patient should be transferred from the operating room while in a semirecumbent or tilted reverse Trendelenburg position (147). As obese patients have greater reduction in lung volumes than normal-weight counterparts following abdominal surgery (115), it comes as no surprise that the recovering patient should be kept in a head-up position in order to minimize intrapulmonary shunting (148). On days 1 and 2 postoperatively, a change from the semirecumbent to the supine position may result in significant decreases in PaO₂. Consequently, obese patients should convalesce in the semirecumbent position while receiving supplemental oxygen (148). If at all possible, intrahospital transfer of a morbidly obese patient is best and most safely accomplished if the patient remains in his or her own hospital bed.

**Ideal FiO₂ (Supplemental Oxygen)**

It should go without saying that one uses the highest concentration of oxygen necessary to maintain life. Nonetheless, high oxygen concentrations have often been associated with atelectasis formation and recurrence (149). In order to avoid this consequence, using as low an oxygen concentration as possible has been recommended. When 100% oxygen is delivered, shunt increases significantly due to atelectasis development, while with 30% oxygen delivery, shunt and atelectasis are minimal (150). Finally, without any preoxygenation, no atelectasis develops after induction (151,152), although there may be other problems unrelated to atelectasis.

Nevertheless, supplemental oxygen carries clear benefits for patients, especially the morbidly obese. There is evidence that suggests an FiO₂ of 0.8 ensures appropriate oxygenation without increasing the risk of absorptive atelectasis, reduces the incidence of postoperative nausea and vomiting (PONV) in patients with an increased risk of gastric aspiration, and improves the host's defense mechanisms against infection. The improvement can be seen not only in the wound site, but also in the respiratory system (153–156). Although not proven in morbidly obese surgical patients, this possible benefit should not be ignored. Ideal FiO₂ should, then, result from a balance between a supplemental quantity of oxygen that is sufficient enough to avoid hypoxemia, reduce postoperative infections, and reduce PONV, but not so high as to facilitate the development and maintenance of atelectasis.

Our recommendation is to deliver 100% oxygen before induction of anesthesia to retard the development of hypoxemia after apnea and, once tracheal intubation is confirmed, reduce the FiO₂ to 0.8, if possible, according to respiratory monitoring.

**Monitoring**

FRC is reduced in the morbidly obese patient; if it drops below closing capacity (CC), the dependent small airways will collapse, promoting:

- Ventilation/perfusion mismatch
- Gas exchange deterioration
- An increase in the shunt fraction
- An increase in the alveolar-arterial oxygen gradient

Consequently, the more obese the patient—the greater will be the alveolar-arterial gas difference, in other words, the less the expiratory gas measurements will correlate with arterial blood gas analysis.

A morbidly obese patient will not be able to reach the same PaO₂/FiO₂ ratio as the normal-weight patient, even if higher inspiratory oxygen concentrations are delivered. Morbid obesity decreases the arterial oxygenation index even further, yet leaves PaCO₂ values unaffected if the patient does not suffer from either OHS or pickwickian syndrome (115); this effect is mainly due to intrapulmonary shunts in the atelectatic-dependent lung areas. In this scenario, arterial blood gas analysis becomes increasingly important because blood gases reflect the respiratory status more accurately than respiratory gas measurements. This does not mean that morbidly obese patients routinely require invasive or special monitoring of respiration (157), but morbid obesity, the presence of comorbidities, and the type of surgery, among other factors, should influence the decision of which monitoring devices need be used. Routine noninvasive monitoring will be sufficient in simple obesity cases, while the presence of OSA, OHS, pickwickian syndrome, daytime hypoxemia, and/or associated COPD should alert the anesthesiologist or intensivist to modify not only the intra- and postoperative respiratory monitoring, but also narcotic use (158). It is a good practice to obtain pulse oximetry or even arterial blood gas analysis values in the awake obese patient prior to any anesthetic premedication in order to obtain a reference reading, and thereby allow a comparison of preoperative values with intra- and postoperative values.

Anesthesia and controlled mechanical ventilation will almost always have a negative impact on oxygenation and alveolar ventilation. In surgeries where large fluid shifts occur, long intraoperative hypotensive episodes are possible, and satisfactory tissue oxygenation cannot be assessed by pulse oximetry or PaO₂ alone. In these cases, decreasing pH values, abnormal anion-glycemic gap or lactate values may be indicative of inadequate oxygen delivery (159). Inadequate oxygen delivery may be reflected in increased, and sometimes “unexplained,” postoperative complications. The degree and duration of postoperative surveillance depend on the surgical intervention, the course of anesthesia, and the patient’s condition. Monitoring should at least include pulse oximetry, respiratory rate, cardiac rhythm monitoring, and blood pressure measurement in the immediate postoperative period. In patients with decreasing oxygen saturations, ABG analysis and chest radiographs may be useful in sorting out the differential diagnosis. Sudden onset of respiratory distress, chest pain, and dyspnea may be indicative not only of a cardiac event, but also of pulmonary embolism; most mortality in the 30-day postoperative period after bariatric surgery is due to pulmonary embolism (160).

Obese patients have increased risk of respiratory-related complications in the postoperative period. In one study, the overall rate of critical respiratory events in obese patients was 3% (8). Interestingly, however, another study showed no significant increase of adverse perioperative events, even in patients with confirmed OSAS when the levels of wakefulness were carefully maintained (161). This reflects the importance of the anesthetic and analgesic management on the speed and quality of recovery of central nervous system (CNS) function. It is of utmost importance to take this into account when considering the anesthetic/analgesic strategy. Combined thoracic epidural/general anesthesia techniques may be quite suitable in these major cases.

Patients with confirmed or suspected OSAS, OHS, and pickwickian syndrome require more stringent observation. In the immediate postoperative period, this may warrant prolonged surveillance...
in the postanesthesia care unit or even admission to the ICU in selected cases, especially in those with surgery lasting for more than 4 hours and in patients with critical comorbidities. The main reasons for ICU admission in morbidly obese patients are disturbances in pulmonary gas exchange, which can be prevented by more prolonged one-on-one surveillance combined with meticulous medical care (162).

Postoperative Considerations

Hypoxemia and Associated Postoperative Respiratory Disorders

Following major open abdominal surgery without postoperative oxygen supplementation, even normal patients experience hypoxemia (SpO2 less than 90%) (163). On the first postoperative day following open bariatric surgery, 75% of morbidly obese patients had a PaO2 less than 60 mm Hg, which usually persisted and worsened in the following days (12). Many clinical processes may be suggested to explain this phenomenon. The most frequently observed is atelectasis, but pulmonary aspiration of oral or gastric secretions, pneumonia, acute lung injury, and acute respiratory distress syndrome (ARDS) should also be considered as possible and relatively common respiratory complications of postsurgical morbidly obese patients.

Finally, it is important to remember tracheal tube displacement as a mechanism of perioperative hypoxemia. Abdominal insufflation, as well as changes in operating room table position—usually to the Trendelenburg position—can cause cephalad movement of the diaphragm, and can lead to migration of an initially correctly positioned endotracheal tube (164,165). This phenomenon in morbidly obese patients undergoing laparoscopy can result in right endobronchial intubation and intraoperative hypoxemia. This mechanism should be considered in the intubated ICU patient because of the frequent, necessary changes in the patient’s position during care (166).

Atelectasis

General anesthesia may impair pulmonary gas exchange, and consequently decrease oxygenation in the general population; atelectasis is a major cause of this kind of impairment (167–170). Alterations in respiratory mechanics induced by general anesthesia, such as decreased chest wall and lung compliance, and a reduction in functional residual capacity promote atelectasis in nonobese patients. Conscious morbidly obese patients already have prominent alterations of their respiratory mechanics (171), and these patients are, in fact, particularly prone to intra- and postoperative atelectasis. During general anesthesia, as well as during the immediate postoperative period, morbidly obese patients are more likely to have significant impairment of pulmonary gas exchange and respiratory mechanics (115,172,173). Thus, it has been noted, even before the induction of anesthesia, that morbidly obese patients had more atelectasis, expressed in the percentage of the total lung area, than nonobese patients. After tracheal decannulation, atelectasis increased in both groups, but remained significantly more severe in the morbidly obese. In the final 24 hours postoperatively, a complete re-expansion of the lung parenchyma occurred in nonobese patients, while the amount of atelectasis remained unchanged in the morbidly obese (94).

The increased atelectasis found in morbidly obese patients explains, at least partially, postoperative pulmonary complications. Various mechanisms have been suggested for the development of atelectasis in the morbidly obese, such as lung parenchyma compression, absorption of alveolar gas in completely or partially collapsed airways, and alterations in surfactant production, function, and/or distribution (174). Our conclusion is that all possible measures to prevent or reduce the severity and duration of atelectasis in this patient population are vital and are listed in Table 102.3.

While some bariatric groups use noninvasive positive pressure ventilation (NIPPV) routinely in the postoperative care of morbidly obese patients immediately after extubation, others are reluctant because of the fear of anastomotic disruption; there are no data to support this concern. Commonly, morbidly obese patients use some form of NIPPV (CPAP or BiPAP) chronically for the treatment of OSA. Postoperatively, morbidly obese patients are at risk for prolonged depressant effects of the drugs administered during surgery. This situation may promote airway collapse not only in those morbidly obese patients with diagnosed OSA and already under preoperative treatment, but also in previously undiagnosed morbidly obese patients (175,176). Airway collapse is most frequent during REM sleep, which is brief in the initial postoperative period, but significantly longer on the third to fifth postoperative nights. The risk for airway collapse increases even days after surgery. This means that oximetric monitoring and supplemental oxygen must continue to be administered during this dangerous period (177).

### Table 102.3

**VITAL MEASURES TO PREVENT OR REDUCE THE SEVERITY AND DURATION OF ATELECTASIS IN THE OBSESE PATIENT**

- Place patients in the semirecumbent position and, if possible, out of bed in a chair as tolerated, as this maneuver may increase functional residual capacity.
- Provide effective analgesia, which will allow early and effective mobilization, cough, and excellent tolerance to physiotherapy.
- Institute aggressive incentive spirometry.
- During the first 3 postoperative days, deliver humidified “supplemental oxygen,” but avoid inspired fraction of oxygen higher than 0.8. Supplemental humidified oxygen will not reduce atelectasis, but will facilitate respiratory secretion clearance, and will prevent hypoxic episodes in efforts to improve the host’s defenses against bacterial infections.
- During surgery or postoperatively in intubated patients, instituting positive end-expiratory pressure is probably effective in increasing functional residual capacity via recruitment of atelectatic regions of the lung. Applying vital capacity maneuvers (also known as recruitment maneuvers) may also reduce the incidence and/or severity of atelectasis while improving the quality and effective time of alveoli recruitment.
- Noninvasive positive pressure ventilation can be used to avoid intubation in selected patients.

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A prospective study of 1,067 bariatric patients evaluated the risk of developing anastomotic leaks and pulmonary complications after gastric bypass. Of the 1,067 patients, 420 had OSAS and 159 were dependent on CPAP. There were 15 major anastomotic leaks, two of which occurred in CPAP-treated patients. No correlation between CPAP utilization and incidence of major anastomotic leakage was demonstrated. No episodes of pneumonia were diagnosed in either group. Based on this study, the conclusion was that CPAP is a useful modality for treating hyperventilation after gastric bypass surgery without increasing the risk of developing postoperative anastomotic leaks (178).

Regarding BiPAP, it appears that when used prophylactically during the first 24 hours postoperatively, it significantly reduces pulmonary dysfunction after gastrectomy in morbidly obese patients and accelerates the re-establishment of preoperative pulmonary function (146).

Pulmonary Aspiration

Even though many mechanisms were classically associated with an increased risk of gastric content aspiration in the morbidly obese, this topic remains controversial. While it is still recommended to take precautions against acid aspiration, massive pulmonary aspiration in morbidly obese patients is a rare event in current anesthesia practice, and the occurrence of uninitiated “microaspirations” in the postoperative period is difficult to assess because of the diagnostic problems observed within this population (144). While massive aspiration is uncommon, the following cautionary list should be carefully noted:

- While in bed, the patient must be adequately positioned in a semirecumbent or reverse Trendelenburg position at all times.
- The care team must be ready for bag-valve-mask ventilation and tracheal intubation in a ramped position, as well as have technical and adequate human resources.
- Drug dosing must be meticulously titrated according to monitoring parameters and clinical response, and based on the IBW.
- Consider the importance of a sufficient and safe analgesic regimen. In the patient with the therapeutic measures required for an expedited recovery.

Radiographic Evaluation and Complications

Radiographic evaluation of the surgically obese patient is complicated and made difficult by the weight limitations of modern scanners and the patient’s inability to cooperate with the transfer. Several radiographic tests, including the upper gastrointestinal series, may require the patient to stand for extended periods of time. Although tomographic tables now routinely handle patients weighing 400 lb (182 kg), these weight limits vary by institutional device. Surgeons and caregivers that are in a position to affect patient selection should consider this when planning operative interventions for obese patients. Further consideration for tables that handle heavier patients should be entertained when new equipment is being purchased. In the absence of excellent radiographic capabilities, these patients may require surgical exploration, and both patients and surgical team members must assume those additional risks.

Chapter 102: The Obese Surgical Patient in the Critical Care Unit

Venous, Arterial, and Nutritional Access

Venous access is difficult in this population. When peripheral access is inadequate, the point of choice may be the jugular vein. Gilbert et al. found this location to have fewer complications and to require fewer conversions to different locations (179). Arterial access is generally recommended as noninvasive blood pressure cuffs can give inaccurate measurements in this patient population. Nutritional access in critically ill obese patients is imperative; despite their weight, these patients can be relatively malnourished. While it is still recommended to use gastrostomy or jejunostomy can be placed. Although not impossible, achieving precarious gastric access can be extremely difficult, especially in a patient following a gastric bypass procedure. In these circumstances, it is a good time to reiterate the importance of communication between the surgical and critical care physicians and staff.

Analgesia

Overview. Acute pain can result in reduced tidal volume, vital capacity, functional residual capacity, and alveolar ventilation (181,182). These factors contribute to atelectasis, V/Q mismatch, hypoxemia, and hypercapnia. Pain-related muscle splinting interferes with the patient’s ability to cough, clear secretions, and efficiently participate in chest physiotherapy, all of which increase the chances for pulmonary complications (182). A major component of segmental and suprasegmental reflex responses is enhanced general sympathetic tone (183). Results of this tone are increased peripheral resistance, stroke volume, and heart rate, which lead to an increase in cardiac output. High blood pressure results in increased myocardial work and myocardial oxygen consumption (184). The rise in heart rate causes decreased diastolic filling time, possibly resulting in reduced oxygen delivery to the myocardium, with a risk of ischemia (185). All of these alterations could result in devastating respiratory and/or cardiovascular complications in at-risk individuals such as the morbidly obese, who commonly are at a higher risk of suffering variable degrees of impaired function affecting both systems.

Every health care provider knows that the efficacy of analgesia must be measured by the ability to cough and move without pain or discomfort, and not only by the absence of pain while in a resting state. All the potential consequences of poor pain control are serious problems in a general population, but they are of outstanding importance in the morbidly obese surgical patient. Early mobilization without discomfort should be considered a major anesthetic target in this population due to the fact that deep vein thrombosis and pulmonary embolism are some of the most frequent causes of mortality during the first 30 postoperative days. In addition, sufficient and safe postoperative pain control would result in a more effective and tolerable respiratory physiotherapy—a critical maneuver in this context—which would certainly reduce the possibilities for other respiratory complications such as atelectasis. Nevertheless, most morbidly obese patients with surgical pain do not receive adequate pain relief (184).
Analgesic Strategies (Intravenous, Thoracic Epidural, Multi-modal Approach). Unfortunately, uncertainty remains as to the superiority of one pain treatment method over another (185). Open versus laparoscopic surgical techniques, personal skills, and experience may influence the patient’s and anesthesiologist’s choice of pain treatment. Pain management strategies may offer specific advantages for specific patient outcomes, such as a reduced rate of pulmonary complications after abdominal surgery and superior pain control with thoracic epidural analgesia (TEA) (185–188). Postoperative epidural analgesia, using either local anesthetics or opioids, may be the route of choice for postoperative analgesia in morbidly obese patients, as it allows a more vigorous cough and chest physiotherapy, better diaphragmatic function, and fewer thromboembolic complications (11,189,190). TEA can be improved by adding opioids and possibly epinephrine to the epidural solution (191,192).

Thoracic epidural analgesia/analgesia may be particularly beneficial in the pathophysiologic context observed in morbid obesity. For example, left ventricular work conditions (both preload and afterload) may be improved by the sympathetic blockade, thus reducing the chances for developing heart failure (193,194). Myocardial oxygen balance may be improved as well due to a decrease in oxygen demand and augmented myocardial perfusion induced by coronary vasodilatation, both secondary to sympathetic block, thereby reducing the risk of ischemia (195–202), TEA does not affect chest wall compliance in the postoperative state, and allows for better diaphragmatic function when compared with general anesthesia alone (190,203–209). Alterations in chest wall compliance and diaphragmatic performance can be considered major determinants of postoperative respiratory dysfunction in most patients, but especially in the morbidly obese after upper open abdominopereineal procedures (182,210–219).

Regarding intravenous analgesia, improved efficacy and safety have been shown when patient-controlled anesthesia management includes adjunct analgesics such as nonsteroidal anti-inflammatory medications and local anesthetic wound infiltration in a multimodal approach (220,221). It must be remembered in the most effective way that a continuous and efficient analgesic scheme would certainly improve patient satisfaction and comfort, and very probably—even though still not proven in the morbidly obese—would reduce morbidity and mortality.

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Section X: Special Patient Population


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