Critical Care of the Morbidly Obese Patient

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Objectives

- Recognize the comorbid conditions that affect the critically ill, morbidly obese patient
- Discuss the special pulmonary challenges in caring for morbidly obese patients in the ICU
- Explain the influence of the inflammatory cascade on critical illness in the morbidly obese patient
- Summarize the unique aspects of the nutritional management of the critically ill morbidly obese patient
- Demonstrate the approach to drug dosing in the morbidly obese patient in the ICU
- Describe the extraordinary nursing care concerns of the critically ill patient who is morbidly obese in the ICU

Key words: morbidly obese patient; obstructive sleep apnea; obesity hypotension syndrome

For thousands of years obesity was rarely seen. It was not until the 20th century that obesity became common, so much so that in 1997 the World Health Organization (WHO) formally recognized obesity as a global epidemic. As of 2005 the WHO had estimated that at least 400 million adults (9.8%) were obese, with higher rates among women than men. The rate of obesity also increases with age, at least up to 50 or 60 years of age. Once considered a problem only of high-income countries, obesity is increasing worldwide. These increases have been felt most dramatically in urban settings. The only remaining region of the world where obesity is not common is sub-Saharan Africa.

The United States has some of the highest obesity rates in the developed world. From 1980 to 2002, obesity rates doubled, reaching the rate of 32% of the adult population. Rates of obesity vary by ethnicity and gender. In the United States, as of 2007, 33% of men and 35% of women were obese. The rates, however, were as high as 50% among African American women in 2005. The prevalence of class III obesity (body mass index ≥40) has increased the most dramatically, from 0.78% in 1990 to 2.2% in 2000. Although the overall rate of obesity began to plateau in the 2000s, severe obesity and obesity in children continue to increase.

Obesity is one of the leading health issues in US society, resulting in about 300,000 deaths per year in the United States. About 65% of Americans are now considered either overweight or obese. According to data from National Health and Nutrition Examination Study collected from the 1970s to 2004, the prevalence of overweight and obesity has increased steadily among all groups of Americans over the past 3 decades. Obesity in other countries presents different problems. Although the rates of obesity in Japan and Korea are far less than in the United States, comorbid conditions occur at lower weights so that obesity is a significant problem in these countries as well.

Nearly one-third of patients in ICUs meet the criteria for obesity, and up to 7% are morbidly obese. Although it may seem apparent that patients with obesity and morbid obesity have higher morbidity and mortality rates when critically ill compared with nonobese patients, and individual studies have indicated as much, this has not been borne out in all studies and meta-analyses, including the most recent one. Nevertheless, patients with morbid obesity present management challenges when they are in the critical care setting, from both the comorbidities that accompany their illness and the differences in management that they require. In the current obesity epidemic, every practitioner of critical care medicine needs to be aware of these challenges and the management strategies that accompany them.

Comorbid Conditions

Workers with morbid obesity have high incidences of type 2 diabetes mellitus (10% in men, 20% in women), coronary artery disease (14% men, 19% women), and hypertension (64%). The prevalence of these conditions in patients who are not able to work, who are more likely in a typical ICU population, is at least this and probably higher. Clinicians taking care of morbidly obese patients must be prepared for exacerbations of preexisting comorbid conditions as well as emergence of previously undiagnosed conditions that crop up in the course of a critical illness.

Diabetes Mellitus

Diabetes mellitus is the most critical of all the comorbid conditions, because diabetes places the patient at risk for all of its own comorbidities. The link on a cellular level has only recently been elucidated. Fat cells release a novel protein called pigment epithelium-derived factor, which triggers a chain of events and interactions that lead to development of type 2 diabetes. When pigment epithelium-derived factor is released into the bloodstream, it causes the muscle and liver to become desensitized to insulin. The pancreas then produces more insulin to counteract these negative effects. This insulin release causes the pancreas to become overworked, eventually slowing or stopping insulin release from the pancreas, leading to type 2 diabetes. Diabetes is
also involved, as seen later in this chapter, in the inflammatory changes accompanying morbid obesity.

Patients who are morbidly obese with preexisting diabetes mellitus are at risk for all the comorbidities of diabetes and disease states that accompany diabetes, such as diabetic nephropathy, poor wound healing, diabetic gastroparesis, and cardiac disease. Many patients with morbid obesity will present with an existing diagnosis of diabetes and will be at risk for the comorbidities; some patients will present without such a diagnosis, but the critical illness will unmask the disease process and the patient will show hyperglycemia during the ICU course. These patients, despite their new diagnosis of diabetes, can show remarkable resistance to exogenous insulin, requiring surprisingly high doses of insulin infusions: doses as high as 25 U/h are not unheard of.

**Cardiac Disorders**

Patients with morbid obesity can show a number of cardiac disorders that make management in the ICU more difficult. These patients have the previously mentioned increased incidence of coronary artery disease and hypertension. Patients with morbid obesity are frequently deconditioned from a cardiac standpoint and frequently have a resting tachycardia that can worsen during the course of critical illness. Patients who are morbidly obese and have obstructive sleep apnea (OSA) have an increased incidence of cor pulmonale and pulmonary hypertension that may make monitoring of central venous pressures less reliable, but rarely will OSA interfere with other management unless it is a known diagnosis prior to the illness. Finally, between tendencies toward diabetic nephropathy and hypertensive renal disease, patients with morbid obesity can show very little renal reserve and can show decreases in renal function following surprisingly little in the way of a renal insult.

**Pulmonary Concerns**

The body system that most frequently is affected by obesity is the pulmonary system. Such effects may be in the form of increased incidence of conditions such as asthma or OSA, difficulties managing the airway of a patient in pulmonary failure, or difficulties with ventilation and weaning a patient who is morbidly obese. The clinician caring for the morbidly obese must be aware of these pulmonary aspects, because they so frequently alter management.

**Pulmonary Mechanics and Mechanical Ventilation**

Patients with morbid obesity frequently have abnormal mechanical properties of the total respiratory system, with decreased compliance of both the lungs and the chest wall. This is likely due to increased blood pulmonary blood volume; increasing lung compliance caused by closure of dependent airways, and a small (if any) contribution from the simple mechanical effect of adipose tissue pressing on the thorax and increasing the effort needed to initiate a breath. Airway resistance is elevated because of reduced lung volumes, with a preserved ratio of forced expiratory volume in the first second of expiration (FEV₁) and forced vital capacity indicating that the resistance is present in the small airways and lung tissue rather than the large airways. Obese patients without obesity hypoventilation syndrome (OHS) have normal respiratory muscle strength, whereas patients with OHS are approximately 30% weaker, thought secondary to an overstretched diaphragm and possibly to fatty infiltration of the diaphragm in some cases. Spirometry frequently shows a decreased expiratory reserve volume and an increase in the FEV₁ to forced vital capacity ratio, with preserved vital capacity, functional residual capacity, total lung capacity, and maximum minute ventilation. The decreased expiratory reserve volume in the setting of a normal vital capacity indicates an increased inspiratory capacity. Patients with OHS will show more severe derangements of pulmonary abnormalities, with decreased total lung capacity, functional residual capacity, FEV₁, and maximum minute ventilation. Diffusion capacity is preserved in both sets of patients, and oxygenation is only mildly impaired and then mostly secondary to hypercapnea in patients with OHS.

There is no consensus on how to choose the settings for mechanical ventilation of the morbidly obese patient, other than it is incorrect to calculate tidal volumes based on actual body weight (ABW). Patients with morbid obesity have normal-appearing chest cavities on chest radiograph, and such radiographs clearly show that these patients have smaller lungs than would be expected based on their weight. A common suggestion is to start with a tidal volume based on ideal body weight (IBW) and then to adjust the settings based on blood gas analysis. Starting with higher levels of positive end-expiratory pressure to overcome basal atelectasis is usually extremely helpful.

The effects of morbid obesity on pure pulmonary outcome may be significant; one study showed that morbidly obese patients with similar Acute Physiology and Chronic Health Evaluation II scores had higher rates of nosocomial pneumonias, acute respiratory distress syndrome, and tracheostomy and a longer median length of mechanical ventilation. The combination of increased abdominal pressure, high volumes and low pH of gastric contents, high incidence of diabetes mellitus, and resultant gastroparesis puts these patients at higher than usual risk of aspiration of gastric contents. Positioning and prophylaxis against acid secretion, even in the absence of a history of gastroesophageal reflux disease, may be helpful, and prophylaxis against deep venous thrombosis is even more important than in nonobese patients.

**Airway**

At first glance, the airway of the morbidly obese patient will usually appear more difficult to intubate than that of a nonobese person. This is primarily due to the clinician’s impaired ability to view the pharynx through the patient’s open mouth, a measurement technique called the Mallem-pati classification. In nonobese patients, the degree to which you can see the posterior oropharynx and tonsillar pillars is
Obstructive Sleep Apnea

From the first descriptions of the Pickwickian syndrome by Burwell in 1956, it has been recognized that morbidly obese patients have problems with apnea-related syndromes. The Pickwickian syndrome is perhaps the most severe of these: this syndrome describes patients who are morbidly obese; have daytime hypersomnolence, dyspnea, plethora (from polycythemia), and cyanosis (from hypoxemia); have both hypoxemia and hypercapnea on arterial blood gases; and have signs of pulmonary hypertension and right ventricular failure. This all stems from the increased prevalence of OSA in patients with morbid obesity. A full discussion of OSA is beyond the scope of this text; an excellent review of OSA as it relates to obesity was provided by Koenig in 2001. Older estimates placed the incidence of OSA in patients with morbid obesity at 42% to 48% in men and 8% to 38% in women; these figures may severely underestimate prevalence, given that newer studies show an incidence of 60% in obese patients and nearly 70% in patients presenting for bariatric surgery.

Patients with OSA can present with chronic hypoxemia or periodic desaturations leading to a chronic polycythemia, chronic hypercapnia leading to pulmonary hypertension, cor pulmonale and right ventricular failure, systemic hypertension, and arrhythmias.

Management of the patient with OSA in the ICU will be affected by the OSA in some manner. Given the use of sedation and narcotics and changes in the patient’s mental status, the clinician can expect earlier and more severe worsening of respiratory status than otherwise expected. Oxygenation levels may take months to improve and may never “normalize,” so one may have to accept lower levels of oxygenation when extubated. Weaning protocols based on oxygenation and levels of CO₂ must be interpreted in light of the patient’s baseline. Mechanical ventilation of the patient with OSA must avoid hyperventilation and the resolution of the existing compensatory metabolic alkalosis.

The Role of Inflammation in Obesity

Recently interest has increased in the role of inflammation in morbid obesity. C-reactive protein, an acute phase reactant, has undergone intense investigation, and it has been found that the elevation of C-reactive protein corresponds to the level of adiposity present in the body. Numerous studies in obese subjects have shown high levels of tumor necrosis factor-α and interleukin-6 (IL-6), the levels of which decrease with weight loss and exercise. Insulin resistance is directly related to the degree of inflammation. The adipose tissue mass seems to dictate the degree of inflammation in a linear fashion. There is progressive infiltration of macrophages in adipose tissue that may be a major trigger or sustainer of inflammation. In obesity and severe sepsis, there is an increase in plasminogen activator inhibitor-1, which can cause a marked decrease in fibrinolysis, and an increase in tumor necrosis factor, which is either related to or the actual cause of an exaggerated insulin resistance in severe sepsis. Finally, there is an increased response of IL-6, the clinical meaning of which is unknown but undoubtedly is somehow related to the exaggerated inflammatory response that morbidly obese patients experience in severe sepsis. This exaggerated inflammatory response relates to the following:

• Adipose macrophage infiltration
• Insulin resistance
• Exaggerated IL-6 response to additional inflammatory stimuli
• Increased leukocyte adhesion
• Increased platelet adhesion
• Increased pulmonary microvascular permeability
• High resting oxidative milieu
• Genomic differences between obese and lean tissue

The clinical implications of this are very critical. It is clear that the inflammatory response in obese patients is...
very different from that in nonobese patients. There are anecdotal reports of increased response to drotrecogin alpha. Patients with morbid obesity, besides having decreased physical reserve from associated cardiac and pulmonary conditions, can deteriorate rapidly and need quick and decisive intervention. A “wait and see” approach is usually not an option because these patients will deteriorate in a vast storm of systemic inflammatory response syndrome that is tough to recover from. All treatments of a severely septic patient, such as antibiotic treatment, source control, and hemodynamic support, need to be instituted swiftly. Even if the patient has not been previously diagnosed with diabetes mellitus, clinicians should provide adequate glucose control and anticipate the need for high levels of insulin infusions.

**Nutritional Aspects**

Patients with morbid obesity in the ICU do not appear, at first glance, to present difficulties with nutrition, and discussions about when to feed and how much to feed sometimes can take on a demeaning tone among uncaring providers. In comparison to nonobese patients, morbidly obese patients in the ICU have the following characteristics:

- Higher glucose levels with more exaggerated response to stress
- Lower human growth hormone levels but normal response to stress
- Higher insulin levels with lower response to stress
- Higher cortisol levels with normal response to stress
- Higher norepinephrine and epinephrine levels with much lower response to stress
- Higher levels of ketones and free fatty acids.
- Normal resting energy expenditure with higher muscle and nitrogen loss in ICU

This means that despite huge fat stores, patients with morbid obesity can develop protein calorie malnutrition very rapidly during critical illness. This is because the increased baseline insulin levels suppress lipid mobilization from fat stores and enhance protein breakdown to fuel gluconeogenesis. The critically ill patient will degrade up to 50% more body protein than the nonobese patient, in part because the inflammatory response increases the need for paralyzing agents, sedation, prolonged bed rest from longer times on the ventilator, and prolonged and higher amounts of inotropic and pressor support and also because of the generalized metabolic insult that these patients sustain. The clinical consequences of protein catabolism are severe, and steps need to be taken to combat this process.

Unfortunately, determining the proper amount of caloric and protein replacement is not easy or well elucidated. The best method to determine caloric needs is indirect calorimetry, because using the Harris-Benedict equation is inaccurate secondary to trouble deciding just what weight to use, with an error rate of 74% using ABW and 36% using adjusted body weight (AdjBW) with an increased incidence of underestimation. In the absence of indirect calorimetry, a current recommendation is to give 20 to 30 kcal/kg/d on an obesity-adjusted weight as follows:

$$\text{IBW + (ABW – IBW) } \times 0.025$$

The main question for feeding morbidly obese patients in the ICU is whether to use hypocaloric feedings, that is, feedings that meet protein requirements but provide fewer calories than expended. Some studies have shown a benefit to hypocaloric feedings, in terms of decreased ICU stay, fewer days of antibiotics, and a trend toward fewer days of mechanical ventilation. Other studies are more equivocal, so the issue is still debated. As with any type of critically ill patient, the enteral route is preferable to the parenteral route.

**Pharmacologic Concerns**

Drug dosing provides daily challenges in the care of the critically ill patient. Volumes of distribution may vary widely depending on the patient’s amount of muscle mass, adipose tissue, and edema, and 3 patients with the same illness and same body weight may have widely differing drug dosing requirements based purely on body composition. Ideally, a size descriptor would be available that would take into account all the important measures: age, sex, race, height, weight, edema status. Unfortunately, none of the available descriptors take into account all of these factors and none are terribly accurate in determining drug dosing for all drugs on a consistent basis. Nonetheless, 2 have become favorites in determining drug dosing, so they are worthy of mention.

Ideal body weight (IBW) is a surrogate for lean body mass and is possibly the most commonly used. Surprisingly, the most common method of calculation, the Devine method, comes from a reference in 1974 with unclear origins, has not been since validated or updated, and is in article about gentamicin dosing. This method is used in many pharmacokinetic studies of obesity and is calculated as follows:

- **IBW (males) = 50 kg + 2.3 kg/in > 5 ft**
- **IBW (females) = 45.5 kg + 2.3 kg/in > 5ft**

Adjusted body weight (AdjBW) is usually used for dosing patients with mild to moderate obesity, and it assumes distribution of drug limited to some portion of excess weight. It is often used if ABW is between 130% and 200% of IBW. It is calculated as follows:

$$\text{AdjBW} = (\text{ABW} – \text{IBW}) \times 0.4 + \text{IBW}$$

Obesity causes functional changes in the body’s handling of drug in a number of areas: blood flow changes, metabolic changes, and binding changes. The changes may affect the volume of distribution (Vd) of a drug or the clearance (Cl) of a drug and may complicate an already confusing situation in the ICU. The situation is further complicated when
clinicians try to determine the patient’s body composition—including muscle, adipose tissue, and edema—and how it changes during the ICU stay, both of which affect drug dosing.

Some generalizations are in order. Patients with an ABW within 120% of IBW are unlikely to have clinical consequences based on weight choice for drug dosing. Vd tends to increase with more lipophilic medications, and, somewhat vice versa, a small Vd (<15 L) reflects restriction to extracellular space. Vd is most unpredictable for highly lipophilic drugs with large Vd. There is no one good single reference or chart to use for dosing. Some specific examples follow.

Vancomycin
Some of this discussion of vancomycin may apply to β-lactam antibiotics. Vancomycin experiences a large increase in Vd but an even larger increase in Cl.\(^3\) As with β-lactams, there is non-concentration-dependent killing, and side effects from overdosing are not nearly as severe as with aminoglycosides. For severe infections, one does not want to err on the low side of dosing, particularly when there are penetration concerns, such as for pneumonia and meningitis. Substantial variations occur in the morbidly obese patient. The changes in Cl are more dramatic than the change in Vd, so there is a shortened drug half-life, leading to the suggestion that the dosing interval be decreased to every 8 hours for vancomycin as well as the suggestion that the drug be administered via continuous infusion. Regardless, measuring of levels is paramount in this patient population.

Low-Molecular-Weight Heparin
Daily dosing of low-molecular-weight heparins is commonly suggested for reasons of cost and convenience. In certain patient populations, however, such dosing may not be sufficient. Trauma patients, even those who are not obese, suffer low troughs with once-daily dosing of subcutaneous enoxaparin.\(^3\) There are increases in drug Cl and Vd in obesity that are not proportional to the increases in body weight, and bleeding concerns arise if ABW is used to calculate drug dosing. The suggestion has been made that using AdjBW with more frequent dosing (ie, every 8 hours) may be preferable to using ABW or IBW for calculating enoxaparin dosing.\(^3\)

Special Nursing Considerations
Patients with morbid obesity present special concerns for nursing care. Patients are at staggering risk for the development of pressure ulcers for a number of reasons. The decreased perfusion of adipose tissue puts that tissue and the overlying skin at risk for reduced blood flow. The weight of the patient above the part of the body that is in contact with the mattress combined with any local low-blood-flow states makes these patients particularly susceptible to development of pressure ulcers, which may show up days or weeks after an episode of shock. Because of size and weight, patients are difficult to turn and keep in a turned position and have difficulty tolerating positions that are not supine. For these reasons, patients who are expected to be in bed for more than a few days should have a special bed or mattress, preferably one that is optimized for bariatric patients and has low air-loss surfaces with pressure relief features. Patients who are in shock should be considered to be at especially increased risk. Early recognition and treatment of pressure ulcers, including surgical débridement are essential.

All areas of the skin, not just those that are under pressure, are at high risk for breakdown and delayed wound healing, especially in the skinfolds, where the moist conditions harbor pathogens and induce breakdown of integumentary barriers. To prevent this, daily inspection and frequent scheduled turning are essential. The placement of powders in skinfolds actually worsens skin breakdown and should be avoided. Skin must be protected against pressure, shearing, and pinching, and this is especially necessary when using artificial lifts and springs to help turn the patient. Tubes such as Foley catheters should not be placed in skinfolds, as this will promote skin breakdown in those areas. If the ICU has a large census of critically ill morbidly obese patients, then procurement of specialized bariatric equipment, such as movable sliding air mattresses, patient lifts, and specialized bed chairs, may be warranted. Other special equipment, such as blood pressure cuffs and laryngoscope handles, is frequently needed.

Proper positioning is crucial for optimal pulmonary toilette. The least beneficial positions are the supine, Trendelenburg, lithotomy, and prone positions: these promote dyspnea, atelectasis, and hypoxemia. Slightly better is the so-called cardiac chair position, but in this position the pannus usually gets in the way of proper diaphragmatic excursion. The most beneficial position is the lateral decubitus position, if the patient will tolerate it, because it allows for displacement of the abdomen and greater diaphragmatic excursion. The 30° to 45° semirecumbent position has similar properties and is helpful in the immediate postoperative period.

Patients with morbid obesity present many challenges to their care in the ICU, but with proper management they can enjoy treatment success near to that of nonobese patients.
REFERENCES