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ABSTRACT

# Extreme obesity in the ICU

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Morbid obesity remains an extremely serious disorder resulting in significant impairment of health all over the world. This is particularly true especially now in the setting of lockdowns and people not exercising due to the recent COVID-19 pandemic. In general, overweight and obese adults are at in increased risk of morbidity and mortality from many acute and chronic medical conditions, including hypertension, dyslipidemia, coronary heart disease, diabetes mellitus, gallbladder disease, respiratory disease, some types of cancer, gout and arthritis. Although body weight that exceeds ideal standards as determined by age, sex and height may be accounted for by a greater muscle mass or bone mass, the majority of individuals who weigh more than 20% over their calculated ideal body weight (IBW) have excessive adipose mass. What is even more alarming is the fact that the incidence of obesity in the USA has increased progressively since 1960, when the first survey was conducted. More recent data has demonstrated that the prevalence of obesity is three times higher in the USA than France, and one-and-a-half times that of England. As obesity is such a pervasive disorder in our society, and because obesity is an important risk factor for many diseases, it is not surprising that many obese patients are treated in the intensive care unit (ICU).

Extreme (morbid) obesity is currently a common medical condition. It is defined as having a body mass index (BMI) of >40 kg/m$^2$ and is associated with an increased risk of mortality. These critically ill morbidly obese patients present the critical care team many unique challenges. As morbid obesity increases the incidence of complications of patients admitted to an ICU, it is likely that these patients will have a longer hospital stay and poorer outcome.

The management of the morbidly obese critically ill patient is a challenging and formidable task. A better understanding of the pathophysiologic changes that occur with obesity and the complications unique to this group of patients may improve their outcome. From a pulmonary standpoint, significant abnormalities in lung function occur with as the BMI increases. The total lung capacity, functional residual capacity (FRC) and vital capacity are reduced by up to 30%. Lung function testing demonstrates a restrictive type of pattern. The work of breathing is increased due to abnormal chest elasticity, increased chest wall resistance, increased airway

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resistance (Raw), abnormal diaphragmatic position and upper airway resistance, as well as the need to eliminate a higher daily production of carbon dioxide. Patients with severe obesity are generally hypoxemic, with a widened alveolar-arterial oxygen gradient caused by ventilation-perfusion mismatching. Alveolar collapse and airway closure at the bases contribute to this phenomenon. The fall in FRC when assuming a supine position further increases ventilation-perfusion mismatching. This may result in severe arterial hypoxemia, and sudden death. These alterations in pulmonary function have important implications in the management of the obese patients requiring mechanical ventilation. Patients with a BMI >40 $kg/m^2$ frequently require greater mechanical ventilation when they present to the ICU with conditions such as exacerbations of chronic obstructive lung disease, pneumonia or sepsis. When using mechanical ventilation in these individuals, the small lung volumes and increased airway resistance necessitate the use of relatively small tidal volumes. These tidal volumes should not be calculated according to the patient's weight, but rather determined by the airway pressures and blood gasses. The use of positive end-expiratory pressure (PEEP) may prevent end-expiratory airway closure and atelectasis, particularly in dorsal lung regions. Weaning the obese patient from mechanical ventilation is frequently a difficult task. A reverse Trendelenburg position at 45 degrees results in a larger tidal volume and lower respiratory rate than the 0, or 90 degree position, and they postulated that this position may facilitate the weaning process. Obese patients have been reported to have a higher incidence of postsurgical pulmonary complications.

Morbid obesity is the single most important risk factor for pulmonary embolism. Obese patients have been documented to have a higher incidence of postoperative thromboembolic disease. Decreased mobility, venous stasis and an increased thrombotic potential may account for this finding. Diminished levels of antithrombin III and circulating fibrinolytic activity have been demonstrated in obese patients.

Endotracheal intubation can be a daunting experience in the morbidly obese patient. In the Australian Incident Monitoring Study, obesity with limited neck mobility and mouth opening accounted for the majority of cases of difficult intubation. Only experienced clinicians should attempt airway management in these patients.

Morbid obesity is characterized by an increase in total blood volume and resting cardiac output. Both increase in direct proportion to the amount the patient weighs over the IBW. The increase in cardiac output is largely due to an increase in stroke volume. The cardiac and stroke index are therefore normal in obese patients. Baseline oxygen consumption is increased, with a normal arterio-venous oxygen difference, suggesting that the cardiac output increases primarily to serve the high metabolic requirements of excessive fat. Although the resting cardiac output is increased, obese patients have been demonstrated to have a depressed ejection fraction, both at rest and after exercise. Cuff sphygmomanometry can be inaccurate in the obese depending on the size of the cuff used, continuous monitoring of systemic blood pressure with an arterial cannula may be prudent in such patients.

Obesity alters the pharmacokinetics of various drugs depending on their physical and chemical properties and mode of metabolism. These factors must be taken into account when dosing obese patients. In many instances toxic drug levels will be obtained if obese patients are dosed based on their actual body weight.

The majority of obese individuals have a larger absolute lean body mass as well as fat mass, when compared with normal individuals of the same age, weight and sex. The lean component of the body weight accounts for 20 to 40% of the excess weight. Calculated for total body weight (TBW), the percentage of lean tissue and water is reduced, but that of fat is increased. Although the cardiac output and total blood volume are increased, the blood flow per gram of fat is less than in non-obese individuals. These changes, together with a drug's lipid solubility, determine the alterations in the volume of distribution (Vd) and clearance (Cl) of drugs in obese patients. The half-life (t½) of a drug is dependent on the interaction between the changes in the Vd and Cl. The changes in the Vd largely determine the loading dose, while the Cl determines the maintenance dosing regimen.

Although obese individuals have excess body fat stores and large lean body stores, they are likely to develop protein energy malnutrition in response to metabolic stress, particularly if their nutritional status

was poor before injury. Nutrition should not be withheld from the obese patients in the mistaken belief that weight reduction is beneficial during critical illness. Traumatized, obese patients mobilize more protein and less fat compared with non-obese subjects. A block in both lipolysis and fat oxidation has been reported in obese patients resulting in a shift to the preferential use of carbohydrates which further accelerates body protein breakdown even further to fuel gluconeogenesis. This increased carbohydrate use for fuel increases the respiratory quotient. The obese patients' energy expenditure should therefore be measured by indirect calorimetry. If indirect calorimetry is not available patients should receive between 20 to 30 Kcal/Kg of IBW/ day. Most of the calories should be given as carbohydrates with fats given to prevent essential fatty acid deficiency. Current consensus recommends a level of 1.5 to 2.0 g/kg of IBW to achieve nitrogen equilibrium.

Lastly, given the epidemiologic trends in obesity, as well as the long-term benefits of bariatric surgery, it's expected to continue as a common treatment option for these patients. A variety of postoperative complications have been described, including iatrogenic splenectomy, wound infection, incisional hernia, early and distal bowel obstruction, anastomotic leaks, pulmonary embolism, postoperative respiratory failure and pneumonia. Sudden cardiac death is a dreaded complication in the immediate postoperative period. Death from any cause is reported to be 0.3% for laparoscopic bariatric procedure within the first 30 days, and it rises to 2.1% when performed via laparotomy.

Careful consideration of all this variables is key when management patients with extreme obesity in the ICU.

**Keywords:** morbid obesity, critically ill, ICU