Obesity is a serious disorder resulting in a significant impairment of health.1 Overweight and obese adults are at 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.1-3 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, most individuals who weigh >20% over their calculated ideal body weight (IBW) have excessive adipose mass.4,5 The body mass index (BMI), which is the ratio of weight (in kilograms) to height (in meters) squared, is the most convenient method of quantifying the degree of obesity.6-8 The National Center for Health Statistics has defined overweight as a BMI of ≥27.8 in men and ≥27.3 in women. Severely overweight is defined as a BMI of ≥31.1 in men and ≥32.3 in women.8 The lower cutoffs correspond to approximately 20% above desirable body weight in the 1983 Metropolitan Life Insurance Company mortality tables, whereas the upper cutoffs correspond to 40% above desirable body weight.8

The most recent National Health and Nutrition Examination Survey reported that 34.9% of US adults are overweight.2,4 Furthermore, the incidence of obesity in the United States has increased progressively since 1960, when the first survey was conducted.2,4,5,9 In addition, recent data have demonstrated that the prevalence of obesity is three times higher in the United States than France, and one and a half times that of England.9

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 ICU. The critically ill obese patient presents the critical care team with many unique problems. The purpose of this article is to review some of the basic concepts related to the care of obese patients in the critical care setting.

**Critical Illness and Obesity**

To our knowledge, the impact of obesity on ICU outcome has not been studied. Obesity was not included as a comorbid variable in the development of the APACHE (acute physiology and chronic health evaluation) II and III prognostic indexes.10,11

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**Data from recent surveys indicate that a staggering 34.9% of US adults are overweight. Obese adults are at increased risk for many chronic medical conditions, and this increases the likelihood of admission to an ICU. The critically ill obese patient presents the ICU team with many unique problems. Obesity may result in significant alterations of pulmonary and cardiac function, as well as the handling of many drugs. An appreciation of these and other changes is essential in the management of the obese ICU patient. The purpose of this article is to review some of the basic concepts related to the treatment of obese patients in the ICU.**

*(CHEST 1998; 113:492-98)*

**Key words**: cardiac; complications; drug dosing; ICU; nutrition support; obesity; pharmacokinetics; pulmonary; surgery

**Abbreviations**: BMI = body mass index; FRC = functional residual capacity; IBW = ideal body weight; Raw = airway resistance; Vd = volume of distribution

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However, Smith-Choban and colleagues reported that morbidly obese patients have an eightfold higher mortality following blunt trauma than nonobese patients. Furthermore, hospitalized obese patients are at an increased risk of developing respiratory and other complications. It is therefore likely that obesity increases the incidence of complications of patients admitted to the ICU, and that this is associated with a longer hospital stay and poorer outcome.

**Respiratory Effects of Obesity**

The effect of obesity on lung function is complex and is influenced by the degree of obesity, age, and type of body fat distribution (central or peripheral). However, the expiratory reserve volume is consistently decreased and the FEV1 to FVC ratio increased. These indexes change in direct proportion to the degree of obesity. The fall in expiratory reserve volume is presumably due to small airway closure. The vital capacity, total lung capacity, and functional residual volume are generally maintained in otherwise normal individuals with mild to moderate obesity but are reduced by up to 30% in grossly obese patients. These latter changes occur predominantly in patients with central obesity. A simple mechanical effect of fat distribution on lung volumes would be the most likely explanation for these findings. The work of breathing is increased due to abnormal chest elasticity, increased chest wall resistance, increased airway 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 functional residual capacity (FRC) falls when assuming a supine position, further increasing ventilation-perfusion mismatching. This may result in severe arterial hypoxemia, and sudden death. Anesthesia further reduces the FRC, with the encroachment of the FRC on the closing volume. Abnormalities in the control of ventilation are common in obese patients. Vgontzas and colleagues demonstrated significant sleep apnea in 40% of men and 3% of women with a BMI >45.3.

These alterations in pulmonary function have important implications in the treatment of obese patients requiring mechanical ventilation. As the lung volumes may be reduced and Raw increased, a tidal volume calculated according to the patient’s actual body weight is likely to result in high airway pressures and alveolar overdistention. The initial tidal volume should therefore be based on the IBW, and then adjusted according to inflation pressures and blood gases. The use of positive end-expiratory pressure 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. Burns and colleagues have demonstrated that in obese patients, the reverse Trendelenburg position at 45° resulted in a larger tidal volume and lower respiratory rate than the 0° or 90° position, and they postulated that this position may facilitate the weaning process.

The risk of aspiration pneumonia is greatly increased in the obese patient. This complication is particularly common in the postoperative period. The risk of aspiration is increased due to several reasons, including the following: a higher volume of gastric fluid, a lower than normal pH of gastric fluid in fasting obese patients, increased intra-abdominal pressure, and a higher incidence of gastroesophageal reflux. This is another strong reason to nurse the obese patient in the semupright position.

Obese patients have been reported to have a higher incidence of postsurgical pulmonary complications. Rose and coauthors reported that acute postoperative respiratory events were twice as likely to occur in obese as compared with nonobese patients. Obese blunt trauma victims have a particularly poor outcome, frequently related to respiratory failure. Postoperative pulmonary dysfunction is accentuated by thoracic and upper abdominal incisions. Preoperative spirometry is suggested in all obese patient undergoing elective surgery in order to predict the likelihood of postoperative respiratory complications. Pain control strategies with minimal respiratory depression, such as continuous epidural patient-controlled analgesia are recommended. Postoperative respiratory monitoring with pulse oximetry, aggressive chest physiotherapy, and early physical mobilization are recommended.

Goldhaber and colleagues reported that obesity was the single most important risk factor for pulmonary embolism. Furthermore, 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. The high risk of thromboembolic disease in obese ICU patients may warrant an aggressive approach to deep venous thrombosis prophylaxis. The role of increased dose subcutaneous
heparin, low-molecular weight heparin, or the combination of pneumatic compression and heparin requires investigation.

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 most cases of difficult intubation.41

**Cardiovascular Effects of Obesity**

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.19,42-45 The increment in cardiac output is due solely to an increase in stroke volume with the heart rate being unchanged.19,42-45 The cardiac and stroke index are normal in otherwise healthy obese patients.19,42-45 The increase in cardiac output is accompanied by a decrease of systemic vascular resistance in normotensive patients. De Divitiis and colleagues43 performed left and right heart catheterization in 10 massively obese (mean BMI of 48.8) but otherwise healthy individuals. These authors noted that the mean oxygen consumption was increased (311 mL/min), and that the oxygen consumption increased linearly with increasing body weight. However, the arteriovenous oxygen difference was normal, suggesting that the cardiac output increases primarily to serve the metabolic requirements of excessive fat.42-44 The distribution of cardiac output has been reported to be similar in obese and lean individuals.45

Although the resting cardiac output is increased, obese patients have been demonstrated to have impaired left ventricular contractility and a depressed ejection fraction, both at rest and after exercise.43,46-49 Decreased myocardial β-adrenergic receptors may contribute to this finding.49,50 Furthermore, left ventricular mass, left ventricular wall thickness, and left ventricular cavity size increase, resulting in left ventricular dilatation and hypertrophy.51,52 These changes are related to both the degree and duration of obesity.51,52 Systemic arterial hypertension is common in the morbidly obese patient, with superimposed left ventricular hypertrophy. Diastolic dysfunction with a prolonged relaxation phase and early filling abnormalities have been reported to be early indicators of cardiac involvement in obesity.51

The left ventricular filling pressure is elevated in obese patients, due to the combination of increased preload and reduced ventricular distensibility.19,42-45 De Divitiis and colleagues43 reported a mean left ventricular end-diastolic pressure of 16.6 mm Hg in their series of patients. Consequently, fluid loading is poorly tolerated. As physical examinations are difficult and the signs of cardiac failure are unreliable in obese patients, invasive hemodynamic monitoring may assist in titrating fluid therapy and assessing cardiac performance. Furthermore, as cuff sphygmomanometry can be inaccurate in the obese (depending on the size of the cuff used) continuous monitoring of systemic BP with an arterial cannula may be prudent in such patients.

**Drug Dosing in Obese Patients**

The distribution, metabolism, protein binding, and clearance of many drugs are altered by the physiologic changes associated with obesity.53-57 Some of these pharmacokinetic changes, however, may negate the consequences of others and the pharmacokinetic alterations may differ in the morbidly obese compared with the mildly or moderately obese.57 In addition, the underlying disease of the patient may substantially influence the pharmacokinetic properties of the drug.58 The net pharmacologic alteration in any patient, therefore, is often uncertain. Nevertheless, for a number of drugs used in the ICU, most notably digoxin, aminophylline, aminoglycosides, and cyclosporine, toxic reactions may occur if the patients are dosed based on their actual body weight.53-57,59,61

The oral absorption of drugs remains essentially unchanged in the obese patient.54 These patients have increased levels of lipoproteins, triglycerides, cholesterol, and free fatty acids that bind to serum proteins and may inhibit the protein binding of drugs.52,63 In addition, the levels of α1-acid glycoprotein have been reported to be increased in obese patients, and this may increase the degree of protein binding of some drugs.64,65 The clinical significance of these changes remains unclear.

The volume of distribution (Vd) of drugs in obese patients is largely dependent on the lipophilicity of the drug.54,56 The Vd of drugs that are weakly lipophilic (aminoglycosides, quinolones) is moderately increased when compared with the Vd in normal individuals, but the Vd corrected by actual body weight is significantly smaller. However, the Vd is normal for some weakly lipophilic drugs (theophylline, histamine2-blockers, neuromuscular blockers). The Vd is increased for many but not all lipophilic drugs. Benzodiazepines, verapamil and sufentanil, have a large Vd, indicating distribution into adipose tissue. However, for other lipophilic drugs, the Vd and Vd per kilogram are decreased (digoxin, cyclosporine, propranolol), suggesting that factors other than lipid solubility affect tissue distribution.54
Although histologic abnormalities are common on liver biopsy specimens in morbidly obese patients,66 the clearance of most drugs that are hepatically metabolized is not reduced. However, with some drugs (methylprednisolone, propranolol), hepatic clearance is markedly reduced.54,67,68 Phase I hepatic metabolic reactions (oxidation, reduction, hydrolysis) are substrate dependent and are usually increased or unchanged in obesity.68 In contrast, metabolism of drugs (such as lorazepam) by some phase II reactions (conjugation by sulfation or glucuronidation) is consistently increased in obesity.69

For drugs that are excreted renally, elimination will depend on the creatinine clearance. A higher glomerular filtration rate has been reported in obese patients with normal renal function,70,71 and this will increase the clearance of drugs that are eliminated primarily by glomerular filtration.72 In obese patients with renal dysfunction, the creatinine clearance, as calculated using standard formulas, correlates very poorly with the measured creatinine clearance.73 Therefore, in the obese patient with renal dysfunction, the dosing regimen of renally excreted drugs should be based on the measured creatinine clearance.

As a consequence of the complexity of the pharmacokinetic changes that may occur in obese patients and the limited data available for many drugs, there is inconsistency and disagreement in the literature regarding drug dosing in obese patients.53-57 For many drugs, it is unclear if weight-related dosage adjustments should be made, and whether these adjustments should be based on the actual body weight, IBW, or a percentage of the actual body weight. Furthermore, monitoring of clinical end points (heart rate, BP, analgesia, sedation, etc) and/or serum drug levels is more important than the empiric dosing of medications based on published pharmacokinetic data.

**Nutritional Requirements**

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.74,75 Nutrition should not be withheld from 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 nonobese subjects.76 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.76 This increased carbohydrate use for fuel increases the respiratory quotient.

Energy expenditure equations are unreliable in critically ill patients, particularly if they are obese.77 It is unclear as to whether the IBW or total body weight should be used in these equations.78 The obese patients’ energy expenditure should therefore be measured by indirect calorimetry.79 If indirect calorimetry is not available, patients should receive between 20 to 30 kcal/kg of IBW per day.80 Most of the calories should be given as carbohydrates with fats given to prevent essential fatty acid deficiency.75 It has been suggested that critically ill obese patients receive nutritional support with a hypocaloric high-protein formulation. It has been postulated that if adequate protein is supplied and obligatory glucose requirements are met, endogenous fat stores will be used for energy.81 In a prospective, double-blind study, Burge and colleagues80 demonstrated that feeding mildly to moderately stressed obese patients with a hypocaloric total parenteral nutrition solution achieved comparable nitrogen balance to that of patients given conventional total parenteral nutrition formulas. Protein requirements in the obese patient may be difficult to determine because of the increased lean body mass. Current consensus recommends a level of 1.5 to 2.0 g/kg of IBW to achieve nitrogen equilibrium.75,80,81

**Gaining Vascular Access**

Poor peripheral venous sites in obese patients necessitate more frequent use of central venous access. A short stubby neck, loss of physical landmarks, and a greater skin-blood vessel distance make internal jugular and subclavian vein cannulation technically difficult.82,83 This results in a higher incidence of catheter malpositions and local puncture complications. A greater number of skin punctures during catheter insertion and delayed catheter changes may lead to more catheter-related infections and thromboses.84 Femoral venous access may not be possible, as these patients usually have severe intertrigo. The use of Doppler ultrasound-guided techniques for obtaining central venous access in high-risk patients has been demonstrated to reduce the number of needle passes to cannulate the vein, with a reduction in the incidence of complications.82,85,86 Arterial line placement can also be challenging in these patients.
**Radiologic Procedures**

Portable bedside radiographs are usually of a very poor quality in the obese patient, limiting the value of this important diagnostic tool. Abdominal and pelvic ultrasonography is limited by extensive abdominal wall and intra-abdominal fat. Percutaneous aspiration and drainage of intraperitoneal and retroperitoneal collections may be hindered by the obese body habitus. Many CT tables have weight restrictions (about 160 kg) that prohibit imaging of the morbidly obese patient.

**Conclusions**

The treatment 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. Further research on the impact of obesity on the outcome of critically ill patients is warranted.

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