Review Article

Clinical and critical care concerns in severely ill obese patient

Sukhminder Jit Singh Bajwa, Vishal Sehgal[1], Sukhwinder Kaur Bajwa[2]

Departments of Anaesthesiology and Intensive Care, and [2]Obstetrics and Gynaecology, Gian Sagar Medical College and Hospital, Ram Nagar, Banur, Punjab, India, [1]Department of Internal Medicine, The Commonwealth Medical College Scranton, PA 18510, USA

ABSTRACT

The incidence of obesity has acquired an epidemic proportion throughout the globe. As a result, increasing number of obese patients is being presented to critical care units for various indications. The attending intensivist has to face numerous challenges during management of such patients. Almost all the organ systems are affected by the impact of obesity either directly or indirectly. The degree of obesity and its prolong duration are the main factors which determine the harmful effect of obesity on human body. The present article reviews few of the important clinical and critical care concerns in critically ill obese patients.

Key words: Body mass index, critically ill, obesity, sleep apnea syndrome

Introduction

The increased prevalence of obesity [defined by a body mass index (BMI) greater than 30 kg/m^2] is worldwide and is associated with abnormally high percentage of fat in the body. However, the incidence and prevalence of obesity varies between countries; whereas in Japan and China only 1 in 20 adult women is obese, in the Netherlands 1 in 10 women are grossly overweight compared to 1 in 4 in the UK and Australia, 1 in 3 in the USA, and a staggering 7 in 10 women in Tonga.[1-3] The data from majority of the developing nations are lacking due to non-uniformity and underreporting. However, the latest report from surveys in India is pointing toward an alarming growth of this modern epidemic[4] [Table 1].

The epidemic of obesity started almost in the mid-70s, especially in the developed countries, with the commonest denominator being steady rise in the apparent food consumption per capita in high-income countries. However, the definition and clinical significance of being overweight and obesity varies by race. In certain ethnic groups, the potential clinical risks in terms of percent body fat are reached at a much lower BMI as in South Asians, co mpared to others such as in Blacks of African origin. For ease of description, obesity is universally classified on the basis of BMI in a much simpler manner [Table 2].

Table 1: The prevalence of obesity in the top 10 states of India

States	Males (%)	Males (rank)	Females (%)	Females (rank)
Punjab	30.3	1	37.5	1
Kerala	24.3	2	34	2
Goa	20.8	3	27	3
Tamil Nadu	19.8	4	24.4	4
Andhra Pradesh	17.6	5	22.7	10
Sikkim	17.3	6	21	8
Mizoram	16.9	7	20.3	17
Himachal Pradesh	16	8	19.5	12
Maharashtra	15.9	9	18.1	13
Gujarat	15.4	10	17.7	7

The critical care of severely ill patients has always been a difficult task for the intensivist. These clinical situations become difficult to handle in the presence of any co-morbid disease. Critically ill obese patients are extremely

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Corresponding Author: Dr. Sukhminder Jit Singh Bajwa, Department of Anaesthesiology and Intensive Care, Gian Sagar Medical College and Hospital, Ram Nagar, Banur, Punjab, India. E-mail: sukhminder_bajwa2001@yahoo.com

Table 2: Classification of obesity as per body mass index

Weight category classification	BMI (kg/m²)
Underweight	<18.5
Normal weight	≥18.5–24.9
Overweight	≥25.0–29.9
Obesity	≥30.0
Obesity Class I	30.0–34.9
Obesity Class II/morbid obesity	35.0–39.9
Obesity Class III/grossly morbid obesity	≥40

challenging as these patients are invariably having compromised dysfunction of one or more organ systems besides the deranged pathophysiology due to obesity. The morbidity increases substantially with increase in body weight, with a subsequent increase in risks of hypertension, diabetes mellitus, coronary artery disease, dyslipidemia, gall bladder disease, osteoarthritis, respiratory problems, and cancers of various organs.[5]

Clinical Challenges

Clinical and nursing care for critically ill, morbidly obese patient is a challenge for the entire intensive care unit (ICU) team. There are only few studies which have comprehensively covered the critical care aspects of severely obese patients in ICU.[6,7] The other dilemmatic factor in assessment was the non-inclusion of obesity in Acute physiological and Chronic Health Evaluation (APACHE) score as well as Simplified Acute Physiological Score (SAPS). In addition, it has been widely observed in various clinical situations that obese patients are more prone to develop complications and mortality following any major medical or surgical illness especially associated with longer ICU stay.[8-10]

Monitoring and investigation difficulties

The monitoring difficulties such as measurement of non-invasive blood pressure pose frequent problems as is the difficulty in posture changing during routine nursing care.[11] Furthermore, many diagnostic modalities are difficult to carry out and the quality of the investigations is often poor. Such difficult patients should ideally be managed in a tertiary care hospital with appropriate resources and availability of experienced staff who are regularly managing these patients.

Difficult vascular access

Not only airway management is challenging, but also securing of vascular access in these patients, whether arterial or venous, is equally difficult. Critically sick, morbidly obese patients require fluid administration which is guided by central venous pressure (CVP) and urine output[12] However, the insertion of central venous catheters in these patients tests the clinical skills of even the best of the intensivists.[13] The usual anatomic landmarks are obscured, the distance from skin to vessel is much further than normal, and the angle of approach may be too steep to allow cannulation even after reaching the vessel. The advent and increasing use of ultrasound-guided vascular access nowadays has brought some relief to the intensivists throughout the globe.[14,15]

Nutritional challenges

The basal metabolic rate and the energy requirements of critically ill patients are generally calculated based on actual body weight using the Harris-Benedict equation.[16] However, overestimation of the metabolic demand and supply ratio can lead to imbalanced nutrition in these patients, resulting in overfeeding. The energy expenditure can be appropriately estimated by indirect calorimetry, but this is not feasible in patients on mechanical ventilation and the limited availability in the ICUs of developing nations is another big problem.[17,18]

Glycemic control

Aggressive glycemic control with insulin has been shown to be beneficial in critically ill adults.[19] However, few studies have shown increased mortality and morbidity associated with intensive glycemic control [target serum glucose 81–108 mg/dL (4.5–6.0 mmol/L)] when compared with conventional therapy [serum glucose less than 180 mg/dL (10 mmol/L)]. The strict glycemic control itself is associated with various significant hazards as have also been demonstrated by NICESUGAR trial. Recently, the general consensus has been that sustained hyperglycemia is less detrimental than the fluctuating blood sugar levels.[20]

Nursing care difficulties

Obese patients are more prone to develop the pressure sores during their ICU stay because of the weight as well as the easily compromised circulation in the dependent areas. Equally difficult is the dressing of wounds, especially on the backside and of the pressure areas.[21,22] Moreover, the posture changing in these patients is a clinically difficult task and adequate manpower is required for careful handling and turning the patient every time. There are potential risks of causing injury during these interventions as well as risk of accidental extubation and dislodgement of invasive vascular lines which can be extremely hazardous. Overall, general nursing care is extremely difficult in grossly obese patients, especially those who are unconscious and/or on mechanical ventilation.[23-25]

Pharmacological challenges

There are no universal guidelines and protocols for administration of dosing schedules in morbidly obese patients and evidence-based methods for determining

appropriate dosing strategies in such patients are lacking. An in-depth knowledge of basic pathophysiologic aspects concerning the volume of distribution and clearance of hydrophilic and lipophilic drugs is necessary for determining appropriate dosage regimens. The altered physiology in obese patients is characterized by a larger volume of distribution (Vd) for lipophilic drugs, increased clearance of hydrophilic drugs, and a decrease in lean body mass and tissue water content, as compared to normal population.[26] These altered mechanisms can predispose the morbidly obese patients to either the limited efficacy of under dosages or systemic toxicity due to over dosages of various pharmacological agents.[27,28]

Pharmacological Considerations During Intensive Care

Dosing schedules and strategies

Weight-based medication schedule is commonly used for avoiding any systemic side effects and lack of clinical efficacy by under dosages. The ideal body weight (IBW) is considered suitable for standard dosing of various drugs like penicillins, cephalosporins, linezolid, corticosteroids, H2-blockers, digoxin, beta-blockers, atracurium, vecuronium, fentanyl, midazolam, lorazepam, phenytoin, and for propofol during sedation in ICU. Total body weight (TBW) is used for administration of succinylcholine, rocuronium, unfractionated heparin, enoxaparin, and vancomycin, while dosing weight (DW) is used for aminoglycosides and fluoroquinolones.

Antimicrobials

Dosing schedule of antibiotics should be determined on an individual basis and the consequences of inadequate treatment should be kept in mind. If possible, doses should be titrated on the basis of plasma levels in high-risk patients.[29] The following evidence-based therapeutic regimens are cited for a possible help in dose adjustments in critically ill obese patients.

- IBW is appropriate for dose calculation and administration in case of cephalosporins, penicillins, and beta-lactams, and preferably the doses should be administered at the upper end of recommended ranges of these drugs. As beta-lactams exert time-dependent bactericidal effects, the doses of these should be adjusted frequently, for example, ampicillin intravenously every 4 hours or as a continuous infusion.
- TBW is ideal for adjusting the dose titration of drugs like vancomycin and related drugs like daptomycin, quinpristin, and dalfopristin. However, a loading dose is recommended to attain rapid therapeutic concentrations and plasma levels can be maintained with divided doses every 8 or 6 hours, rather than a standard every-12-hour regimen.
- DW is the standard selection for aminoglycosides as these drugs are hydrophilic and frequency of administration is determined by renal function and adjusted based on serum drug concentrations.
- Dosages at the upper end of recommended levels are suggested for fluoroquinolones in obese patients as the optimal dose schedule for obese patients is not well cited in literature.

Sedatives, analgesics, and muscle relaxants

However, the dosing schedule for opioids and sedatives like benzodiazepines should be carefully titrated as these drugs have potential to cause respiratory depression in morbidly obese patients.[30] Therefore, initially a small dose can be administered and the strength can be increased on the basis of desired clinical effect. For drugs like fentanyl, remifentanyl, and morphine, IBW is the appropriate standard for the formulation of dosing schedule.[31] The availability of naloxone should be ensured before administration of opioids, especially for non-intubated patients.[32]

- IBW is again the criterion of choice for administration of drugs like propofol, midazolam, and lorazepam as these pharmacological agents are lipophilic and redistribute widely to adipose tissue following initial rapid distribution to central nervous system when administered intravenously.[33]
- TBW should be considered when administering neuromuscular blocking agents such as succinylcholine and rocuronium, while IBW should be the basis for administration of muscle relaxants such as atracurium and vecuronium.[34]

Anticoagulants

The volume of distribution of anticoagulants like heparin in obese patients differs from that in non-obese patients since adipose tissue has a lower blood volume than lean tissue; as a result, heparin dosing requirements do not increase linearly with body weight. There are no established guidelines and protocols for administration of optimal dosing schedule for heparin and low-molecular-weight heparins (LMWHs) in obesity. The literary evidence, however, suggests the use of TBW to calculate the initial bolus dose and infusion rate to achieve a therapeutic partial thromboplastin time (PTT).[35] As per various guidelines on parenteral anticoagulants, it is suggested that weight-based dosing is preferable to fixed dosing in obese patients. PTT should be the criterion for adjusting the initial rate of these pharmacological agents after an elapse of first 6 hours. Also, it is recommended that initial administration of these drugs should be on the higher side, whether as bolus or continuous infusion. The functional heparin

assay [the anti-factor Xa activity (anti-Xa)] provides a reference standard for testing the *in vivo* activity of heparin and may be more useful for heparin monitoring than the PTT in obese patients. Enoxaparin and LMWH, whenever administered, should be adjusted according to TBW.[36,37] There are concerns regarding the absorption mechanisms of LMWH, whenever it is administered through subcutaneous route as there is variable peripheral perfusion due to morbid obesity.[38] Whenever possible, monitoring of action of these anticoagulants should be carried out by estimating levels of anti-Xa factor. Enoxaparin administered as 40 mg every 12 hours and as 60 mg every 12 hours subcutaneously provides effective prophylaxis against venous thromboembolism in bariatric patients up to a BMI 50 kg/m^2 and with a BMI exceeding 50 kg/m^2, respectively.[39]

Cardiac medications

For administration of cardiac drugs like beta-blockers, digoxin, and calcium channel blockers, IBW should be the basis for titration of doses. This holds good for the maintenance and supplemental doses of these cardiac drugs as the IBW helps in determination of appropriate dosing schedule to bring about the desired clinical effect.

Respiratory Evaluation and Considerations

The pulmonary mechanics are grossly deranged in obese patients and are influenced by degree of obesity, age, and body fat distribution, and result in marked reduction in expiratory reserve volume (ERV) and forced exdpiratory volume in 1st second (FEV1) to forced vital capacity (FVC) ratio. These changes occur especially in central obesity and the decrease in ERV causes small airway closure.[40]

On the other side, the work of breathing is increased because of decreased compliance of the chest wall, increased airway resistance, abnormal diaphragmatic position, upper airway resistance, and a continuous need for exhalation of carbon dioxide produced from high metabolic rate.[41,42] As a result, these patients become generally hypoxemic and have a greater alveolar–arterial oxygen gradient, leading to ventilation–perfusion mismatch. It ultimately results in profound atelectasis with subsequent intrapulmonary shunting, leading to severe hypoxemia and rapid desaturation.[43,44]

Obstructive hypoventilation syndrome and sleep apnea syndrome

The control of normal physiological ventilation is disturbed in obese patients due to encroachment of FRC on the closing volume.[45-47] It has been postulated that there is possible deranged production of anti-obesity hormone leptin, which can be a potential risk factor for development of sleep apnea syndrome.[48] Obesity hypoventilation syndrome and obstructive sleep apnea syndrome are potential risks for the development of peri-operative and postoperative respiratory depression in the morbidly obese patients. The challenges are accentuated during airway management while intubating these patients in ICU as they generally have smaller airways and a component of respiratory failure due to chronic hypercarbia state and various associated co-morbidities.[49]

Analgesic considerations

Any painful trauma or pathology in these patients inhibits the normal breathing movements due to perceived pain, especially in the abdominal surgeries. As such, compromise of the breathing movements can enhance the possibilities of postoperative atelectasis. We are using and strongly recommend the administration of epidural analgesia for any such pathology to control the harmful effects of postoperative pain during ICU stay.[50]

All these deranged pulmonary dynamics have significant implications for mechanical ventilation during ICU stay. The tidal volume, which should be based on IBW, minute ventilation, inflation pressure, peak and static airway pressures have to be carefully titrated to prevent any incidence of hypoxemia or barotraumas.[41] Basal atelectasis, impaired pulmonary functions, and reduced clearing of secretions predispose these patients to a higher incidence of ventilator-associated pneumonias.[51]

Airway Assesment and Management in Obese Patients

Airway management is extremely difficult in few of the cases and even an experienced intensivist can have a tough intriguing day at the office while managing such challenging situations. The large amount of redundant oropharyngeal tissue, small oral aperture, and short/thick neck with limited range of neck movements can make laryngoscopic visualization of the airway opening difficult.[52]

Predictors of difficult airway

Most of the obese patients can be managed using standard techniques and airway securing by direct laryngoscopy.[52] However, in morbid obesity, the predictors of difficult airway may include, but are not limited to, short neck with limited range of movements, inadequate mouth opening, higher Mallampatti score, heavy jaw, history of sleep apnea, presence of chronic respiratory airway disease, etc.[53]

Difficult airway adjuncts

The ideal technique in these circumstances mandates securing of airway with awake fiberoptic intubation, although direct laryngoscopy can be successful in majority of morbidly obese patients. Nowadays, use of videoscopes, glidescopes, and advanced version of the laryngeal mask airways has made the life of an intensivist quite simpler. As a result, the airway management scenario has become easier even in the most of the difficult airways, with the advent of modern airway equipment and adjuncts.

Risk of aspiration

One should be cautious while making attempts at intubation in these patients as this approach may carry a greater risk of aspiration if there is difficulty securing the airway. Aspiration prophylaxis involves administration of ranitidine, pentaprazole, sodium citrate, metoclopramide, etc., prior to intubation.

Ventilatory management

The heavy non-compliant chest wall decreases the static pulmonary compliance in morbidly obese as opposed to the common belief that the pulmonary parenchymal restriction causes it.[54] The tidal volume should be adjusted as per body weight and should be kept initially at 8 mL/kg of IBW in patients in whom mechanical ventilation is necessary. However, patients with acute respiratory distress should be ventilated with much lower tidal volume, but can be compensated by increasing the respiratory rate to maintain normal minute ventilation and thus avoiding hypoxemia and hypercarbia.[55] During prolonged ventilation, ventilatory settings are determined by peak airway pressures and serial measurements of arterial blood gas values.[56]

Sedation and Analgesia

The advent of dexmedetomidine has been revolutionary in clinical practice and is a good addition to the armamentarium of the intensivist.[57] Previously, sedation and analgesia was usually a challenging task. At present, the easy availability of newer sedative agents like benzodiazepines, propofol, dexmedetomidine, etc., as well as newer and safer opioids such as fentanyl, sufentanyl, and remifentanyl has made the task much easier.[58] These drugs enable a precise titration of sedation and analgesia levels on individual and daily basis. The preservation of hemodynamic stability with these drugs is an added advantage, especially in patients requiring long-term intensive care.[59]

Extubation, Recovery, and Weaning

General principles should be followed in morbidly obese patients and they should be extubated in fully awake state on achieving a good breathing pattern with adequate tidal volume. The individual clinical acumen is of immense benefit during extubation as premature extubation can have catastrophic consequences. The role of non-invasive positive pressure ventilation has gained significant popularity recently, but it requires the patient to be extremely co-operative.[60] The ideal preparation post extubation should include incentive spirometry which can possibly decrease the likelihood of respiratory complications.[61] The most challenging aspect in grossly obese patients in ICU is the difficult weaning from the mechanical ventilation.[62] Though various positions such as 30°, 45°, 90°, etc., have been tried to facilitate the weaning process, nothing conclusive has been established.[62]

Surgical Airway

The history of obstructive sleep apnea is very important in the context of respiratory care, especially if the patient is a regular user of non-invasive positive pressure ventilation. Elective tracheostomy is preferable for critically ill bariatric patients if it is anticipated that these patients can require mechanical ventilation for a prolonged duration. However, considering the short and thick neck, tracheostomy can often be technically difficult, and a carefully coordinated plan between general surgeon, ENT surgeon, and anesthesiologist must be designed prior to this intervention.[63]

Cardiovascular Assessment

The degree and increased duration of obesity is associated with a proportionate increase in total blood volume and resting cardiac output, which mainly occurs with increase in stroke volume while the heart rate remains stable and so does the cardiac index.[64,65] With progressive increase in obese tissues, there is impaired left ventricular (LV) contractility and decreased ejection fraction due to increased LV mass and thickness, as well as LV dilatation and hypertrophy.[65-68] The functional abnormalities are evident in the electrocardiographic findings also which may include, but are not limited to, leftward shift of QRS axis and prolonged PR, QRS, and QTc intervals.[67]

Complications and Management

Respiratory failure

Obesity complicated by the presence of hypoventilation sleep syndrome is a potential risk factor for developing hypercarbic respiratory failure, and administration of opioids in these patients increases the chances of respiratory depression. Postoperatively, respiratory failure is accentuated by the presence of severe pain which

can compromise the respiration due to greatly restricted breathing movements due to pain, especially in abdominal surgeries. These profound changes can lead to development of atelectasis and respiratory arrest in morbidly obese patients. Epidural analgesia is a good alternative for relief of pain as compared to systemic analgesics as various side effects of systemic analgesics can be avoided.[50]

All these deranged pulmonary dynamics have significant implications for mechanical ventilation during ICU stay. Ventilatory settings have to be considered on an individual basis, and as such, tidal volume should be based on IBW. Other ventilatory parameters like minute ventilation, inflation pressure, peak and static airway pressures have to be carefully titrated and adjusted also on an individual basis so as to prevent any incidence of hypoxemia or barotraumas.[41] Special considerations should be given to basal atelectasis, impaired pulmonary functions, and reduced clearing of secretions, as these can predispose obese patients to a higher incidence of ventilator-associated pneumonias.[51]

Hemodynamic instability and cardiovascular derangement

The cardiovascular system bears the maximum brunt and obesity can cause profound detrimental effects on the anatomical and physiological hemodynamic state. The total blood volume and cardiac output are elevated (a hyperdynamic state) in a manner linearly related to excess body weight. The deranged oxygen supply–demand ratio is probably driven by the increased oxygen requirements of excess adipose tissue.[69-71] The prolonged duration of these disturbances can cause permanent impairment of cardiac function, resulting in decreased LV contractility, LV hypertrophy, decreased cardiac compliance, and diastolic ventricular dysfunction. The resultant changes in these critically ill patients can cause inability to tolerate the difficulties handling the fluid loads.[72]

Thromboembolic prophylaxis

The risk of venous thrombosis and embolism is moderately higher in morbidly obese patients as compared to normal population. Critically ill obese patients are the ideal candidates for the prophylaxis of venous thrombosis. Limited to decreased mobility, pulmonary hypertension, venous stasis, and a potential hypercoagulable state are a few risk factors which predispose these morbidly obese patients to a higher risk of venous thrombosis and embolism.[73] The prophylactic use of compression stockings and sequential compression devices and pharmacological prophylaxis in the form of either LMHW or un-fractionated heparin (5000 units subcutaneously three times daily) should be considered in these patients. Obese patients with a greater-than-average risk of venous thrombo-embolism (VTE), including those with documented pulmonary hypertension or profoundly limited mobility, may benefit from continuation of their deep venous thrombosis (DVT) prophylaxis even after discharge from the hospital. The use of inferior vena cava filter is of immense benefit in patients who either have a contraindication to anticoagulation or have pulmonary hypertension and would have difficulty tolerating the hemodynamic insult of even a small pulmonary embolism (PE).[74]

Glycemic derangement

Glucose metabolism definitely gets deranged during prolonged ICU stay due to stress and, as such, glycemic control with insulin and dextrose infusion has been shown to be beneficial in critically ill adults.[19] The incidence of morbidity and mortality can increase in obese patients with impaired glucose levels as has been demonstrated by some studies. The morbidity index has shown higher values with intensive glycemic control [target serum glucose 81–108 mg/dL (4.5–6.0 mmol/L)] when compared with conventional therapy [serum glucose less than 180 mg/dL (10 mmol/L)]. Recently, the general consensus has been that sustained hyperglycemia is less detrimental than the fluctuating blood sugar levels, and thus the goal is to maintain normoglycemia.[20]

Infection and sepsis

Sepsis is a constellation of clinical signs and symptoms that complicate severe infection and is characterized by systemic inflammation and widespread tissue injury. The manifestations of sepsis are usually widespread and include cardinal signs of inflammation, including vasodilatation, increased microvascular permeability, and leukocyte accumulation in tissues remote from the original insult.

The risk of sepsis development is also higher in obese patients with associated co-morbidities such as diabetes and hyperglycemia that lead to impaired wound healing due to poorly vascularized adipose tissue and an elevated baseline inflammatory state, thus increasing the risk of sepsis.

The management of sepsis in severely ill, morbidly obese patients is a big challenge. Besides taking care of sepsis, many other appropriate measures have to be adopted simultaneously, such as source control, appropriate cultures of blood, urine, and sputum, and the administration of broad-spectrum intravenous empiric antibiotic therapy (at least two agents, covering a combination of gram-positive, gram-negative, and anaerobic organisms).

Aggressive fluid therapy is the initial treatment of choice in patients suffering from severe sepsis.[75] The vascular status in septic patients can be optimized with 1 L of 0.9% normal

saline or Ringer lactate solution which is administered until a CVP of 6–12 mmHg is achieved. Norepinephrine is the vasopressor of choice to maintain hemodynamic stability and should be started if the patient remains hypotensive after adequate volume resuscitation, with a goal of achieving mean arterial pressure (MAP) of 60–65 mmHg. Renal failure should be managed with renal replacement therapy on a daily basis in spite of no established benefits of continuous compared to intermittent renal replacement therapy.[76]

Pressure sores and stress ulcer prophylaxis

The risk of developing pressure sores and stress ulcer is greatly increased in obese individuals as compared to normal individuals. Stress ulcer prophylaxis mandates administration of either H2 receptor antagonists like ranitidine or proton pump inhibitors such as omeprazole, rabiprazole, and pentaprazole. The incidence of pressure sores is also higher in obese patients because of the weight as well as the easily compromised circulation in the dependent areas.[21,22] Moreover, the change of posture and dressing difficulties in these patients is a clinically difficult task and requires a good coordination between nursing staff during routine and general nursing, especially in unconscious and paralyzed patients. The use of soft cotton pads beneath the pressure areas and air mattresses is extremely helpful in these circumstances. There are potential risks of causing injury during these interventions as well as risk of accidental extubation and dislodgement of invasive vascular lines.[23-25] Nausea and vomiting can be safely managed in these patients by administration of newer long-acting 5HT3 antagonists like palonosetron and ramosetron.[77]

Conclusions

Morbidly obese patients are at a higher risk of development of various serious complications during postoperative period and prolonged ICU stay. Harris-Benedict equation is the commonly used equation which is based on actual body weight for calculation of total metabolic energy requirements of critically ill patients. However, there are possible chances of overestimating the metabolic demand of critically ill obese patients using this equation and this can lead to overfeeding. Morbidly obese patients should be treated in propped-up position in the ICU so as to minimize any respiratory compromise. Intensive care admission is mandatory if any morbidly obese patient demonstrates cardiorespiratory compromise in the immediate postoperative period. Any unexplained hemodynamic instability should warrant a close co-operation of the operating surgeon, anesthesiologist, and intensivist so as to detect the actual etiology in a timely manner.

References

1. Stevens J. Ethnic-specific cutpoints for obesity vs country-specific guidelines for action. Int J Obes Relat Metab Disord 2003;27:287-8.
2. Berghöfer A, Pischon T, Reinhold T, Apovian CM, Sharma AM, Willich SN. Obesity prevalence from a European perspective: A systematic review. BMC Public Health 2008;8:200.
3. Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM. Prevalence of overweight and obesity in the United States, 1999-2004. JAMA 2006;295:1549-55.
4. Third National Family Health Survey. Mumbai: International Institute for Population Sciences; 2006.
5. Calle EE, Thun MJ, Petrelli JM, Rodriguez C, Heath CW Jr. Body-mass index and mortality in a prospective cohort of U.S. adults. N Engl J Med 1999;341:1097-105.
6. Marik P, Varon J. The obese patient in the ICU. Chest 1998;113:492-8.
7. El-Solh AA. Clinical approach to the critically ill, morbidly obese patient. Am J Respir Crit Care Med 2004;169:557.
8. Fisher BL, Schauer P. Medical and surgical options in the treatment of severe obesity. Am J Surg 2002;184:9.
9. Goldhaber SZ, Goldstein E, Stampfer MJ, Manson JE, Colditz GA, Speizer FE, *et al*. A prospective study of risk factors for pulmonary embolism in women. JAMA 1997;277:642-5.
10. Rose DK, Cohen MM, Wigglesworth DF, DeBoer DP. Critical respiratory events in the postanesthesia care unit: Patient, surgical and anesthetic factors. Anesthesiology 1994;81:410-8.
11. Beevers G, Lip GY, O'Brien E. ABC of hypertension. Blood pressure measurement. Part I-sphygmomanometry: Factors common to all techniques. BMJ 2001;322:981.
12. Marik PE, Baram M, Vahid B. Does central venous pressure predict fluid responsiveness? A systematic review of the literature and the tale of seven mares. Chest 2008;134:172.
13. Brusasco C, Corradi F, Zattoni PL, Launo C, Leykin Y, Palermo S. Ultrasound-guided central venous cannulation in bariatric patients. Obes Surg 2009;19:1365-70.
14. Miller AH, Roth BA, Mills TJ, Woody JR, Longmoor CE, Foster B. Ultrasound guidance versus the landmark technique for the placement of central venous catheters in the emergency department. Acad Emerg Med 2002;9:800-5.
15. Gann M Jr, Sardi A. Improved results using ultrasound guidance for central venous access. Am Surg 2003;69:1104.
16. Elamin EM. Nutritional care of the obese intensive care unit patient. Curr Opin Crit Care 2005;11:300.
17. Cutts ME, Dowdy RP, Ellersieck MR, Edes TE. Predicting energy needs in ventilator-dependent critically ill patients: Effect of adjusting weight for edema or adiposity. Am J Clin Nutr 1997;66:1250-6.
18. McClave SA, Martindale RG, Vanek VW. ASPEN Board of Directors; American College of Critical Care Medicine; Society of Critical Care Medicine. Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient: Society of Critical Care Medicine (SCCM) and American Society for Parenteral and Enteral Nutrition (ASPEN). JPEN J Parenter Enteral Nutr 2009;33:277-316.
19. van den Berghe G, Wouters P, Weekers F, Verwaest C, Bruyninckx F, Schetz M, *et al*. Intensive insulin therapy in the critically ill patients. N Engl J Med 2001;345:1359.
20. Finfer S, Chittock DR, Su SY, Blair D, Foster D, Dhingra V, *et al*. Intensive versus conventional glucose control in critically ill patients. N Engl J Med 2009;360:1283.
21. Brem H, Nierman DM, Nelson JE. Pressure ulcers in the chronically critically ill patient. Crit Care Clin 2002;12:683-94
22. Mathison CJ. Skin and wound care challenges in the hospitalized morbidly obese patient. J Wound Ostomy Continence Nurs 2003;30:78-83.

23. Humphreys SL. Obesity in Patients and nurses increases the nurse's risk of injury lifting patients. Bariatric Nurs Surg Patient Care 2007;2:3-6.
24. Charlebois D, Wilmoth D. Critical Care of Patients with Obesity. Crit Care Nurse 2004;24:19-29.
25. Owens TM. Morbid Obesity the Disease and Comorbidities. Crit Care Nurs Q 2003;26:162-5.
26. Hanley MJ, Abernethy DR, Greenblatt DJ. Effect of obesity on the pharmacokinetics of drugs in humans. Clin Pharmacokinet 2010;49:71.
27. Cheymol G. Effects of obesity on pharmacokinetics implications for drug therapy. Clin Pharmacokinet 2000;39:215-31.
28. Erstad BL. Dosing of medications in morbidly obese patients in the intensive care unit setting. Intensive Care Med 2004;30:18.
29. Craft MK, Reed MJ. Immunologic changes in obesity. Crit Care Clin 2010;26:629-31.
30. Gan TJ. Pharmacokinetic and pharmacodynamic characteristics of medications used for moderate sedation. Clin Pharmacokinet 2006;45:855.
31. De Jonghe B, Cook D, Griffith L, Appere-de-Vecchi C, Guyatt G, Theron V, *et al*. Adaptation to the Intensive Care Environment (ATICE): Development and validation of a new sedation assessment instrument. Crit Care Med 2003;31:2344-54.
32. Young C, Knudsen N, Hilton A, Reves JG. Sedation in the intensive care unit. Crit Care Med 2000;28:854-66.
33. Angelini G, Ketzler JT, Coursin DB. Use of propofol and other nonbenzodiazepine sedatives in the intensive care unit. Crit Care Clin 2001;17:863-80.
34. Kress JP, Pohlman AS, O'Connor MF, Hall JB. Daily interruption of sedative infusions in critically ill patients undergoing mechanical ventilation. N Engl J Med 2000;342:1471.
35. Wilson SJ, Wilbur K, Burton E, Anderson DR. Effect of patient weight on the anticoagulant response to adjusted therapeutic dosage of low-molecular weight heparin for the treatment of venous thromboembolism. Haemostasis 2001;31:42-8.
36. Nutescu EA, Spinler SA, Wittkowsky A, Dager WE. Low-molecular-weight heparins in renal impairment and obesity: Available evidence and clinical practice recommendations across medical and surgical settings. Ann Pharmacother 2009;43:1064.
37. Borkgren-Okonek MJ, Hart RW, Pantano JE, Rantis PC Jr, Guske PJ, Kane JM Jr, *et al*. Enoxaparin thromboprophylaxis in gastric bypass patients: Extended duration, dose stratification, and antifactor Xa activity. Surg Obes Relat Dis 2008;4:625.
38. Duplaga BA, Rivers CW, Nutescu E. Dosing and monitoring of low-molecular-weight heparins in special populations. Pharmacotherapy 2001;21:218-34.
39. Myzienski AE, Lutz MF, Smythe MA. Unfractionated heparin dosing for venous thromboembolism in morbidly obese patients: Case report and review of the literature. Pharmacotherapy 2010;30:324.
40. Salome CM, King GG, Berend N. Physiology of obesity and effects on lung function. J Appl Physiol 2010;108:206-11.
41. Pelosi P, Croci M, Ravagnan I, Vicardi P, Gattinoni L. To tal respiratory system, lung, and chest wall mechanics in sedated-paralyzed postoperative morbidly obese patients. Chest 1996;109:144-51.
42. Koenig SM. Pulmonary complications of obesity. Am J Med Sci 2001;321:249-79.
43. El Shobary H, Backman S, Christou N, Schricker T. Use of critical care resources after laparoscopic gastric bypass: effect on respiratory complications. Surg Obes Relat Dis 2008;4:698.
44. Levi D, Goodman ER, Patel M, Savransky Y. Critical care of the obese and bariatric surgical patient. Crit Care Clin 2003;19:11.
45. Hedenstierna G.Gas exchange during anaesthesia. Br J Anaesth 1990;64:507-14.
46. Vgontzas AN, Tan TL, Bixler EO, Martin LF, Shubert D, Kales A. Sleep apnea and sleep disruption in obese patients. Arch Intern Med 1994;154:1705-11.
47. Frey WC, Pilcher J. Obstructive sleep-related breathing disorders in patients evaluated for bariatric surgery. Obes Surg 2003;13:676.
48. O'Donnell CP, Schaub CD, Haines AS, Berkowitz DE, Tankersley CG, Schwartz AR, *et al*. Leptin prevents respiratory depression in obesity. Am J Respir Crit Care Med 1999;159:1477-84.
49. den Herder C, Schmeck J, Appelboom DJ, de Vries N. Risks of general anaesthesia in people with obstructive sleep apnoea. BMJ 2004;329:955.
50. Steinbrook R. Surgery for severe obesity. N Engl J Med 2004;350:1075.
51. Marik PE, Careau P.A comparison of mini-bronchoalveolar lavage and blind-protected specimen brush sampling in ventilated patients with suspected pneumonia. J Crit Care 1998;13:67-72.
52. Brodsky JB, Lemmens HJ, Brock-Utne JG, Vierra M, Saidman L. Morbid obesity and tracheal intubation. Anesth Analg 2002;94:732-6.
53. Gonzalez R, Bowers SP, Venkatesh KR, Lin E, Smith CD. Preoperative factors predictive of complicated postoperative management after Roux-en-Y gastric bypass for morbid obesity. Surg Endosc 2003;17:1900-4.
54. Biring MS, Lewis MI, Liu JT, Mohsenifar Z. Pulmonary physiologic changes of morbid obesity. Am J Med Sci 1999;318:293.
55. Brower RG, Ware LB, Berthiaume Y, Matthay MA. Treatment of ARDS. Chest 2001;120:1347.
56. Ladosky W, Botelho MA, Albuquerque JP Jr. Chest mechanics in morbidly obese non-hypoventilated patients. Respir Med 2001;95:281.
57. Sudheesh K, Harsoor SS. Dexmedetomidine in anaesthesia practice: A wonder drug? Indian J Anaesth 2011;55:323-4
58. Gehlbach BK, Kress JP. Sedation in the intensive care unit. Curr Opin Crit Care 2002;8:290.
59. Panzer O, Moitra V, Sladen RN.Pharmacology of sedative-analgesic agents: Dexmedetomidine, remifentanil, ketamine, volatile anesthetics, and the role of peripheral mu antagonists. Crit Care Clin 2009;25:451-69.
60. Masa JF, Celli BR, Riesco JA, Hernández M, Sánchez De Cos J, *et al*. The obesity hypoventilation syndrome can be treated with noninvasive mechanical ventilation. Chest 2001;119:1102.
61. Gass GD, Olsen GN.Preoperative pulmonary function testing to predict posto-perative morbidity and mortality. Chest 1986;89: 127-35.
62. Burns SM, Egloff MB, Ryan B, Carpenter R, Burns JE. Effect of body position on spontaneous respiratory rate and tidal volume in patients with obesity, abdominal distension and ascites. Am J Crit Care 1994;3:102-6.
63. Mansharamani NG, Koziel H, Garland R, LoCicero J 3rd, Critchlow J, Ernst A. Safety of bedside percutaneous dilatational tracheostomy in obese patients in the ICU. Chest 2000;117:1426.
64. Backman L, Freyschuss U, Hallbert D, Melcher A. Cardiovascular function in extreme obesity. Acta Med Scand 1983;149:437-9.
65. Foster GD. Principles and practices in the management of obesity. Am J Respir Crit Care Med 2003;168:274-80.
66. Alpert MA, Singh A, Terry BE, Kelly DL, Villarreal D, Mukerji V. Effect of exercise on left ventricular systolic function and reserve in morbid obesity. Am J Cardiol 1989;63:1478-82.
67. Berkalp B, Cesur V, Corapcioglu D, Erol C, Baskal N. Obesity and left ventricular diastolic dysfunction. Int J Cardiol 1995;52:23-6.
68. Merlino G, Scaglione R, Carrao S, D'Amico C, Paterna S, Licata A, *et al*. Association between reduced lymphocyte betaadrenergic receptors and left ventricular dysfunction in young obese subjects. Int J Obes Metab Disord 1994;18:699-703.
69. Dellinger RP. Cardiovascular management of septic shock. Crit Care Med 2003;31:946.
70. Yaegashi M, Jean R, Zuriqat M, Noack S, Homel P. Outcome of morbid obesity in the intensive care unit. J Intensive Care Med 2005;20:147-54.

71. Abir F, Bell R. Assessment and management of the obese patient. Crit Care Med 2004;32:S87.
72. El-Solh A, Sikka P, Bozkanat E, Jaafar W, Davies J. Morbid obesity in the medical ICU. Chest 2001;120:1989-97.
73. Clagett GP, Anderson FA Jr, Geerts W.. Prevention of venous thromboembolism. Chest 1998; 114:531
74. Wu EC, Barba CA. Current practices in the prophylaxis of venous thromboembolism in bariatric surgery. Obes Surg 2000;10:7-13.
75. Rivers E, Nguyen B, Havstad S, Ressler J, Muzzin A, Knoblich B, *et al*. Early goal-directed therapy in the treatment of severe sepsis and septic shock. N Engl J Med 2001;345:1368-77.
76. Schiffl H, Lang SM, Fischer R. Daily hemodialysis and the outcome of acute renal failure. N Engl J Med 2002;346:305.
77. Bajwa SS, Bajwa SK, Kaur J, Sharma V, Singh A, Singh A, *et al*. Palonosetron: A novel approach to control postoperative nausea and vomiting in day care surgery. Saudi J Anaesth 2011; 5:19-24.

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