# The Crashing Obese Patient

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**KEYWORDS**

• Obesity • Airway • Pharmacology • Sepsis • Trauma

**KEY POINTS**

- Obesity imparts anatomic and physiologic changes that have an impact on resuscitative treatments in critical illness, including airway and ventilator management, pharmacology, and fluid management.
- Obesity is a risk factor for difficult bag-valve mask ventilation and may predict difficult intubation.
- Obese patients are at risk for inaccurate medication dosing, including underdosing and overdosing of resuscitative medications, such as sedatives, paralytics, and antibiotics.
- Obese patients are at risk for under-resuscitation in the setting of critical illness and injury.

## INTRODUCTION

Obesity is defined as a body mass index (BMI) greater than or equal to 30 kg/m$^2$ and can be subdivided into 3 classes (**Table 1**). In the past 3 decades, obesity rates have increased dramatically worldwide.[1] Current obesity rates in the United States are approaching 40%, with increases in rates across ages, races, and social demographics.[1,2] As rates of obesity continue to climb, emergency physicians will increasingly resuscitate critically ill obese patients. Obesity imparts important anatomic and physiologic changes, including alterations to airway anatomy, respiratory mechanics, cardiovascular function, and drug metabolism. The emergency physician should be well acquainted with the challenges faced in the resuscitation of the critically ill or injured obese patient and be equipped with strategies to overcome them.

## ACCESS AND MONITORING

Obesity poses challenges in the most basic aspects of patient care, including venous access and cardiovascular monitoring. A BMI greater than 30 mg/kg$^2$ has been shown to predict difficult intravenous (IV) access.[3] Obscured landmarks and increased tissue depth can complicate both central venous and intraosseous access in obese patients.[4,5] Ultrasound guidance can improve the likelihood of successful placement

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**Table 1**
**Body mass index categorization**

| Category | Body Mass Index ($kg/m^2$) |
|---|---|
| Underweight | <18.5 |
| Normal weight | 18.5 to <25 |
| Overweight | 25 to <30 |
| Obesity class I | 30 to <35 |
| Obesity class II | 35 to <40 |
| Obesity class III | 40 and above |

of a peripheral IV in patients with difficult access.[6] Ultrasound guidance also can improve successful cannulation and complication rates in central venous access in the obese.[7] If available, ultrasound guidance for central venous access placement is recommended.[8]

Accurate blood pressure monitoring can be difficult in obese patients in part due to an increase in arm circumference and an exaggerated conical shape of the upper arm that limit the fit of noninvasive blood pressure cuffs.[9,10] An inappropriately fitting noninvasive blood pressure cuff can provide inaccurate blood pressure readings—an effect that is exaggerated in the critically ill obese patient.[10] When available, a properly fitted upper arm cuff provides the most accurate noninvasive blood pressure measurements followed by a properly fitting wrist cuff.[11] In the critically ill obese patient requiring close and accurate blood pressure monitoring, early arterial access should be considered.

## AIRWAY ANATOMY

Obesity is associated with several important anatomic changes that influence emergent airway management. Neck extension may be limited due to dorsocervical fat deposition and increased circumference. Redundant soft tissues in the oropharynx can lead to airway obstruction, and excess thoracoabdominal fat can limit lung expansion.[12] These changes contribute to the demonstrated association between obesity and difficult bag-valve mask ventilation.[13] The influence of BMI on the rates of difficult intubation is less clear, with studies yielding conflicting results.[14,15] Obesity and obesity-related anatomic changes frequently are included in various difficult airway prediction tools.[16–18] Although it remains unclear whether obesity is an independent risk factor for a difficult airway, the emergency physician should consider this possibility and approach these airways with caution.

## RESPIRATORY PATHOPHYSIOLOGY

Alterations to respiratory physiology also are observed in obese patients. Lung volumes are significantly reduced, including functional residual capacity (FRC), respiratory reserve volume, vital capacity, total lung capacity, and residual capacity.[19] Volume loss increases with BMI, with reductions of 0.5% to 5% observed for each unit increase in BMI.[19] Reductions in FRC are associated with closure of small airways and increased airway resistance.[20] Premature airway closure results in under-ventilated lung space, producing a shunt phenomenon and contributing to rapid desaturation.[21] Additionally, compliance can be limited by decreased lung volumes and excess chest wall adipose. Supine positioning exacerbates reductions in lung volumes.

Obese patients demonstrate higher rates of oxygen consumption and carbon dioxide production that contribute to the adoption of a resting rapid shallow breathing pattern with baseline respiratory rates of 15 to 21 breaths per minute observed in the severely obese.[19,22,23] Obesity also is linked to several distinct respiratory disease states, including obstructive sleep apnea (OSA) and obesity hypoventilation syndrome (OHS).

The cumulative result of these anatomic and physiologic changes in the obese patient is a dramatic reduction in pulmonary reserve with risk for rapid desaturation during intubation and challenges with mechanical ventilator support.

## APPROACH TO THE OBESE AIRWAY

Airway management of the obese patient should begin with effective preoxygenation. Traditional methods of preoxygenation, including face mask oxygen with 100% fractional inspired oxygen concentration and bag-valve mask ventilation, often are insufficient.[22] Noninvasive positive pressure ventilation (NIPPV) can improve preoxygenation and prolong time to desaturation.[22,24] To avoid premature airway closure observed in the supine position, obese patients should be preoxygenated in a seated or semiupright position.[25]

Safe apnea time may be prolonged further through the use of apneic oxygenation, although the utility and best means of delivery are yet to be determined.[26,27] Alterations to routine intubation techniques should be employed to maximize safety in airway management of the obese. A ramped or head-up position during intubation technique can improve laryngoscopic view compared with supine positioning.[28] Ramping consists of supporting a patient's upper back and head with towels, pillows, or specifically designed devices to achieve alignment of the external auditory meatus and the sternal notch. Head-up positioning aims to achieve the same alignment by raising the head of the bed to approximately 25° and supporting the head with additional pillows or towels as needed (**Fig. 1**).

An obese neck can make surgical airway management difficult. Some experts advocate making a partial-thickness, vertical incision to facilitate palpation of deeper structures.[29] If a tracheostomy device is used, care should be taken to choose a device with sufficient length to securely enter the trachea.

## RESPIRATORY SUPPORT

NIPPV is commonly used in the outpatient and perioperative treatment of OSA and OHS.[30,31] Although it is less well studied in the treatment of acute respiratory failure, it is used widely with good success.[32–34] Treatment protocols relying on bilevel


**Fig. 1.** Comparison of 3 intubation positions. (*A*) Sniffing position—patient flat with head elevated. (*B*) Ramped position —patient's head and shoulders elevated. (*C*) Semirecumbent position—head of bed elevated to approximately 25° with further head elevation with towels or pillows.

positive pressure ventilation emphasizing relatively high inspiratory (12–25 cm $H_2O$) and expiratory (5–9 cm $H_2O$) pressures have been described, with success rates approaching those of similar protocols for chronic obstructive pulmonary disease.[33,34] Predictors of failure of NIPPV treatment include pneumonia and mutiorgan dysfunction, whereas idiopathic hypercarbic respiratory failure has been shown to be predictive of success.[32]

Mechanical ventilation support strategies should take into account the altered respiratory physiology of obese patients. Muscle paralysis and sedation can further impair lung function, compliance, and gas exchange through worsened lung volume loss and atelectasis.[35] Improper ventilation strategies can result in significant respiratory or hemodynamic instability.

Obese patients can be supported successfully with both volume-controlled and pressure-controlled ventilation modes, with no single mode demonstrating superiority.[22] Increased airway resistance and decreased compliance can produce inadequate ventilation and resultant respiratory acidosis in pressure-controlled ventilation. Conversely, volume-controlled ventilation carries the risk of barotrauma because high pressures may be required to deliver the specified volume.

The use of low-tidal-volume (6–8 mL/kg) ventilation strategies in patients with acute respiratory distress syndrome (ARDS) is well supported and its use in patients without ARDS is gaining support.[36–38] In volume-controlled modes, tidal volume should be calculated based on ideal body weight (IBW). Use of total body weight (TBW) may result in significant ventilator-associated lung injury from barotrauma.

Obese patients can experience significant alveolar derecruitment and expiratory flow limitation during mechanical ventilation.[22,25,35] Application of positive end-expiratory pressure (PEEP) can improve respiratory mechanics and atelectasis. Obese patients may require higher PEEP than nonobese patients (eg, 10 cm $H_2O$).[22] Obese patients also can demonstrate expiratory flow limitation with resultant air-trapping and auto-PEEP, also referred to as intrinsic PEEP.[35] In this instance, it is important to monitor for the development of auto-PEEP using an expiratory hold maneuver on the ventilator. The application of extrinsic PEEP should be limited to two-thirds of the intrinsic PEEP.[22,25] High PEEP also can precipitate hemodynamic compromise through limitations of venous return, right ventricular output, and pulmonary perfusion.[25]

The increased work of breathing, $CO_2$ production, and oxygen consumption observed in the obese results in a rapid, shallow breathing pattern.[22,23] This resting rate should be mirrored in ventilator settings and monitored for adequate expiration.[22,35]

As with preoxygenation and intubation, positioning is an important component of successful mechanical ventilation of the obese patient. Patients should be kept in a head-elevated position (ie, reverse Trendelenburg) so as to lessen the volume loss observed in supine positioning.[35]

## PHARMACOLOGY

Changes in body composition, cardiac output, and renal and hepatic function can have important impacts on pharmacokinetics and pharmacodynamics of many important medications (**Fig. 2**). The use of weight-based dosing scalars can lead to incorrect dosing of medications through the use of an inappropriate dosing weight. Obese patients often are excluded from pharmaceutical dosing studies, limiting understanding of effective dosing in this population.[25,39,40] At increasing weights, adipose tissue contributes disproportionately to TBW, resulting in larger than expected volumes of

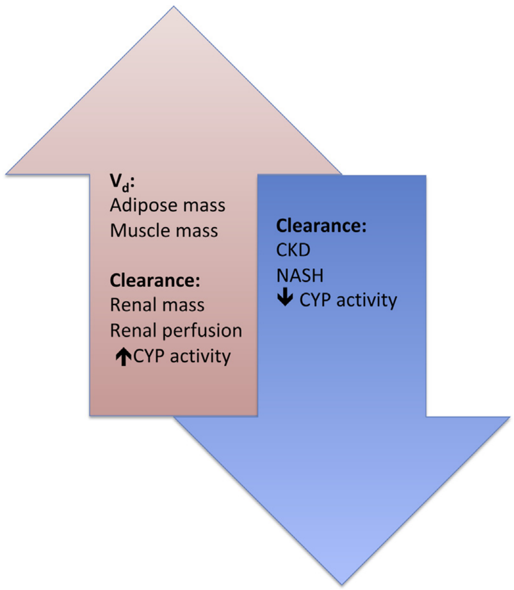


**Fig. 2.** Impacts of obesity on drug pharmacokinetics and pharmacodynamics. CKD, chronic kidney disease; CYP, cytochrome P450; NASH, nonalcoholic steatohepatitis.

distribution ($V_d$) of lipophilic compounds (**Fig. 3**).[39] Alterations in $V_d$ are particularly relevant to emergency physicians, because they have significant impacts on loading doses. Drug clearance has a more significant impact on maintenance doses and dosing intervals.[40] Drug clearance is determined primarily by hepatic and renal functions, both of which can be variably affected by obesity, with an overall trend toward more rapid drug clearance.[41] Several different dosing weights are commonly used in obese patients (**Table 2**). The appropriate dosing weight takes into consideration drug

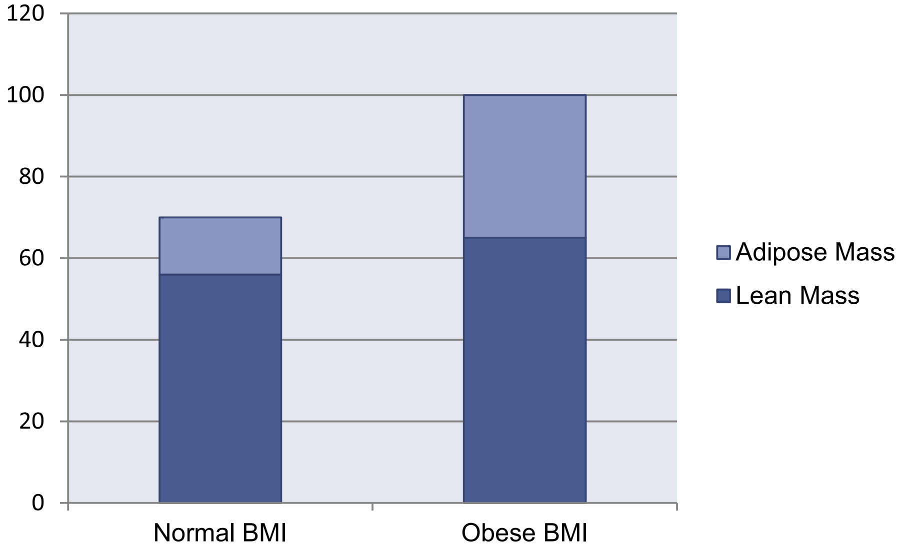


**Fig. 3.** Tissue composition at normal BMI and obese BMI.

**Table 2**
**Dosing weight definitions**

| Weight | Abbreviation | Definition |
|---|---|---|
| Ideal body weight | IBW | The weight at a given height that is associated with the lowest mortality |
| Total body weight | TBW | Measured weight without adjustment |
| Adjusted body weight | AdjBW<br>ABW | IBW + *cf*(TBW − IBW) |
| Lean body mass | LBM | Estimated mass of nonfat tissues, including muscle, bone, and so forth, with various calculations available. |

*Abbreviation: cf*, correction factor, most commonly 0.4.

lipophilicity, protein binding characteristics, and risk of toxicity. In general, more lipophilic compounds are best dosed according to TBW, whereas more hydrophilic compounds are best dosed according to IBW or adjusted body weight (ABW). The following sections highlight particularly relevant resuscitative medications and special cases in obese pharmacology.

### *Sedatives and Paralytics*

Incorrect dosing of sedatives and paralytics in rapid sequence intubation (RSI) can affect intubation conditions adversely. Obese patients frequently experience underdosing of common RSI medications, such as etomidate and succinylcholine.[42] In general, medications used for RSI should be dosed according to TBW or IBW, depending on drug lipophilicity, with some notable exceptions (**Table 3**). Rocuronium, although weakly lipophilic, has been demonstrated to cause prolonged neuromuscular blockade in obese patients when dosed at TBW and, therefore, should be dosed according IBW.[39] Propofol is highly lipophilic and dosing studies have demonstrated that dosing according to IBW during RSI achieves similar sedation goals and intubating conditions, while avoiding cardiovascular complications associated with higher doses.[39] Given the rapid clearance of propofol, however, obese patients may require higher dosing more reflective of TBW when propofol infusions are used for sedation in mechanically ventilated patients or during procedural sedation. Ketamine, another highly lipophilic drug, demonstrates adequate sedation when dosed according to weights between IBW and TBW. Dosing based on estimated lean body mass has demonstrated good anesthetic outcomes.[43] Finally, despite their highly lipophilic nature, benzodiazepines have demonstrated prolonged effects in obese patients when maintenance infusions are dosed according to TBW. Therefore, it is recommended to calculate the initial dose of a benzodiazepine based on TBW, with infusions dosed according to IBW.[44]

### *Analgesics*

Opioid medications are lipophilic and demonstrate large $V_d$. Clearance is variably affected, with some drugs demonstrating prolonged half-lives.[44] This prolonged half-life, coupled with high rates of OSA and OHS, contributes to an increased risk of opiate-related respiratory complications.[39] Dosing of these opioid medications should consider the intended effect and clinical scenario. When used in the peri-intubation time period, consideration should be given to the rapid dispersal to tissues;

**Table 3**
**Weight-based dosing for selected medications**

| Drug Class | High Standard | Total Body Weight | Ideal Body Weight | Lean Body Mass | Adjusted Body Weight |
|---|---|---|---|---|---|
| RSI Medications | | Etomidate<br>Succinylcholine<br>Midazolam<br>Fentanyl | Propofol<br>Rocuronium<br>Vecuronium | Ketamine | |
| Antibiotics | Cephalosporins<br>Carbapenems<br>Fluoroquinolones | Vancomycin | | | Aminoglycosides |
| Cariovascular Medications | | Calcium channel blockers<br>Epinephrine<br>Norepinephrine | β-Blockers<br>Lidocaine<br>Procainamide<br>Digoxin | | |

dosing based on TBW likely is beneficial.[25] When dosed for analgesia, dosing should reflect IBW so as to minimize risk for respiratory complications.[39,44,45]

### *Antibiotics*

Early, appropriate antibiotic administration is a critical component of sepsis care.[46] It is incumbent on emergency physicians to accurately dose antibiotics to expediently achieve therapeutic levels. As with other drug classes, drug-specific dosing recommendations for antibiotics in obese patients are sparse. Vancomycin is among the best-studied antibiotics in obese patients. Generally, the initial dose of vancomycin is best dosed according to TBW, owing to relatively large $V_d$; however, increases in $V_d$ do not scale proportionally with BMI; therefore, patients with very large BMIs may require lower doses.[47] Subsequent doses are best determined via therapeutic drug monitoring, because Cl can be significantly altered by both obesity and sepsis-related organ dysfunction.[47] Although evidence of improved therapeutic drug levels with this dosing strategy have been reported for decades, underdosing is still observed among many obese patients.[48] A study of more than 2500 ED patients demonstrated an 8-fold increase in the likelihood of underdosing of vancomycin for every 10 kg increase in body weight.[48]

Aminoglycosides have also been relatively well studied in the obese population. The most commonly recommended dosing strategy is based on an ABW, in which a correction factor, typically 0.4, is used for excess weight above IBW.[47]

Penicillins, cephalosporins, carbapenems, and fluoroquinolones require relatively little dosing adjustment and should be dosed at the higher end of the standard recommended range.[47]

Antibiotic therapy for the critically ill obese patient can be challenging, because multiple patient and disease factors can alter the pharmacokinetics and pharmacodynamics of commonly used medications. Where available, therapeutic drug monitoring can play a crucial role in treatment.[40,47]

### *Cardiovascular Medications*

Unlike many of the medications discussed previously, cardiovascular drugs like β-adrenergic receptor blockers, lidocaine, procainamide, and digoxin are relatively hydrophilic and have little alteration in their $V_d$ with increasing BMI. Because their $V_d$ are relatively unchanged, dosing according to IBW is recommended.[25,41] Conversely, calcium channel blockers are more lipophilic and should be dosed according to TBW.[25]

Both weight-based and non–weight-based dosing strategies for vasoactive medications like norepinephrine have demonstrated similar efficacy in achieving clinically meaningful outcomes like mean arterial blood pressure goal and mortality.[49,50] When weight-based dosing strategies are used, TBW should be used for dosing calculations.

## CONDITION-SPECIFIC CONSIDERATIONS

### *Cardiovascular Disease and Cardiac Arrest*

Obesity is a significant contributor to cardiovascular disease mortality.[1] Even in the absence of common congestive heart failure risk factors, obesity can lead directly to the development of heart failure. This often is termed, *obesity cardiomyopathy*. In this condition, hypertension, OSA, and OHS lead to alterations in cardiac structure and function, ultimately producing a reduction in ejection fraction. Despite the well-documented association with cardiovascular disease and cardiovascular death, the impact of obesity on outcome after cardiac arrest is less clear.[51,52] Obesity does have measurable impacts on the delivery of high-quality cardiopulmonary resuscitation. Increased abdominal girth may shift the optimal location for chest compressions to a more cephalad position than advanced cardiac life support guidelines would suggest.[53] Additionally, the force required to generate appropriate chest compression depth may lead to more rapid compressor fatigue.[54]

### *Trauma*

In addition to increased rates of illness, obese patients also demonstrate higher rates of traumatic injury. When compared with patients with normal BMI, overweight individuals have an increased rate of injury of approximately 10%, whereas the risk of obese individuals can be as high as 48%[55] Obese patients demonstrate altered patterns of injury, particularly in blunt trauma, with fewer liver and head injuries but increased rates of extremity and thoracic injury.[56] Although increased abdominal adipose tissue may protect from intra-abdominal injury, increased inertia from excess weight may contribute to more severe extremity injury and the increased frequency of severe extremity injury from relatively low mechanism trauma.[57]

Many studies have demonstrated worsened morbidity in obese trauma patients.[58–61] Mortality results are mixed, with the overall trend favoring worsened mortality with high BMI.[58–61] Increased morbidity is driven largely by posttrauma complications, including infection, respiratory failure, venous thromboembolism, and multiorgan failure.[58–61] Increased incidence of respiratory disease, airway obstruction, chronic inflammation, and poor glycemic control may contribute to these complications.[57,61] The poor reliability of traditional resuscitation endpoints like central venous pressure monitoring can result in under-resuscitation of obese trauma patients.[61] Other contributors to worsened outcomes span the spectrum of care of the trauma patient, including challenges with prehospital care (limitations of routine equipment, prolonged extrication times),[57] limitations of diagnostic imaging,[62] and challenges in operative management with intraoperative patient positioning, table size, and tissue depth at operative sites.[57]

Care of the injured obese patient should focus on aggressive supportive care, including respiratory support, appropriate perioperative antibiotic dosing, and aggressive early resuscitative care.

### *Sepsis*

The link between obesity and chronic disease, morbidity, and decreased life expectance has been well established.[1] The effects of obesity in critical illness, however, including sepsis, remain unclear. A systematic review noted significant heterogeneity in the available literature and demonstrated no clear effect of obesity on mortality risk in sepsis.[63] Adipose produces a variety of inflammatory mediators leading to a state chronic inflammatory state.[64] It has been suggested that this may lead to an altered host response during infection, conferring a protective effect.[65]

Despite inconsistent effects on mortality, obesity has a well-demonstrated impact on morbidity. Hospital and ICU lengths of stay, cost of care, and ventilator dependence frequently are increased in the obese.[64,66–68] Additionally, several studies note that the average age of obese cohorts are younger than those of normal BMI or underweight BMI, suggesting a potential increased risk of developing sepsis in this group.[66,67] Obese patients also have altered patterns of infection compared with their normal and underweight counterparts, with fewer incidents of pulmonary infections and increased rates of skin and soft tissue infections and of gram-positive infections.[64–66]

The Surviving Sepsis Campaign recommends a weight-based fluid resuscitation strategy but does not specify a dosing weight.[46] Obese patients are frequently under-resuscitated compared with normal weight and underweight patients.[64,68,69] ABW with a correction factor of 40% for body weight in excess of IBW has been associated with improved morbidity and mortality.[68] Prospective studies are needed in order to determine the best fluid resuscitation strategy in the obese patient with sepsis-induced hypotension. Early administration of appropriately dosed antibiotics remains critically important in the care of the septic obese patient.

## SUMMARY

Obesity imparts important anatomic and physiologic changes that have an impact on resuscitative measures in critical illness. Obesity poses significant challenges to the most basic aspects of resuscitative care, including IV access and cardiovascular monitoring. Invasive procedures like central venous and arterial access may be necessary to provide appropriate monitoring and treatment. Altered airway anatomy and respiratory mechanics contribute to a decrease in respiratory reserve and can contribute to rapid desaturation during RSI. Although it remains unclear if obesity is an independent risk factor for difficult intubation, preparing for a potentially difficult intubation is prudent because obesity and obesity-associated conditions frequently are considered in difficult airway scoring systems. Emergency physicians should be cognizant of altered pharmacokinetics in the obese and choose appropriate dosing weights based on drug and patient characteristics and therapeutic intent.

## DISCLOSURE

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## REFERENCES

1. Afshin A, Forouzanfar MH, Reitsma MB, et al. Health effects of overweight and obesity in 195 countries over 25 years. N Engl J Med 2017;377(1):13–27.
2. Flegal KM, Kruszon-Moran D, Carroll MD, et al. Trends in obesity among adults in the United States, 2005 to 2014. JAMA 2016;315(21):2284–91.
3. Sebbane M, Claret PG, Lefebvre S, et al. Predicting peripheral venous access difficulty in the Emergency Department using body mass index and a clinical evaluation of venous accessibility. J Emerg Med 2013;44(2):299–305.

4. Helwani M, Saied N, Ikeda S. Accuracy of anatomical landmarks in locating the internal jugular vein cannulation site among different levels of anesthesia trainees. J Educ Perioper Med 2008;10(2):E050.
5. Kehrl T, Becker BA, Simmons DE, et al. Intraosseous access in the obese patient: assessing the need for extended needle length. Am J Emerg Med 2016;34(9):1831–4.
6. Costantino TG, Parikh AK, Satz WA, et al. Ultrasonography-guided peripheral intravenous access versus traditional approaches in patients with difficult intravenous access. Ann Emerg Med 2005;46(5):456–61.
7. Brusasco C, Corradi F, Zattoni PL, et al. Ultrasound-guided central venous cannulation in bariatric patients. Obes Surg 2009;19(10):1365–70.
8. Brass P, Hellmich M, Kolodziej L, et al. Ultrasound guidance versus anatomical landmarks for internal jugular vein catheterization. Cochrane Database Syst Rev 2015;(1):CD006962.
9. Anast N, Olejniczak M, Ingrande J, et al. The impact of blood pressure cuff location on the accuracy of noninvasive blood pressure measurements in obese patients: an observational study. Can J Anaesth 2016;63(3):298–306.
10. Araghi A, Bander JJ, Guzman JA. Arterial blood pressure monitoring in overweight critically ill patients: invasive or noninvasive? Crit Care 2006;10(2):R64.
11. Irving G, Holden J, Stevens R, et al. Which cuff should I use? Indirect blood pressure measurement for the diagnosis of hypertension in patients with obesity: A diagnostic accuracy review. BMJ Open 2016;6(11):1–9.
12. Isono S. Obesity and obstructive sleep apnoea: Mechanisms for increased collapsibility of the passive pharyngeal airway. Respirology 2012;17(1):32–42.
13. Langeron O, Masso E, Huraux C, et al. Prediction of difficult mask ventilation. Anesthesiology 2000;92(5):1229–36.
14. Wang T, Sun S, Huang S. The association of body mass index with difficult tracheal intubation management by direct laryngoscopy: A meta-analysis. BMC Anesthesiol 2018;18(1):1–13.
15. Moon TS, Fox PE, Somasundaram A, et al. The influence of morbid obesity on difficult intubation and difficult mask ventilation. J Anesth 2019;33(1):96–102.
16. Wilson ME, Spiegelhalter D, Robertson JA, et al. Predicting difficult intubation. Br J Anaesth 1988;61(2):211–6.
17. Kuzmack E, Inglis T, Olvera D, et al. A novel difficult-airway prediction tool for emergency airway management: validation of the HEAVEN criteria in a large air medical cohort. J Emerg Med 2018;54(4):395–401.
18. Reed MJ, Dunn MJG, McKeown DW. Can an airway assessment score predict difficulty at intubation in the emergency department? Emerg Med J 2005;22(2):99–102.
19. Salome CM, King GG, Berend N. Physiology of obesity and effects on lung function. J Appl Physiol 2010;108(1):206–11.
20. Sebastian JC. Respiratory physiology and pulmonary complications in obesity. Best Pract Res Clin Endocrinol Metab 2013;27(2):157–61.
21. Benumof J, Dagg RBR. Critical hemoglobin desaturation will occur before return to an unparalyzed state following 1 mg/kg intravenous succinylcholine. Anesthesiology 1997;97(4):979–82.
22. De Jong A, Chanques G, Jaber S. Mechanical ventilation in obese ICU patients: From intubation to extubation. Crit Care 2017;21(1):1–8.
23. Chlif M, Keochkerian D, Choquet D, et al. Effects of obesity on breathing pattern, ventilatory neural drive and mechanics. Respir Physiol Neurobiol 2009;168(3):198–202.

24. Baillard C, Fosse JP, Sebbane M, et al. Noninvasive ventilation improves preoxygenation before intubation of hypoxic patients. Am J Respir Crit Care Med 2006;174(2):171–7.
25. Parker BK, Manning S, Winters ME. The crashing obese patient. West J Emerg Med 2019;20(2):323–30.
26. Heard A, Toner AJ, Evans JR, et al. Apneic oxygenation during prolonged laryngoscopy in obese patients: A randomized, controlled trial of buccal RAE tube oxygen administration. Anesth Analg 2017;124(4):1162–7.
27. Ramachandran SK, Cosnowski A, Shanks A, et al. Apneic oxygenation during prolonged laryngoscopy in obese patients: a randomized, controlled trial of nasal oxygen administration. J Clin Anesth 2010;22(3):164–8.
28. Collins JS, Lemmens HJM, Brodsky JB, et al. Laryngoscopy and morbid obesity: A comparison of the “sniff” and “ramped” positions. Obes Surg 2004;14(9): 1171–5.
29. Freeman BD. Tracheostomy update: when and how. Crit Care Clin 2017;33(2): 311–22.
30. Pierce AM, Brown LK. Obesity hypoventilation syndrome: Current theories of pathogenesis. Curr Opin Pulm Med 2015;21(6):557–62.
31. Chau EHL, Lam D, Wong J, et al. Obesity hypoventilation syndrome: a review of epidemiology, pathophysiology, and perioperative considerations. Anesthesiology 2012;117(1):188–205.
32. Lemyze M, Taufour P, Duhamel A, et al. Determinants of noninvasive ventilation success or failure in morbidly obese patients in acute respiratory failure. PLoS One 2014;9(5):5–11.
33. Carrillo A, Ferrer M, Gonzalez-Diaz G, et al. Noninvasive ventilation in acute hypercapnic respiratory failure caused by obesity hypoventilation syndrome and chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2012; 186(12):1279–85.
34. Masa JF, Utrabo I, Gomez de Terreros J, et al. Noninvasive ventilation for severely acidotic patients in respiratory intermediate care units: Precision medicine in intermediate care units. BMC Pulm Med 2016;16(1):1–13.
35. Junhasavasdikul D, Telias I, Grieco DL, et al. Expiratory Flow Limitation During Mechanical Ventilation. Chest 2018;154(4):948–62.
36. Petrucci N, De Feo C. Lung protective ventilation strategy for the acute respiratory distress syndrome. Cochrane Database Syst Rev 2013;2013(2):CD003844.
37. De Jong A, Verzilli D, Jaber S. ARDS in obese patients: specificities and management. Crit Care 2019;23(1):74.
38. Fuller BM, Mohr NM, Drewry AM, et al. Lower tidal volume at initiation of mechanical ventilation may reduce progression to acute respiratory distress syndrome: A systematic review. Crit Care 2013;17(1):R11.
39. Ingrande J, Lemmens HJ. Dose adjustment of anaesthetics in the morbidly obese. Br J Anaesth 2010;105(Suppl):16–23.
40. Barras M, Legg A. Drug dosing in obese adults. Aust Prescr 2017;40(5):189–93.
41. Blouin RA, Warren GW. Pharmacokinetic considerations in obesity. J Pharm Sci 1999;88(1):1–7.
42. Bhat R, Mazer-Amirshahi M, Sun C, et al. Accuracy of rapid sequence intubation medication dosing in obese patients intubated in the ED. Am J Emerg Med 2016; 34(12):2423–5.
43. Wulfsohn NL. Ketamine dosage for induction based on lean body mass. Anesth Analg 1972;51(2):299–305.

44. De Baerdemaeker LEC, Mortier EP, Struys MMRF. Pharmacokinetics in obese patients. Contin Educ Anaesthesia, Crit Care Pain 2004;4(5):152–5.
45. Nelson JA, Loredo JS, Acosta JA. The obesity-hypoventilation syndrome and respiratory failure in the acute trauma patient. J Emerg Med 2011;40(4):e67–9.
46. Rhodes A, Evans LE, Alhazzani W, et al. Surviving sepsis campaign: international guidelines for management of sepsis and septic shock: 2016. Intensive Care Med 2017;43(3):304–77.
47. Meng L, Mui E, Holubar MK, et al. Comprehensive Guidance for Antibiotic Dosing in Obese Adults. Pharmacotherapy 2017;37(11):1415–31.
48. Fuller BM, Mohr N, Skrupky L, et al. Emergency Department vancomycin use: dosing practices and associated outcomes. J Emerg Med 2013;44(5):910–8.
49. Adams C, Tucker C, Allen B, et al. Disparities in hemodynamic resuscitation of the obese critically ill septic shock patient. J Crit Care 2017;37:219–23.
50. Radosevich JJ, Patanwala AE, Erstad BL. Norepinephrine dosing in obese and nonobese patients with septic shock. Am J Crit Care 2016;25(1):27–32.
51. Shahreyar M, Dang G, Waqas Bashir M, et al. Outcomes of In-hospital cardiopulmonary resuscitation in morbidly obese patients. JACC Clin Electrophysiol 2017;3(2):174–83.
52. Geri G, Savary G, Legriel S, et al. Influence of body mass index on the prognosis of patients successfully resuscitated from out-of-hospital cardiac arrest treated by therapeutic hypothermia. Resuscitation 2016;109:49–55.
53. Lee J, Oh J, Lim TH, et al. Comparison of optimal point on the sternum for chest compression between obese and normal weight individuals with respect to body mass index, using computer tomography: A retrospective study. Resuscitation 2018;128:1–5.
54. Secombe PJ, Sutherland R, Johnson R. Morbid obesity impairs adequacy of thoracic compressions in a simulation-based model. Anaesth Intensive Care 2018;46(2):171–7.
55. Finkelstein EA, Chen H, Prabhu M, et al. The relationship between obesity and injuries among U.S. adults. Am J Health Promot 2007;21(5):460–8.
56. Stroud T, Bagnall NM, Pucher PH. Effect of obesity on patterns and mechanisms of injury: Systematic review and meta analysis. Int J Surg 2018;56:148–54.
57. Spitler CA, Hulick RM, Graves ML, et al. Obesity in the Polytrauma Patient. Orthop Clin North Am 2018;49(3):307–15.
58. Newell MA, Bard MR, Goettler CE, et al. Body mass index and outcomes in critically injured blunt trauma patients: weighing the impact. J Am Coll Surg 2007;204(5):1056–61.
59. Byrnes MC, McDaniel MD, Moore MB, et al. The effect of obesity on outcomes among injured patients. J Trauma 2005;58(2):232–7.
60. Bochicchio GV, Joshi M, Bochicchio K, et al. Impact of obesity in the critically ill trauma patient: a prospective study. J Am Coll Surg 2006;203(4):533–8.
61. Winfield RD, Delano MJ, Lottenberg L, et al. Traditional resuscitative practices fail to resolve metabolic acidosis in morbidly obese patients after severe blunt trauma. J Trauma 2010;68(2):317–30.
62. Modica MJ, Kanal KM, Gunn ML. The obese emergency patient: imaging challenges and solutions. Radiographics 2011;31(3):811–23.
63. Trivedi V, Bavishi C, Jean R. Impact of obesity on sepsis mortality: A systematic review. J Crit Care 2015;30(3):518–24.
64. Wacharasint P, Boyd JH, Russell JA, et al. One size does not fit all in severe infection: Obesity alters outcome, susceptibility, treatment, and inflammatory response. Crit Care 2013;17(3):R122.

65. Falagas ME, Kompoti M. Obesity and infection. Lancet Infect Dis 2006;6(7): 438–46.
66. Arabi YM, Dara SI, Tamim HM, et al. Clinical characteristics, sepsis interventions and outcomes in the obese patients with septic shock: An international multicenter cohort study. Crit Care 2013;17(2):R72.
67. Nguyen AT, Tsai CL, Hwang LY, et al. Obesity and mortality, length of stay and hospital cost among patients with sepsis: A nationwide inpatient retrospective cohort study. PLoS One 2016;11(4):1–12.
68. Taylor SP, Karvetski CH, Templin MA, et al. Initial fluid resuscitation following adjusted body weight dosing is associated with improved mortality in obese patients with suspected septic shock. J Crit Care 2018;43:7–12.
69. Kuttab HI, Lykins JD, Hughes MD, et al. Evaluation and Predictors of Fluid Resuscitation in Patients With Severe Sepsis and Septic Shock. Crit Care Med 2019; 47(11):1.