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SPECIAL FEATURE

Critical Care in the Obese Patient

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Dr. Thompson reports no financial relationships relevant to this field of study.

Obesity is a growing epidemic that affects the health of millions of Americans and will require healthcare systems to anticipate growth in the numbers of obese patients. The National Institutes of Health and World Health Organization classify body mass index (BMI) in kg/m^2 as overweight (25.0-29.9), class I obesity (30.0-34.9), class II obesity (35.0-39.9), and class III or extreme obesity (> 40.0).[1] BMIs of > 40.0 and > 50.0 have been classified previously as morbid obesity and super morbid obesity, respectively.[2] Trends in obesity based on the National Health and Nutrition Examination Survey (NHANES) suggest an increase in age-adjusted prevalence of obesity in the United States from 33.7% in 2007-2008 to 39.6% in 2015-2016.[3] Given that obesity carries with it an increased risk of medical conditions that can result in an intensive care unit (ICU) admission (e.g., coronary artery disease, diabetes, stroke, pulmonary embolism),[4] the prevalence of obesity in those admitted to the ICU also has risen, with rates varying from approximately 20% to 36.5%.[5,6] With the growth in the numbers of patients undergoing bariatric surgery (252,000 U.S. and Canadian cases in 2018),[7] more patients are being treated in the ICU for complications of that procedure. In addition, obesity is an independent risk factor for mortality in the ICU.[8] This article will highlight some important practical aspects of care that arise in the management of critically ill obese patients, along with the unique physiology resulting from obesity.

RESPIRATORY MANAGEMENT

Obesity results in an alteration of the lung volumes and capacities, including a significant drop in functional residual capacity (FRC), vital capacity (VC), and tidal volume, while the residual volume (RV) and total lung capacity (TLC) are less affected.[9] The expiratory reserve volume (ERV = FRC-RV) drops exponentially with increasing BMI, with the effect most pronounced in those with more central obesity. Even a BMI of

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30 kg/m^2 can reduce ERV by 53% compared to normal BMI. A 1% decrease in ERV can be expected for every additional unit of BMI.[10] As a result of the lower tidal volumes and higher metabolic rate, the respiratory rate typically is higher.[9,11]

As might be expected, total respiratory compliance is lower by as much as two-thirds in obese patients, with contributions from fat deposition in the chest wall and abdomen, incomplete relaxation of respiratory muscles, and lower lung compliance. In fact, respiratory compliance appears to be inversely related to BMI,[12] resulting from increased pulmonary blood volumes, closure of dependent airways, and increased alveolar surface tension at lower FRC.[9] With rising BMI, the FRC can drop below the closing volume at which basilar alveoli start to collapse, resulting in ventilation perfusion (V/Q) mismatch and hypoxemia. The drop in FRC with anesthesia of normal BMI subjects is even more pronounced in patients with obesity.[11] Similarly, obesity can dramatically decrease the time to desaturation in a pre-oxygenated patient who is apneic, such as during induction of anesthesia.[11]

Airway resistance also is increased in obese patients, in part because of the lower FRC.[9] The lower respiratory compliance and higher airway resistance seen in these patients leads to an increase in the work of breathing. As a result, the oxygen cost of breathing at rest (the percentage of body oxygen consumption dedicated to respiratory muscle work) rises from a normal of less than 3% in nonobese subjects to a level that is four- to 10-fold higher in obese patients.[9]

Considering the respiratory physiologic changes in obesity noted above, methods to improve ventilation in these patients include reverse Trendelenburg position at 30-45 degrees,[13,14] prophylactic bilevel positive airway pressure in the postoperative period,[15] and higher levels of positive end-expiratory pressure (PEEP) up to 10 or even 15 cm H_2O in those on mechanical ventilation.[16,17] Recruitment maneuvers followed by PEEP 10 cm H_2O during anesthesia and paralysis of obese patients likely is better than either alone in improving atelectasis, although the generalizability of all of these anesthesia studies to the ICU remains unclear.[16,18,19] At present, while it is not unreasonable to trial a patient on higher PEEP followed by a reassessment of oxygenation, there are no strong data to indicate that all obese patients should be treated with higher PEEP. Similarly, there are insufficient data to suggest the routine use of recruitment maneuvers in obese patients.[20]

Short-term anesthesia studies on ventilator mode in obese patients have been conducted and suggest special considerations for the obese patient with ARDS. In a secondary analysis of the ARDSNet trial, O'Brien found no difference in outcome in acute lung injury when comparing normal, overweight, and obese subjects who were ventilated with 6 mL/kg predicted body weight (PBW), knowing that the study excluded patients with a weight (kg):height (cm) ratio ≥ 1.[21] To achieve a tidal volume of 6 mL/kg PBW, one should aim for a plateau airway pressure of < 30. However, without data to support them, some would argue for the acceptance of a higher plateau pressure if needed, knowing that this does not necessarily translate into a high transpulmonary pressure, but rather reflects the lower chest wall compliance and higher abdominal pressure. When available, use of esophageal manometry can help to assess the contribution of the chest wall to total respiratory compliance.

An "obesity paradox" has been described when looking at mortality in ARDS and septic shock[5,22] in that obese patients tend to have a better outcome compared to normal weight or underweight patients. However, further analysis has demonstrated that differences exist in the demographics (age, APACHE III score, types of infection) and treatment (intravenous [IV] fluid volumes used for resuscitation, antibiotic doses) of these groups and may better explain the differences in mortality. Further work needs to be done. It may be that we could improve the mortality of nonobese patients by studying some of these apparent paradoxes. Mogri et al provide a summary of practical tips for ventilating obese patients.[23]

OBSTRUCTIVE SLEEP APNEA AND OBESITY HYPOVENTILATION SYNDROME

The possibility of obstructive sleep apnea (OSA) or obesity hypoventilation syndrome

(OHS) must be considered in obese patients, especially when managing the airway. Because many OSA patients remain undiagnosed,[24] screening tools such as the STOP BANG questionnaire, which gives the patient a point for each of eight criteria (snoring, tiredness, observed apnea, pressure [hypertension], BMI, age, neck circumference, gender), are available. It was developed originally as a presurgical screening tool but has been expanded since for use in a broader population.[25] The prevalence of moderate to severe sleep apnea in the general population is near 4% for women and 9% for men,[26] and it increases to 48% for people with a BMI ≥ 30 kg/m^2 in one study.[27] The diagnosis of OSA puts patients at higher risk of acute hypercarbic and hypoxic respiratory failure, difficult intubation, pulmonary embolism, delirium, cardiac ischemia, arrhythmia, and other complications, especially in the postoperative period.[24]

Similarly, compared to those with OSA alone, those with OHS are at higher risk of complications such as respiratory failure, heart failure, prolonged intubation, hypertension, insulin resistance, pulmonary hypertension, postoperative ICU transfer, and longer ICU and hospital lengths of stay, especially in the postoperative period.[28] A BMI > 30 kg/m^2 and $PaCO_2$ > 45 mmHg are critical elements of the diagnosis. While a diagnosis of OHS cannot be made when other etiologies of hypercarbia such as use of opiates and sedatives are present, precautions should be taken in these patients, including close respiratory monitoring and limiting the use of sedating medications when possible, especially since patients with untreated sleep-disordered breathing have an increased sensitivity to opiates. The prevalence is estimated to be 10-20% in obese patients with OSA and 0.15-0.3% in the general adult population.[29] The risk of OHS goes up with BMI, and some estimate it at 50% in those with BMI ≥ 50 kg/m^2. The combination of OSA, BMI > 30 kg/m^2, and serum bicarbonate ≥ 28 mmol/L puts the patient at significantly higher risk of OHS, while a baseline serum bicarbonate ≤ 27 mmol/L makes a diagnosis of OHS much less likely (good negative predictive value).[29]

NUTRITION

Despite sufficient caloric intake, many obese patients remain malnourished, with a high rate of micronutrient deficiencies. In those who have undergone bariatric surgery, the American Society for Parenteral and Enteral Nutrition (ASPEN) recommends: initiation of thiamine prior to giving dextrose-containing IV fluids; evaluation for and treatment of micronutrient deficiencies, such as calcium, thiamine, vitamin B12, fat-soluble vitamins (A, D, E, K), and folate; and evaluation and treatment for deficiencies in the trace minerals iron, selenium, zinc, and copper.[30]

Obese patients with critical illness, similar to nonobese patients, are at risk for loss of muscle mass. Thus, hypocaloric, high-protein nutritional support often is recommended in these patients during times of critical illness, except in situations where high protein intake may be detrimental (progressive renal insufficiency, severe hepatic insufficiency, diabetic ketoacidosis, hypoglycemia, age > 60 years, or severe immune compromise).[30] Indirect calorimetry, when available, is the best means of matching caloric replacement with expenditure. Short of this, the best equations for calculating caloric and protein needs in these patients are debated, making consultation with the nutritional support team critical.[31]

VENOUS THROMBOEMBOLISM

The risk of venous thromboembolism (VTE) is approximately 2.5 times higher in obese patients compared to nonobese patients, potentially due to altered levels of platelet activator inhibitor, plasma fibrinogen, factors VII and VIII, and von Willebrand factor; increased platelet activation; decreased mobility; venous stasis; and increased thrombin generation.[32,33] Obese patients are more likely to have postoperative thromboembolism. Pulmonary embolism is the most common cause of postoperative mortality after bariatric surgery, accounting for approximately 50% of deaths in some studies.[34,35]

For deep venous thrombosis (DVT) prophylaxis with enoxaparin or dalteparin, standard doses (30 mg every 12 hours and 5,000 U once daily, respectively) are recommended up to BMI 40 kg/m^2, with a 30% increase in dose for those ≥ 40 kg/m^2. Even higher doses (enoxaparin 60 mg every 12 hours) are recommended by some for patients with BMI ≥ 50 kg/m^2 who are undergoing bariatric surgery and are at high risk of DVT.[36,37] Similarly, higher doses of unfractionated heparin for DVT prophylaxis are recommended by some experts.[38]

The diagnosis of VTE can be challenging in the obese patient, given the diminished quality of images seen as the result of higher BMI across all imaging modalities.[33,35] Computed tomography (CT) remains the best modality for diagnosing PE. Lower extremity venous duplex exam remains the imaging modality of choice for DVT. Rather than using the typical linear ultrasound probe, a curvilinear ultrasound probe with 2-3 MHz frequency may result in better image quality.[35]

Treatment for DVT and pulmonary embolism (PE) in obese patients is similar to treatment in nonobese patients, with a few caveats. When using unfractionated heparin, using actual body weight rather than ideal body weight is recommended.[39,40] Similarly, the American College of Chest Physicians (ACCP) Guidelines for

anticoagulation suggest that weight-based (actual body weight) dosing for low molecular weight heparin (LMWH) is preferred over fixed dosing for obese patients; studies are limited in patients with weights > 144 kg (enoxaparin) and > 190 kg (dalteparin).[41] For those with weights greater than these, no upper dose limit is recommended, but closer monitoring, potentially with Factor Xa levels, may be indicated.

Treatment recommendations for DVT and PE with the direct oral anticoagulants (DOACs) in those with BMI 40 kg/m^2 or less are no different than in nonobese patients. However, DOACs generally are not recommended for use in patients with BMI ≥ 40 kg/m^2 or weight > 120 kg because of the limited clinical data on safety and efficacy. If DOACs are used in those with BMI ≥ 40 kg/m^2, it is suggested that drug-specific peak and trough levels (anti-Factor Xa, ecarin time, or dilute thrombin time, depending on the agent, or mass spectrometry drug levels) be monitored, limiting their use for many centers.[42] As more data emerge on the safety and efficacy of these agents in patients with BMI > 40 kg/m^2, the guidelines for use of DOACs in this population likely will evolve.[43]

PHARMACOLOGY

There is a paucity of research and information regarding drug dosing in this population, so consultation with the pharmacy service to assist with drug dosing and pharmacokinetics often is beneficial. Adjusted dosing of medications often requires the clinician to consider several theoretical considerations besides the usual renal and hepatic metabolism of the medication,[44,45] including weight-based dosing. Weight-based dosing must consider whether the medication is best dosed on actual body weight (ABW) vs. ideal (IBW) vs. lean (LBW) vs. adjusted (ABWadj) body weight, knowing that one drug may require loading based on one weight criteria and maintenance dosing based on another weight criteria. Debate continues regarding the best size descriptor to use when calculating doses of renally cleared medications, and it is important to know which equations investigators used in the original dosing trials.[46] Obesity may affect drug pharmacokinetics in several ways, including:

- Absorption: increased oral absorption due to increased gastric emptying, decreased subcutaneous absorption, failure of intramuscular (IM) administration due to short needles.
- Volume of distribution (Vd): increased Vd for lipophilic drugs. Thus, lipophilic medications often are loaded based on ABW while hydrophilic medications are dosed more often on LBW or IBW.
- Metabolism through increased P450 2E1 activity and other effects.
- Elimination: longer half-life of lipophilic drugs, increased glomerular filtration rate in obese patients with normal renal function, more difficulty calculating creatinine clearance in obesity and critical illness.

MANAGEMENT OF THE BARIATRIC SURGERY PATIENT

Because of the obesity epidemic, the number of patients undergoing bariatric surgery has grown, especially since it is one of the most effective treatments now available for the treatment of obesity and its complicating comorbidities. Because it is not uncommon for these patients to have complications requiring an ICU stay, a discussion of critical care in the obese patient would not be complete without some mention of bariatric surgery and its complications. As noted in the introduction, approximately 252,000 bariatric procedures were done in the United States and Canada in 2018.[7] Of these, 61% were sleeve gastrectomy (SG), 17% were roux-en-Y gastric bypass (RYGB) procedures, 1% were gastric banding, 15% were revisions of prior surgeries, and the rest were much less commonly performed procedures. The mechanism of weight loss with SG is primarily restriction in the size of the stomach, while the RYGB not only results in restriction of gastric size, but also malabsorption, which needs to be considered when delivering enteral feedings in the ICU.

Mortality rates generally are < 1% after surgery, and < 10% of patients will require ICU admission. The most common complications leading to mortality and to ICU admission are PE, anastomotic leak, and cardiac events. The type of surgery affects the risk of complications and ICU admission, with fewer ICU admissions seen with laparoscopic compared to open procedures, and fewer seen with SG and gastric banding compared to RYGB.[47] Additional factors associated with higher rates of ICU admission include age > 50 years, BMI ≥ 60 kg/m^2, and the need for reoperation.

After PE, anastomotic leak is the second most common cause of preventable death after bariatric surgery. It occurs in up to 5% of patients, often leading to sepsis and carrying with it a mortality rate of 6-17%. Mortality rates are highest in those with a delay in diagnosis.[47] Upper gastrointestinal contrast examination with water-soluble oral contrast is the investigation of choice to diagnose anastomotic leak in bariatric surgery patients.[35,48] Small bowel obstruction, especially after RYGB and often from anastomotic stricture, internal hernia, and volvulus, is another relatively common postoperative complication that is best imaged by CT scan. Gastrointestinal bleeding is not uncommon, occurring in 1-2% of patients after RYGB, often at a staple line.

SUMMARY

Our healthcare system has made great strides in the care of many chronic diseases. Unfortunately, we continue

to see a growing number of patients with obesity, the disease at the root of many of the chronic diseases facing our patients. As critical care practitioners, we will continue to see these patients in our ICU more frequently, requiring us to manage the unique medical problems and physiology seen in this population. Much of the data described earlier are based on anesthesia studies and studies of the otherwise healthy obese patient, while few studies have been done assessing the obese patient in the ICU. Thus, critical care of obese patients is an area ripe for further research as we face this challenge in the future. ■

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ABSTRACT & COMMENTARY

Pressure Support vs. T-Piece Trials for Successful Extubation: An End to the Controversy?

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SYNOPSIS: In a randomized clinical trial of 1,153 adults who were ready for weaning after at least 24 hours of mechanical ventilation, researchers found that a spontaneous breathing trial with 30 minutes of pressure support ventilation compared with two hours of T-piece ventilation led to significantly higher successful extubation rates.

SOURCE: Subirà C, Hernández G, Vásquez A, et al. Effect of pressure support vs T-piece ventilation strategies during spontaneous breathing trials on successful extubation among patients receiving mechanical ventilation: A randomized clinical trial. *JAMA* 2019;321: 2175-2182.

Daily spontaneous breathing trials (SBTs) are the best method to assess readiness for extubation and liberation from ventilator support. Although this has been known for decades, controversy remains regarding the mode and duration of optimal SBT. Subirà and colleagues performed a multicenter, randomized clinical trial at 18 intensive care units (ICUs) in Spain. They recruited 1,153 adults who were ready for weaning after at least 24 hours of mechanical ventilation. The researchers compared rates of successful extubation for an SBT involving 30 minutes of pressure support ventilation (PSV) to an SBT involving two hours of T-piece ventilation. The first approach is considered less demanding for patients, while the second approach is considered more demanding. From January 2016 to April 2017, investigators enrolled 578 patients in the two-hour T-piece SBT and 557 patients in the 30-minute SBT with 8 cm H_2O PSV. All patients were followed until July 2017. Readiness for weaning was based on resolution or improvement of the condition that led to intubation, hemodynamic stability, Glasgow Coma Scale score of 13 or greater, respiratory stability, and noncopious secretions. Patients were excluded if they had a tracheostomy or do-not-intubate orders.

Successful extubation occurred in 473 patients (82.3%) in the PSV group and 428 patients (74.0%) in the T-piece group (difference 8.2%, 95% confidence interval [CI], 3.4-13.0%; P = .001). Among the secondary outcomes, reintubation was 11.1% for the

PSV group compared to 11.9% for the T-piece group (difference -0.8%; 95% CI, -4.8% to 3.1%; *P* = 0.63). The median ICU length of stay was nine days for the PVC group vs. 10 days for the T-piece group (mean difference -0.3 days; 95% CI, -1.7 to 1.1 days; *P* = 0.69); median hospital length of stay was 24 days vs. 24 days (mean difference 1.3 days; 95% CI, -2.2 to 4.9 days; *P* = 0.45); hospital mortality was 10.4% vs. 14.9% (difference -4.4%, 95% CI, –8.3% to -0.6%; *P* = 0.02); and 90-day mortality was 13.2% vs. 17.3% (difference -4.1%, 95% CI, –8.2% to 0.01%; *P* = 0.04; hazard ratio 0.74, 95% CI, 0.55-0.99), respectively. This clinical trial revealed that an SBT consisting of 30 minutes of PSV, compared with two hours of T-piece ventilation, led to significantly higher rates of successful extubation, supporting a shorter, less challenging ventilation strategy for spontaneous breathing trials.

■ COMMENTARY

The importance of timely liberation from mechanical ventilation is profoundly important. More than two decades ago, Ely and colleagues found that daily screening of the respiratory function in adults followed by trials of spontaneous breathing can shorten the duration of mechanical ventilation, lower the rate of reintubation, and lower ICU costs.[1] Although it is well known that SBTs are the best way to assess whether patients are ready to discontinue mechanical ventilation, the ideal mode and duration of SBT remains controversial. To further investigate the mode and duration of SBT, Subirà and colleagues conducted a multicenter, randomized clinical trial in 18 ICUs in Spain comparing a two-hour T-piece SBT to the 30-minute PSV SBT with 8 cm H_2O inspiratory pressure and 0 cm H_2O positive end-expiratory pressure (PEEP). Results revealed that the use of 30 minutes of PSV, a less demanding ventilation strategy, led to higher rates of successful extubation without a higher reintubation rate in the 72 hours after extubation. Furthermore, these patients also were significantly less likely to die in the hospital or during the 90 days after randomization.

Although there has been substantial literature supporting the safety of PSV SBT, many clinicians still prefer T-piece. Many clinicians believe T-piece best reflects the physiologic conditions after extubation. This idea was further supported by a recent meta-analysis which showed that T-piece requires the same amount of work after extubation.[2] Despite the evidence that T-piece may be a better weaning mode physiologically, this philosophy was challenged after a meta-analysis of four randomized trials from 2017 reported that SBT with PSV resulted in a higher rate of successful extubation at 48 hours when compared with T-piece (85% vs. 77%) along with an insignificant reduction in ICU mortality.[3] The article by Subirà and colleagues further substantiates these findings.

While it is interesting that Subirà's data further supports SBT with PSV, it also triggers the question of whether all patients will benefit from the same strategy. For instance, are advanced heart failure patients or chronic obstructive pulmonary disease (COPD) patients the same as a patient with a heroin overdose or a patient with resolving septic shock? Interestingly, Subirà's findings were consistent for COPD patients but, of course, the severity of COPD was unknown. One would consider that a patient with severe emphysema, a very compliant state, may gain additional support with a pressure support trial of 8 cm H_2O. A recent study from Pelligrini and colleagues further investigated this concern. This group compared 30 minutes of T-piece and PSV at 10 cm H_2O with all COPD patients undergoing noninvasive ventilation after extubation and found the SBT technique did not influence mechanical ventilation duration for patients with COPD unless the patient was within the difficult/prolonged weaning COPD subgroup.[4] There also is concern for patients with heart failure. Although a PEEP of 5 cm H_2O and pressure support ≤ 8 cm H_2O may be considered a trivial amount of support, even small amounts of pressure support and PEEP in patients with decompensated heart failure can have significant hemodynamic effects and may reduce work of breathing.[5] There is concern that removal of this support may result in rapid deterioration of left ventricular function leading to pulmonary edema. T-piece trials may be considered in patients with impaired cardiac function as this mode may reveal the need for further optimization to prevent re-intubation.

It also is important to question whether the techniques that were used postextubation provided a benefit for these patients and made an impact on outcomes. Prior to randomization, attending physicians had to determine the extubation strategy (i.e., whether to reconnect the patient to the ventilator for one hour before extubation and whether to administer noninvasive ventilation [NIV] or high flow nasal cannula [HFNC] after extubation). Interestingly, physicians were not blinded, and more patients were subjected to prophylactic HFNC or NIV in the PSV group (24.7% vs. 18.7%). Of course, concern was raised that this may have impacted the results, especially if these strategies were used for postextubation respiratory failure rather than prophylaxis. The authors addressed these concerns and explained that respiratory failure postextubation occurred in 103 patients in the T-piece group (21.2%) and 110 patients in the PSV group (20.7%) (*P* = 0.84). NIV was provided to 43 patients in the T-piece group (41.7%) and to 48 patients in the PSV group (43.6%) (*P* = 0.78). HFNC was provided to 28 patients in the T-piece group (27.2%) and to 19 patients (17.3%) in the PSV group (*P* = 0.08). Four patients in each group received both treatments.

The decision to use NIV or HFNC as prophylaxis was prior to randomization, which reinforces the idea that the higher successful extubation rate in PSV was not due to more aggressive use of HFNC or NIV in patients with postextubation respiratory failure. The higher use of prophylactic HFNC or NIV in the PSV group may have been due to chance or been chosen by the clinician to provide more confidence for a successful outcome after a less demanding SBT.

Subirà and colleagues support the use of 30 minutes of PSV during an initial SBT for most patients. Unfortunately, their data also may raise questions in regard to higher risk patient populations and should inspire clinicians to further investigate the most successful approach to liberation from mechanical ventilation for these specific patient populations. ■

REFERENCES

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4. Santos Pellegrini JA, et al. Pressure-support ventilation or T-piece spontaneous breathing trials for patients with chronic obstructive pulmonary disease - A randomized controlled trial. *PLoS One* 2018;13:e0202404.
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Correction

In the March 2020 issue, the dosage of mannitol was listed incorrectly. The sentence should say: Mannitol is dosed intravenously 1 g/kg bolus followed by 0.25 g/kg to 0.5 g/kg every four to six hours to target a serum osmolality of 290-300 mosm/L and the plasma osmolal gap of < 55.

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CME/CE QUESTIONS

1. For deep venous thrombosis prophylaxis in the obese patient, the recommended dose of low molecular weight heparin is:
a. the same as the nonobese patient when body mass index (BMI) is < 45 kg/m^2.
b. 30% higher in those with BMI ≥ 40 kg/m^2.
c. 50% higher in those with BMI ≥ 35 kg/m^2.
d. based on ideal body weight.

2. Regarding cardiovascular physiology in the obese patient, which of the following statements is true?
a. Obesity increases the total blood volumes and cardiac output.
b. Cardiac index and stroke volume index usually go up.
c. Left ventricular filling pressures and systemic vascular resistance usually are normal.
d. Obese patients are not at higher risk of left ventricular diastolic dysfunction.

3. The Subirà study revealed that compared to two hours of T-piece ventilation, which ventilation weaning strategy led to significantly higher rates of successful extubation?
a. 30 minutes of pressure support ventilation
b. 60 minutes of pressure support ventilation
c. 120 minutes of pressure support ventilation
d. 30 minutes of T-piece ventilation