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Review Article

Perioperative Care of the Obese Cardiac Surgical Patient

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Morbid obesity is associated with impairment of cardiovascular, pulmonary, gastrointestinal, and renal physiology with significant perioperative consequences and has been linked with higher morbidity and mortality after cardiac surgery. Cardiac surgery patients have a higher incidence of difficult airway and difficult laryngoscopy than general surgery patients do, and obesity is associated with difficult mask ventilation and direct laryngoscopy. Positioning injuries occur more frequently because obese patients are at greater risk of pressure injury, such as rhabdomyolysis and compartment syndrome. Despite the association between obesity and several chronic disease states, the effects of obesity on perioperative outcomes are conflicting. Studies examining outcomes of overweight and obese patients in cardiac surgery have reported varying results. An "obesity paradox" has been described, in which the mortality for overweight and obese patients is lower compared with patients of normal weight. This review describes the physiologic abnormalities and clinical implications of obesity in cardiac surgery and summarizes recommendations for anesthesiologists to optimize perioperative care of the obese cardiac surgical patient.

Key Words: obesity; morbid obesity; cardiac surgery; postoperative outcomes

OBESITY IS ONE of the leading causes of preventable death and chronic diseases in the United States.[1] The greatest increase in the number of obese individuals over the past 2 decades has occurred in the morbidly obese category, defined as those with a body mass index (BMI) greater than 40 kg/m^2.[2] Because morbid obesity is associated with comorbid conditions such as coronary artery disease, valvular pathology, and aortic disease, these patients comprise an increasing proportion of the cardiac surgery population. A review of the Society of Thoracic Surgeons database revealed that of 559,004 patients presenting for isolated first time coronary artery bypass grafting (CABG), 7.5% were moderately obese (BMI $\geq$35) and 3.4% were morbidly obese (BMI $\geq$40).[3] Morbid obesity is associated with impairment of cardiovascular, pulmonary, gastrointestinal, and renal physiology with significant perioperative consequences and is linked with higher morbidity and mortality after cardiac surgery.[4] Thus a comprehensive understanding of the pathophysiology of morbid obesity is important for the anesthesiologist to risk stratify and optimize perioperative care. The authors focus on the known literature that supports care of the obese cardiac surgical patient and note where gaps exist within the literature.

Definition of Obesity

Obesity is frequently classified using BMI, which is an index of weight and height. BMI is calculated by weight in

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Table 1
Classification of Obesity

Classification	BMI (kg/m^2)
Underweight	< 18.5
Normal	18.5-24.9
Overweight	≥ 25
Obese	≥ 30
Class I	30-34.9
Class II	35-39.9
Class III	≥ 40

NOTE. Classification of Obesity According to the World Health Organization.[5]
Abbreviation: BMI, body mass index.

kilograms divided by the square of height in meters (kg/m^2). Table 1 shows the cutoff values for BMI classification into the categories of underweight, normal, overweight, and obese.[5]

Cellular Physiology in Obesity

The proliferation of adipose tissue and its direct effect on organ systems has been studied for decades; however, the cellular and humoral effects of fat mass only have recently been characterized. Contemporary models of obesity characterize it as a predominantly endocrine disease, with metabolically active adipocytes releasing chemical mediators. These mediators, called adipokines, contribute to local inflammation in adipose tissue and systemic inflammation by increasing hypothalamic sympathetic outflow.[6] Leptin is the best known of these adipokines, but fat cells also produce tumor necrosis factor alpha, inflammatory interleukins, monocyte chemoattractant protein-1, plasminogen activator inhibitor-1, resistin, and angiotensinogen.[7] These mediators contribute to atherosclerosis, insulin resistance, and liver injury and promote an inflammatory state at baseline in obese patients presenting for cardiac surgery.

Obesity contributes to hypertension by activating the renin-angiotensin-aldosterone system and impairing kidney function in the setting of systemic inflammation. Angiotensinogen, produced by adipocytes, may potentiate the renin-angiotensin system and promote renal sodium retention.[8] Fat deposits in the kidneys impair renal function by physical compression and local inflammation.[8] The relationship between obesity and insulin resistance is well defined. Adipokines have been shown in animal models to contribute to insulin resistance centrally by stimulating hypothalamic glucocorticoid release and in peripheral tissues by shifting the balance of lipid metabolism in favor of oxidation.[9] The consequent decrease in lipid synthesis promotes peripheral insulin resistance. Fatty infiltration of the liver results in the common pattern of compressive and inflammatory injury seen in other tissues but also involves a unique pattern of injury. In hepatocytes, free fatty acids contribute to disruption of endoplasmic reticulum homeostasis, leading to cell death and liver damage.[10]

Cardiovascular Effects of Obesity

Morbid obesity has a disruptive effect on the cardiovascular system, in which remodeling occurs to accommodate the higher demand for cardiac output. Stroke volume increases first to meet this demand and does so in proportion to lean body mass.[11] The left atrium and left ventricle both enlarge from volume overload, and the left ventricle undergoes eccentric hypertrophy. The hypertrophied, stiffened left ventricle contributes to progressive diastolic dysfunction.[12] The distribution of fat deposition in the heart also can lead to pathologic changes. Fatty infiltration of the interatrial septum is associated with atrial tachyarrhythmias, whereas right ventricular fat can cause interruption of ventricular impulses, leading to ventricular arrhythmias.[13] Epicardial fat deposits may result in a pressure-induced restrictive cardiomyopathy. Obesity also contributes to coronary artery disease before clinical manifestations are detected. Autopsies performed on adolescents 15 to 34 years old who died of natural causes have revealed that the extent of atherosclerotic lesions in their right coronary arteries correlated directly with abdominal fat and BMI.[14]

Cardiac and stroke indices using body surface area (BSA) can be misleading in the obese population.[15] As a result, one must not rely solely on these derived values when considering an intervention such as starting an inotropic medication. Non-index cardiac output and stroke volume should be considered along with the entire clinical picture.

Respiratory Physiology in Obesity

Morbidly obese patients have an increased demand for ventilation with a lower ventilatory reserve. Ventilatory demand is higher due to an increase in total metabolic activity and higher oxygen consumption. A higher percentage of this oxygen consumption is required to ventilate obese individuals because of respiratory muscle inefficiency. Functional residual capacity (FRC) and expiratory reserve volume decrease in obesity due to abdominal and thoracic fat deposition. In the supine position, expiratory reserve volume can approach closing volume, resulting in the closure of the smaller airways.[16] Obesity also is associated with obstructive sleep apnea (OSA). Obesity-associated fatty deposits in the neck and thorax can increase the collapsibility of the smaller airways seen in OSA. The inflammatory mediators produced by adipocytes, such as the interleukins, can cause central nervous system depression and loss of neuromuscular control.[17]

Gastrointestinal Effects of Obesity

Morbidly obese patients presenting for cardiac surgery are more likely to experience higher intra-abdominal pressures, gastroesophageal reflux disease (GERD), and hiatal hernias.[18] GERD may be a consequence of chronic gastric compression with opening of the lower esophageal sphincter. Anatomic changes to the gastroesophageal junction may lead to hiatal hernias. Obesity is associated with fatty infiltration of the liver that progresses to steatosis and cirrhosis. Steatosis occurs from excess intrahepatic triglycerides resulting from higher uptake than oxidation and export.[19] A combination of pressure-induced changes, oxidative stress from free triglycerides, and local paracrine inflammation produced by adipokines results in hepatocyte destruction, fibrosis, and cirrhosis.

Preoperative Considerations

The physiologic changes associated with morbid obesity lead to a variety of clinical and logistical challenges in the perioperative arena. The preoperative evaluation of the morbidly obese patient should incorporate features relevant to the patient's presenting diagnosis along with an evaluation of obesity-related conditions. The distribution of excess fat is particularly relevant because abdominal fat is more strongly associated with cardiovascular disease, GERD, and insulin resistance. Distribution of fat over the neck may predict challenging airway management and OSA. Pharmacologic prophylaxis against aspiration of gastric contents should be considered because morbidly obese patients have a higher risk of GERD.[18] Vascular access, peripheral nerve blocks, and neuraxial anesthesia may be more difficult in the obese patient.[20]

Airway Management

Airway management of the obese cardiac surgical patient may pose a challenge. Cardiac surgery patients have a higher incidence of difficult airway and difficult laryngoscopy than do general surgery patients, even when stratified with propensity matching.[21,22] It is unclear why cardiac surgical airways tend to be more challenging compared with general surgical patients but may have to do with the systemic nature of the disease and associated risk factors. Furthermore, obesity also is associated with difficult airway management, including difficult mask ventilation and direct laryngoscopy.[22,23] With a combination of these factors, airway management in the obese cardiac surgical patient potentially may be even more challenging. However, there are no studies that specifically address airway management in obese cardiac surgical patients. This discussion is inferred from the broader literature on airway management in the obese surgical patient.

Although obesity may be a risk factor for difficult airway management, the evaluation of the difficult airway in the obese patient does not diverge significantly from the routine assessment for all patients. Factors to consider include a combination of mouth opening, thyromental distance, Mallampati classification, neck mobility, prognathism, body weight (BMI >30), and history of difficult airway.[23] In addition, larger neck circumference (>42.5 cm), inability to prognath, and OSA may predict increased risk of difficult mask ventilation in the obese patient.[24] All morbidly obese individuals presenting for surgery should undergo a validated screening tool for OSA such as the STOP-BANG Questionnaire (Table 2) because it is commonly undiagnosed in this group.[25] In addition to complicating airway management, OSA also is associated with sensitivity to opioids and may necessitate the use of shorter-acting opioids and smaller doses throughout the perioperative period. Diagnosing OSA with the STOP-BANG Questionnaire, even in the immediate preoperative period, can facilitate postextubation planning. These patients often require close monitoring in the intensive care unit (ICU) and may benefit from the use of continuous positive airway pressure (CPAP). The preoperative airway evaluation is important to decide whether airway management should be performed with the patient awake or asleep and which technique for intubation is most likely to be successful. Appropriate management can include direct laryngoscopy, video laryngoscopy, and flexible bronchoscopy; in fact, it is prudent to have more than 1 approach available should difficulty be encountered. It also is important to consider that rescue with a supraglottic airway device or cricothyroidotomy are potentially more difficult in the obese patient. When awake intubation is considered, diligent topical anesthesia is essential to avoid adverse hemodynamic effects, such as tachycardia and hypertension, which may be mitigated with careful administration of sedative agents, depending on the patient and the situation.

Obese patients are prone to upper airway obstruction with the administration of sedating drugs, and OSA is highly prevalent in cardiac surgical patients, with reports as high as 47%.[26,27] Prevention and elimination of upper airway obstruction during the preoperative, induction, and postoperative phases are paramount. Pulmonary and cardiovascular changes associated with obesity also contribute to difficult airway management. Reduction in FRC, proclivity for atelectasis during hypoventilation and apnea, and restrictive lung disease contribute to higher risk for hypoxia and hypoventilation in the perioperative period, further complicating airway management and ventilation. Decreased chest wall compliance in the obese patient may cause expiratory flow limitation and gas trapping due to early airway closure and subsequent generation of intrinsic positive end-expiratory pressure (PEEP). This is further complicated by cardiovascular disease and risk for pulmonary hypertension associated with obesity and obesity-related hypoventilation syndrome.

Table 2
STOP-BANG Sleep Apnea Screening Questionnaire[25]

1. Do you **S**nore loudly enough to be heard through a closed door?	
Yes (1 point)	No (0 points)
2. Do you often feel **T**ired or sleepy during daytime?	
Yes (1 point)	No (0 points)
3. Has anyone **O**bserved you stop breathing during your sleep?	
Yes (1 point)	No (0 points)
4. Do you have or are you being treated for high blood **P**ressure?	
Yes (1 point)	No (0 points)
5. **B**MI $\geq$ 35 kg/m^2?	
Yes (1 point)	No (0 points)
6. **A**ge $\geq$ 50 years?	
Yes (1 point)	No (0 points)
7. **N**eck Circumference	
Males: Is your shirt collar 17 inches (43 cm) or larger?	
Females: Is your shirt collar 16 inches (41 cm) or larger?	
Yes (1 point)	No (0 points)
8. Male **G**ender?	
Yes (1 point)	No (0 points)

Score ≥ 3: At high risk for obstructive sleep apnea.
Score <3: Low risk for obstructive sleep apnea.

Table 3
Airway Management: Interventions and Physiologic Benefits

Intervention	How to Perform	Proposed Physiologic Benefit
Quantitative preoxygenation[28]	FeO_2 > 0.9 Monitor FeO_2 on ventilator	Maximizes: • Alveolar oxygen tension • Nitrogen washout
Head-above-feet[30–32]	• Reverse Trendelenburg 30 degrees • HOB 25-30 degrees	Improves: • FRC • Airway patency • Respiratory mechanics • Preoxygenation
Ear to sternal notch[33,34]	Ramp: • Blankets • Positioner • Bed adjustment	Improves: • Airway patency • Mouth opening • Mask ventilation • Laryngoscopic view
Preoperative positive pressure[35]	• Mask seal, close pop-off valve • Pressure support • CPAP or BiPAP machine	Maintains: • Airway patency • Alveolar patency Preoxygenation
Intraoperative positive pressure[36–38]	• PEEP of 10 cmH_2O • Recruiting maneuvers (Valsalva)	Maintains: • Alveolar patency • FRC
Postoperative positive pressure[39,40]	PEEP, CPAP, BiPAP, high-flow nasal cannula	• Maintains: • Airway patency • Alveolar patency
Passive apneic oxygenation[41,42]	With patent glottis: • Nasal cannula at 15 L • High-flow nasal cannula (eg, OptiFlow) Nasal anesthesia mask (eg, SuperNO$_2$VA)	• Prolongs the time to desaturation • Maintains alveolar oxygen tension May delay rise in CO_2

Abbreviations: BiPAP, bilevel positive airway pressure; CO_2, carbon dioxide; CPAP, continuous positive airway pressure; FeO_2, fractional expired oxygen; FRC, functional reserve capacity; HOB, head of bed; PEEP, positive end expiratory pressure.

Fortunately, several of these anatomic and physiologic factors that make airway management challenging in the obese cardiac surgical patient can be mitigated by minimizing airway obstruction, maintaining alveolar patency, optimizing FRC, and maximizing alveolar oxygen tension. Positioning, positive pressure, and passive apneic oxygenation are 3 interventions that are useful to overcome some of the challenges of airway management and ventilation in the obese surgical patient. Preoxygenation to an end-expiratory oxygen tension of 90% is a useful quantitative measure to ensure that adequate alveolar oxygen levels have been achieved.[28] Positive pressure with CPAP has been shown to maintain airway patency during anesthesia-induced obstruction.[29] A summary of airway management interventions and physiologic benefits is described in Table 3.[28,30–42]

Positioning to Optimize the Airway

There are no studies evaluating the optimal position for intubation in obese cardiac surgical patients. In general, respiratory mechanics are better in the sitting position compared with the supine position. Positioning the head above the feet preserves FRC, improves preoxygenation, and may prolong the time to desaturation during apnea. Reverse Trendelenburg may be superior to the back-up position, and both appear to be better than the supine position.[30,31] Preoxygenation with the head of the bed elevated to 25 degrees prolongs the time to desaturation during apnea.[32] Positive pressure during induction improves preoxygenation and helps to maintain alveolar patency.[35] This is particularly useful while sedating the cardiac patient for arterial line placement before induction of anesthesia. In addition, head-above-feet positioning may improve mask ventilation and laryngoscopy.[34] Continuing reverse Trendelenburg positioning during surgery also may improve respiratory mechanics in the obese surgical patient.[31] Careful attention to hemodynamics in cardiac surgical patients is important to ensure adequate blood pressure and cerebral perfusion in the head-elevated position. Positioning the patient for preoxygenation and intubation by ramping with blankets or a positioning device or by adjusting the operating room table so that the tragus of the ear is at the level of the sternal notch facilitates mask ventilation and direct laryngoscopy.[33] Such positioning effectively achieves a proper “sniffing” position, which allows for full mouth opening, placement of the facemask, and insertion of the laryngoscope.[34] With video laryngoscopy, such positioning has not necessarily been shown to improve the laryngeal view, but in the authors’ experience, it makes blade positioning significantly easier and faster.

Passive Apneic Oxygenation

Passive apneic oxygenation has gained significant traction as a means to maintain oxygen levels during apnea and extend the period before desaturation occurs. The most dramatic examples involve the use of a high-flow nasal cannula (Optiflow; Fisher & Paykel, Auckland, New Zealand) for transnasal humidified rapid-insufflation ventilator exchange (termed THRIVE). With THRIVE, high-flow up to 70 L per minute (LPM) results in complex fluid dynamics that with cardiac pulsations allow for some degree of gas exchange and a delayed rise in partial pressure of carbon dioxide during apnea.[41] Furthermore, these high flows can produce 3 to 5 cmH_2O of PEEP. Current research with similar devices and higher flow rates is ongoing. When the high-flow nasal cannula is used in the authors' cardiovascular surgical ICU and extubation is required, the authors continue it through the intubation phase but do not routinely use the technique in the operating room for cardiac surgical patients. In many situations, a simple nasal cannula (or 2) with maximum flow is used to provide some degree of passive oxygenation, which can achieve flows over 15 LPM per nasal cannula and may modestly increase the time before desaturation during apnea, even in the obese patient.[42] The authors often use this technique in their cardiac operating rooms when intubating obese patients. To date, there are no studies evaluating passive apneic oxygenation during intubation that specifically address outcomes in cardiac surgical patients. Nasal anesthesia masks that allow for flow rates of 30 LPM are now commercially available ($SuperNO_2VA$; Vyaire Medical, Lake Forest, IL) with the potential advantage of allowing both positive pressure ventilation and relatively high flows during apnea. Clinical evidence for the device in the literature currently is limited.

Ventilation Management

Once the airway is secured and the patient is placed on the ventilator, PEEP and Valsalva maneuvers have been shown to effectively maintain alveolar patency, with PEEP levels of 10 cmH_2O commonly used in obese patients undergoing noncardiac surgery.[36,37] When titrated carefully, a PEEP of 10 cmH_2O often does not result in hemodynamic compromise, even in cardiovascular patients. In cardiac surgical patients (but not specifically obese patients), evidence increasingly supports low tidal volume ventilation at 6 mL/kg using predicted body weight compared with 10 mL/kg in the operating room.[43] High tidal volume ventilation in the ICU after cardiac surgery appears to be associated with worse outcomes, including higher rates of organ dysfunction.[44] Cardiac surgical patients with hypoxia after cardiopulmonary bypass appear to have improved outcomes with intensive recruitment maneuvers involving higher PEEP (13 cmH_2O compared with 8 cmH_2O) with recruitment maneuvers.[45] The Protective Ventilation During General Anesthesia for Surgery in Obese Patients (PROBESE) study is a multicenter, 2-arm, international randomized controlled trial currently enrolling patients to study the effect of intraoperative ventilation with higher versus lower levels of PEEP.[38] Lowering the fraction of inspired oxygen (FiO_2) also may help to prevent atelectasis because non-absorbed nitrogen may stent open alveoli,[36] but PEEP appears to be sufficient to preserve alveolar patency even when high FiO_2 is used.[39] Thus maintenance with higher FiO_2 in the cardiac surgical patient may be warranted to prevent hypoxia, maintain lower pulmonary vascular resistance, and allow for higher oxygen tension during extubation. During the extubation phase, transitioning to pressure support (if available on the ventilator) and then PEEP (by dialing the pop-off valve when the patient is spontaneously breathing) maintains positive pressure and alveolar patency. In addition, extubation to prophylactic nasal CPAP in cardiac surgical patients has been shown to decrease pulmonary complications, reintubation rates, and readmissions to the ICU.[40] If confirmed by additional studies, noninvasive ventilation after cardiac surgery may prove to be a valuable component of perioperative management.

Positioning Difficulties and Complications

Although positioning the patient to optimize the airway and breathing is important, special attention also is necessary to avoid falls and positioning-related injury. Obese patients are at increased risk for falls from operating room (OR) and procedural tables, especially when steep Trendelenburg, reverse Trendelenburg, or side-to-side rotation is used, and associated falls have resulted in patient deaths.[46] When selecting an OR table for morbidly obese patients, the manufacturer's weight rating should be consulted to avoid tipping. It is important to note that an OR table used in the reverse position may have a lower weight capacity and that the weight limit only applies when the bed is locked.[47] Transitioning a poorly mobile or unconscious patient from one bed to another can be safely facilitated using a commercially available inflatable mattress such as the Airpal Patient Transfer System (Hill-Rom, Chicago, IL).

No literature exists regarding positioning injuries specific to obese cardiac surgical patients. However, it is known that obese patients are at greater risk of pressure injury, such as rhabdomyolysis and compartment syndrome.[48] This risk is exacerbated when the patient's weight is disbursed over a smaller area, such as in the lateral position. Although most cardiac surgical cases are performed with the patient in the supine position, thoracic approaches such as right lateral position are used in minimally invasive or robotic mitral valve procedures and left lateral position are often used in thoracic aortic repairs. This risk of injury is compounded by the increased surgical challenges related to obesity, which may mandate longer surgical times. The gluteal and shoulder compartment are at risk, and assessment can be difficult.

Pharmacokinetics in Obesity

Obese patients undergoing cardiac surgery exhibit altered pharmacokinetics due to the pathophysiologic changes associated with excess weight. Allometry is the study of the relationship of body size to body characteristics and properties, including anatomy and physiology.[49] These nonlinear and frequently complex relationships attempt to explain why a 7-kg pediatric patient does not necessarily receive one-tenth the dose of a drug given to a 70-kg adult and why a 210-kg adult does not necessarily receive 3 times the dose.[50] Although the data regarding the effect of obesity on various pharmacokinetic parameters frequently are unclear or contradictory, certain fundamental rules generally can be followed. It is beyond the scope of this article to cover all pharmacodynamic implications; however, it is critically important to identify drugs with a narrowed therapeutic window in this population. For example, when giving narcotics and sedatives, even a small dosing error may result in airway obstruction or apnea, and this may be further compounded by difficult airway management.[51] It is important to note that pharmacokinetics recommendations specific to the cardiac obese surgical patient do not currently exist.

Volume of Distribution

Obese patients typically exhibit an increased volume of distribution (Vd), which varies due to specific drug properties. Despite limited blood flow to adipose tissue compared with lean tissue, lipophilic drugs have the greatest increase in Vd in obese patients.[52] Calculating the exact change in Vd is further complicated by patient-specific factors, including tissue size, tissue permeability, and plasma-protein binding concentrations.[53] Obese patients have an increased circulating blood volume, and although it is not proportional to weight, this results in a clinically significant increase in Vd for drugs that remain primarily intravascular.[54] These factors must be balanced with other pathophysiologic processes in cardiac surgery patients. For example, hypertension is associated with decreased circulating blood volume, whereas renal failure and hepatic failure may be associated with fluid retention and decreased plasma-binding proteins. Some research has indicated that plasma-binding affinity may be altered in obesity independent of protein concentrations, although this remains controversial.[55–57] Furthermore, obese and non-obese individuals may have similar tissue concentrations but different drug concentrations in plasma.[58]

Clearance and Elimination Half-Life

Drug clearance is not independently elevated in obesity; however, it generally is higher due to increases in cardiac output, hepatic blood flow, renal blood flow, renal mass, and glomerular filtration rate.[59,60] Gaps exist in the literature with direction to drug clearance in the cardiac surgical patient specifically. As such, clearance generally is approached with obesity and cardiac disease as separate factors. As with Vd, this must be balanced with other comorbidities found in cardiac surgery patients, such as diabetes or hypertension, that may result in impaired renal and hepatic clearance.[61] Obesity results in fatty infiltration and may have an unpredictable effect on the metabolic activity of various enzymes.[53] The physiochemical properties of a drug, including lipophilicity, usually have limited effects on clearance.[59]

Although the half-life ($t^{1/2}$) of a drug is inversely related to clearance, the observed $t^{1/2}$ in obese patients is unpredictable.[57] An increase in Vd may offset clearance (CL) as illustrated in the following equation[53]:

$$t^{1/2} = (Vd) \times (0.693)/CL$$

As a rule, Vd is most important in determining an induction or loading dose, and clearance is an important determinant of maintenance dosing.[59]

Table 4
Indirect Measures of Body Composition

Measure	Formula
TBW	kg
BMI	TBW/m^2
BSA	[(TBW) × cm / 3,600]$^{1/2}$
IBW	
Male	49.9 + 0.89 × (cm − 152.4)
Female	45.4 + 0.89 × (cm − 152.4)
Percent IBW	(TBW × 100) / IBW
Adjusted body weight	IBW + 0.4 × (TBW − IBW)
Lean body weight	
Male	(9,270 × TBW) / (6,680 + 216 × BMI)
Female	(9,270 × TBW) / (8,780 + 244 × BMI)

Abbreviations: BMI, body mass index; BSA, body surface area; IBW, ideal body weight; TBW, total body weight.

Body Composition

Assessing the degree of obesity is important for determining alterations in drug regimens. Total body fat is an important variable for lipophilic drugs, and the remaining fat free mass is useful as an estimate of physiologically active tissue.[59] It should be noted, however, that obese patients have an increase in both adipose tissue and fat free mass compared with other individuals of the same age, sex, and height.[52]

Indirect measures of body composition are clinically useful because they can be calculated by parameters readily available in the medical record (Table 4). These include total body weight (TBW), BMI, BSA, ideal body weight (IBW), percent IBW, adjusted body weight, and lean body weight. These measures are frequently used as dosing scalars that aim to correct for pharmacokinetic alterations in obesity. Although BMI and BSA are the most common way to classify obesity, they do not accurately predict the amount of adipose tissue and therefore are problematic for purposes of pharmacokinetics.[59] IBW adjusts for differences in sex; however, using this calculation results in all patients of the same height receiving equivalent dosing regardless of weight.[62]

Drug Dosing in the Obese Patient

There is no single dosing scalar that should be used for all drugs in obese patients undergoing cardiac surgery.[63] To understand the implications of obesity on a given drug, pharmacokinetic studies are performed that compare the Vd corrected by TBW in obese and non-obese individuals. When this ratio is lower in obese individuals, it indicates that the drug does not undergo significant uptake into adipose tissue

Table 5
Recommended Dosing For Drugs Frequently Used in Cardiac Surgery

Drug	Dosing Scalar
Cisatracurium	IBW[51]
Dexmedetomidine	LBW[50]
Etomidate	LBW[51]
Fentanyl	LBW[62]
Heparin	
Loading	Reduced initial dose[67]
Maintenance	TBW[67]
Lidocaine	IBW[50]
Midazolam	
Loading	TBW[62]
Maintenance	IBW[62]
Neostigmine	TBW[62]
Propofol	
Induction	LBW[51]
Maintenance	TBW[51]
Remifentanil	LBW[51]
Rocuronium	IBW[51]
Succinylcholine	TBW[51]
Sugammadex	TBW[66]
Sufentanil	LBM[50]
Vecuronium	IBW[51]

Abbreviation: IBW, ideal body weight; LBW, lean body weight; TBW, total body weight.

and dosing should be corrected for body composition and based on lean body weight or IBW rather than TBW.[59] However dosing of some lipophilic drugs such as fentanyl based strictly on TBW may result in overdose and is not supported by pharmacokinetic studies in the obese.[51]

Desflurane is the least lipophilic and least soluble of the modern volatile agents and thus may result in faster emergence in the obese population. By the same principle, nitrous oxide has been described as an ideal anesthetic for the obese patient.[64] Although rapid offset may be more pronounced with longer exposure, the result may not be clinically relevant in cardiac surgery patients who are not immediately extubated in the operating room.[65] Central sensitivity to lipophilic agents also may play a role.

Acknowledging the unpredictability of drug effect in the obese patient, the goal of pharmacologic management should be to use a multimodal anesthesia strategy.[50] Although limited outcomes data exist, it is logical to minimize dosing and limit lipophilic drugs when suitable alternatives exist. It also is imperative to take into consideration the consequence of underdosing certain drugs such as neuromuscular blockade reversing agents.[66] Monitoring effects of medications can allow for careful titration. For example, frequent measurement of activated clotting time can be used to detect an overdose of heparin before cardiopulmonary bypass and allow for dosing adjustments.[67] Dosing scalars for drugs frequently used in cardiac surgery patients are shown in Table 5.

Obesity and the Cardiopulmonary Bypass Machine

The challenges of managing morbidly obese patients also extend to the cardiopulmonary bypass period. The extracorporeal pump should accommodate the obese patient's larger blood volume, provide adequate cardiac output, and deliver enough oxygen to meet the higher metabolic demand seen in this cohort. BSA typically is used to calculate the pump flow, but there is controversy regarding whether it should be adjusted in morbidly obese patients. Small studies have evaluated the effect of lower flow on indices of end organ perfusion in morbidly obese patients with favorable results, but additional research is necessary to evaluate outcomes.[68] In cases of extreme morbid obesity, the patient's calculated blood volume may exceed that of the venous reservoir. Isolated case reports of morbidly obese patients with BMI >45 have described the use of multiple venous reservoirs in series to facilitate exsanguination.[69] Similarly, an additional oxygenator can be added in parallel to the existing one to increase the surface area for gas exchange and improve extracorporeal oxygen delivery.[70,71] Propofol pharmacokinetic parameters were similar in obese and non-obese patients receiving infusions during cardiopulmonary bypass.[72]

Heart Transplantation in the Obese Patient

Body weight is the most commonly used measure to determine donor-to-recipient size matching. The International Society for Heart and Lung Transplantation recommends that a heart from a donor weighing $<70\%$ than the recipient should not be accepted unless the donor is a male weighing >70 kg. Bergenfeldt et al examined data from 52,455 adult heart transplantations and found that inappropriate weight match (donor weight $<70\%$ of recipient's weight) was associated with increased 30-day mortality in the non-obese recipients but not in the obese recipients.[73] This may suggest that the current International Society for Heart and Lung Transplantation guidelines may be too conservative when the heart transplantation recipient is obese.

Minimally Invasive Cardiac Surgery in the Obese Patient

Adequate surgical exposure using a minimally invasive approach may be difficult in the obese patient. Percutaneous peripheral cannulation often is more challenging in an obese patient, and a cut down for vessel exposure may be required. However, due the correlation of obesity with deep sternal wound infections, smaller incisions through a mini-sternotomy or other minimally invasive approach may be preferred to a large sternotomy or thoracotomy.[74,75]

Postcardiac Surgery Challenges

Morbidly obese individuals have a higher rate of deep sternal wound infections, longer ventilator times, and renal failure.[4,74] Improving postoperative pulmonary care begins with optimal ventilator and pharmacologic management in the operating room. Lung protective ventilation should be instituted to avoid volutrauma. Minimization of opioids and reversal of neuromuscular blockade are necessary to avoid prolonged postoperative mechanical ventilation. Morbidly obese patients can present with varying degrees of mobility, and every effort should be made to mobilize them to a chair and to ambulate them with assistance. Executing a goal-directed perioperative plan may be the key to improving perioperative outcomes after cardiac surgery in morbidly obese patients.

The Obesity Paradox

Obesity is well known to be associated with several chronic disease states, such as diabetes mellitus, hypertension, and coronary artery disease. It is a significant public health concern and is associated with increased all-cause mortality.[1,2] In cardiac surgery, the effects of obesity on perioperative outcomes are conflicting. Studies examining outcomes of overweight and obese patients in cardiac surgery have reported varying results. An "obesity paradox" has been described, in which the mortality for overweight and obese patients is lower compared with that of normal weight patients.[66,67,69–72,76,77] This U-shaped association with respect to mortality and BMI indicates highest mortality for those at the extremes of weight and lowest mortality for the overweight and moderately obese patient, indicating a possible protective effect in these groups.[78,79]

In a 2002 study by Prabhakar et al, the Society of Thoracic Surgeons database was used to evaluate increased BMI in 559,004 patients undergoing CABG. Patients with a BMI > 35 to 39.9 had a moderate increase in mortality compared with normal or mildly obese patients.[3] This is an expected finding, but conflicting studies further showed a protective effect of obesity. Johnson et al studied 78,762 adults undergoing first-time CABG or combined CABG/aortic valve replacement to examine the relationship between BMI and adverse outcomes.[78] They found that overweight and obese patients experienced lower mortality compared with cardiac surgical patients who were normal, underweight, or morbidly obese. Several other studies demonstrated findings that correlated with the obesity paradox and a protective effect in mild to moderate obesity.[79–85] Mortality often is found to be highest at the extremes of weight, both severely underweight and in the morbidly obese.[3,4,79,83,85–88] A summary of the recent studies is described in Table 6.

The possible protective effect of obesity is counterintuitive and not well explained. The paradox exists in heart failure as well, and many studies demonstrate that obese patients have a better prognosis than do their leaner counterparts with heart failure.[89] It is important to consider the limitations of using BMI to define body composition. BMI does not distinguish fat from lean mass and therefore does not adequately reflect

Table 6
Obesity Outcomes Summary

Year	Investigator	Patients (n)	Population	Conclusion
2002	Prabhakar et al[3]	559,004	CABG	BMI > 40 is independent predictor for adverse outcomes and prolonged hospitalization
2003	Reeves et al[80]	4,372	CABG	BMI 30-35 demonstrated no increase in mortality and morbidity
2007	Rahmanian et al[86]	6,940	CABG, valve, combined CABG-valve	BMI < 20 demonstrated decreased long-term survival; BMI > 30 independent predictor of surgical mortality in isolated valve surgery
2011	Stamou et al[81]	2,440	CABG, valve, combined CABG-valve	BMI 25-29.9 demonstrated better early hospital outcomes and improved survival
2011	Thourani et al[87]	4,247	Valve, combined CABG-valve	BMI < 24 demonstrated increased in-hospital and long-term mortality
2011	Roberts et al[82]	1,040	Isolated AVR for AS	BMI in the low 30s demonstrated improved survival
2012	Vaduganathan et al[83]	2,640	Valve, combined CABG-valve	BMI > 25 demonstrated increased survival; BMI < 18.5 increased mortality
2012	Smith et al[79]	1,066	Isolated AVR for AS	BMI < 20 demonstrated decreased survival; increasing BMI not associated with worsened outcomes; overweight patients have survival benefit
2015	Johnson et al[78]	78,762	First time CABG or combined CABG/AVR	BMI 25-34.9 demonstrated lower rates of mortality and adverse perioperative outcomes
2015	Konigstein et al[84]	409	TAVR	BMI > 25 demonstrated decreased 1-year mortality
2016	Gao et al[85]	4,740	CABG, valve, or combined CABG-valve	BMI < 18.5 and > 40 demonstrated significantly greater in-hospital mortality, 30-day mortality, surgical mortality, and patient readmission within 30 days
2017	Ghanta et al[4]	13,637	CABG, AVR, MVR, combined CABG-valve	BMI > 40 demonstrated increased rates of mortality and major morbidity
2017	O'Byrne et al[88]	18,337	Congenital cardiac surgery age 10-35 years	Severely underweight and obese (by BMI percentile) patients had higher risk of surgical mortality
2017	Lv et al[76]	12,330	TAVR	Obese patients had lower mortality than did normal weight patients
2017	Hartrumpf et al[77]	15,314	All major cardiac surgery	BMI 25-25.9 demonstrated lowest mortality

Abbreviations: AS, aortic stenosis; AVR, aortic valve replacement; BMI, body mass index; CABG, coronary artery bypass grafting; MVR, mitral valve replacement; TAVR, transcatheter aortic valve replacement.

Table 7
Gaps in the Evidence for Perioperative Recommendations of Obese Cardiac Surgical Patients

System	Gap in Current Evidence Specific to Obese Cardiac Surgical Patients
Airway management	There is a lack of literature in optimal intubation and airway maneuvers and/or outcomes in obese cardiac surgical patients, and data are insufficient on the incidence of increased difficult airway.
Respiratory management	Very little has been published to date specific to intraoperative or postoperative respiratory strategies for obese cardiac surgical patients.
Induction agents	Data are lacking on outcomes with different induction types or agents specifically in obese cardiac surgical patients; optimal techniques are unknown.
Anesthetic agents	Data are sparse on which anesthetic agents should be use or avoided in the obese cardiac surgical patient exist; most of what is referred to in the literature is generalized to all obese surgical patients.
Pharmacokinetics	Data exist on drug clearance, Vd, and other pharmacokinetics for obese surgical patients but not specific to cardiac surgery.
Surgical positioning	Although there have been published data on obese patients and risk of positioning injuries during surgery, data specific to the risk of obese cardiac surgical patients are scarce.[38]

Abbreviation: Vd, volume of distribution.

adiposity. Furthermore, using BMI alone does not distinguish obese patients with normal metabolic profiles from those who have diabetes.[90] Other indices such as waist circumference or waist-to-hip ratio may better identify higher-risk patients.[79,85,90] Obese patients also often are found to be younger and male and have a higher incidence of hypertension and diabetes compared with normal weight patients.[85] Younger obese patients may seek medical care earlier and be treated more aggressively than older, normal weight patients.[84] These varying preoperative profiles may influence outcomes, and both underweight and overweight patients should have a nutrition evaluation preoperatively to correct imbalances before surgery.[91]

Conclusion

Obese and morbidly obese patients increasingly are presenting for cardiac surgery and pose challenges for the cardiac anesthesiologist. Morbid obesity is associated with higher morbidity and mortality after cardiac surgery. Beyond predicting postoperative poor clinical outcomes, there is an increased cost associated with morbidly obese cardiac surgical patients and significant intraoperative modifications that must be addressed. From airway management to drug dosing, anesthesiologists who care for these patients must take precautions and formulate plans to avoid outcomes such as respiratory failure, nerve injuries from positioning, renal injury, and sternal wound infections. Table 7 summarizes the lack of evidence specifically examining patients who are both obese and undergoing cardiac surgery. Management of the obese cardiac surgical patient often has to be inferred from studies examining obese patients undergoing general surgery. Although some studies have described the "obesity paradox," in which moderate obesity may have protective effects in some cardiac surgery models, morbid obesity is a clear risk. Although it is known that morbidly obese cardiac surgical patients may represent a perioperative challenge, understanding the physiologic changes associated with this disease state is imperative to the ability to modify the anesthetic plans. Future studies targeting protocols and recommendations for the perioperative care of the morbid obese are warranted.

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