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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

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