Challenges of Anesthesia

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The anesthesiologist will care for an increasing number of obese patients undergoing bariatric surgery. However, anesthesia for bariatric surgery is challenging and must take into account the specific pathophysiology, co-morbidities, and related complications of obesity, as morbid obesity is an independent determinant of death among surgical critically ill patients. Progress in anesthesia knowledge and techniques contributes essentially to a safe perioperative management of the patient presenting for bariatric surgery [1-6].

14.1 Risk Assessment

Data regarding the safety and risk of bariatric surgery are limited. The reported in-hospital mortality for bariatric surgery is 0.1–0.2%; pulmonary complications occur in 4–7%, cardiac complications in 1.0–1.4% of patients [7]. Obese patients have a greater prevalence of co-morbidities such as coronary artery disease, hypertension, type-2 diabetes, steatohepatitis, sleep apnea, and pulmonary hypertension elevating their perioperative risk [8]. Bariatric surgery should thus be considered intermediate- to high-risk noncardiac surgery [9]. However, obesity alone does not seem to be a risk factor for postoperative complications [10]. Childhood obesity as well is associated with a number of medical co-morbidities including type-2 diabetes, asthma, sleep apnea, and heart disease. A recent study revealed a greater incidence of difficult mask ventilation, airway obstruction, bronchospasm, major oxygen desaturation, and overall critical respiratory events in these patients compared with non-obese children, but there were no serious sequelae [11]. Pulmonary embolism is a leading cause of perioperative mortality in bariatric surgery. Risk factors for thromboembolism include history of a prior thromboembolic event, central fat distribution, smoking, female gender, age, venous insufficiency, sleep apnea, hypercoagulable state, and use of oral contraceptives [12]. Patients at highest risk may profit from preoperative placement of inferior cava filters [13].

14.2 Pathophysiological Changes

14.2.1 Pulmonary Function

Oxygen consumption and carbon dioxide production are increased in the obese, owing to the metabolic activity of the excess fat and the increased workload on supportive tissues. In consequence, the minute ventilation rate is increased [14]. The work of breathing is increased, because more energy must be expended to carry the body mass, while the respiratory muscle function is impaired. Morbid obesity is associated with an exponentially decreasing functional residual capacity (FRC), expiratory reserve volume, and total lung capacity [15]. The residual volume usually is not affected. In the supine position, the expiratory reserve volume can fall below the closing volume, resulting in gas trapping with ventilation-perfusion mismatch, shunting, and hypoxemia. Anesthesia is a further aggravation, such that a 50% reduction in observed FRC occurs in the anesthetized obese patient compared with a 20% fall in the non-obese subject [16]. The addition of positive end-expiratory pressure (PEEP) to the ventilation achieves an improvement in both FRC and arterial oxygen tension, but only at the expense of cardiac output and oxygen delivery [17, 18]. Increased pulmonary blood volume and increased chest wall mass from adipose tissue lead to a reduced compliance of the respiratory system. Abnormal diaphragm position and upper airway resistance increase the work of breathing [19].

14.2.2 Obstructive Sleep Apnea

Undiagnosed obstructive sleep apnea (OSA) is very common in severely obese patients. More than 70% of persons presenting for bariatric surgery were found by polysomnography to have sleep apnea [20, 21]. Risk factors for OSA and clinical signs are male gender, increased neck circumference (men >44 cm / women >41 cm), visceral obesity, snoring, and daytime fatigue. The final diagnosis can be obtained by polysomnography, but to date no study has confirmed a reduced perioperative risk when polysomnography is performed prior to surgery. The major pathophysiologic consequences of severe sleep apnea include arterial hypoxemia, recurrent arousals from sleep, increased sympathetic tone, pulmonary and systemic hypertension, and cardiac arrhythmias. Possible mechanisms include hyperleptinemia, insulin resistance, elevated angiotensin II and aldosterone levels, oxidative and inflammatory stress, impaired baroreflex function, and endothelial dysfunction [22]. Magnetic resonance imaging shows that obesity causes OSA by deposition of adipose tissue into pharyngeal tissues, predominantly the lateral pharyngeal walls [23]. In addition, the extraluminal pressure is increased by superfi-cially located fat masses, leading to external compression of the upper airway [24]. Several studies confirm an increased risk of difficult tracheal intubation [25, 26]. Failed intubation occurs in as many as 5% of attempted cases [24]. Patients with OSA may be very sensitive to sedative medications. Such commonly used anesthetic drugs as propofol, thiopental, opioids, benzodiazepines, and even small doses of neuromuscular blocking agents are proven to cause pharyngeal collapse [27]. Even minimal sedation may cause...