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Obstructive Sleep Apnea in Bariatric Surgery Patients

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Introduction

Obstructive sleep apnea (OSA) is a chronic disease that affects up to 24 % of North American adults and is characterized by partial or complete airway obstructions that occur during sleep [1]. Patients with OSA suffer from loud snoring, fragmented sleep, daytime somnolence, and cardiorespiratory sequelae (hypoxia) which may go undiagnosed for years. There is a strong link between obesity and OSA, with the typical patient being overweight with numerous comorbidities; thus, a keen understanding of the perioperative diagnosis and management of sleep apnea is paramount when caring for bariatric surgery patients. Sleep apnea is also one of the many comorbidities that improve or resolve after bariatric surgery [2]. This chapter will review the pathophysiology, clinical features, preoperative evaluation, perioperative management, and postoperative outcomes of OSA in patients undergoing bariatric surgery.

Epidemiology and Pathophysiology

The general prevalence of OSA with daytime somnolence has been reported in the range of 3–7 %, but there are numerous factors like gender, age, comorbid conditions, alcohol, smoking, and obesity that influence the prevalence of OSA [3–5]. The incidence of OSA in bariatric surgery patients is up to 30 times greater than in the general population, and studies have shown that underdiagnosis is commonplace among obese patients. In fact, sleep studies performed during the preoperative assessment for bariatric surgery have suggested an overall prevalence of 48–91 % [6–10].

As previously mentioned, OSA is characterized by periodic episodes of hypopnea (partial airway obstruction) or apnea (complete airway obstruction) during sleep that are a direct result of narrowing in the upper airway. These events, which typically lead to loud snoring and restlessness, prevent the patient from achieving restful sleep and typically lead to significant daytime somnolence. Furthermore, the resulting hypoxia can have severe cardiovascular consequences, with OSA being linked to hypertension, ischemic heart disease, arrhythmias, stroke, and sudden/premature death, among other conditions [11–14]. The mechanism of periodic apneic or hypopneic episodes is thought to be due to increased amounts of upper airway (pharyngeal and tongue) soft tissue which preclude the passage of air to the larynx and, ultimately, the lungs [15]. While this can be seen as an anatomical variance in nonobese patients, this phenomenon is certainly more prevalent in overweight populations.

There are many factors that contribute to the degree of collapse of the upper airway that is seen in patients with OSA, and it is worth noting that these changes in airway caliber may not be present during wakefulness. With the onset of sleep, there is a physiologic reduction in neural-mediated activation and tone of the upper airway muscles. The decrease in airway tone is actually more than what is seen for the respiratory muscles proper, and the negative intrathoracic and intra-airway pressures that are generated during inspiration are transmitted to the more pliable pharynx. This normal physiologic phenomenon may have no consequences for those with normal amounts of upper airway soft tissue but can cause significant airway narrowing in patients with bulky upper airway soft tissue [16]. It has been hypothesized that the improvements seen in OSA patients who lose weight by any means (including bariatric surgery) are at least partially a result of a decrease in upper airway tissue bulk [17]. It is also recognized that increased levels of inflammatory cytokines and decreased expression of anti-inflammatory regulators are present in obese subjects with OSA, but their role in the pathophysiology is still unknown [17, 18].

The consequences of periodic hypopneic and apneic events are numerous, and a complete review is perhaps beyond the scope of this chapter. However, it is important to note that the effects reach far beyond disturbances in sleep patterns. For instance, the brief desaturations that occur while sleeping lead to drops in oxyhemoglobin concentrations and a subsequent decrease in both heart rate and blood pressure. When the obstruction is relieved, there is a reflex

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surge in sympathetic autonomic tone, which leads to tachycardia and hypertension and can predispose to cardiac arrhythmias [19]. Some subjects with OSA also demonstrate hypercapnia and chronic respiratory acidosis during wakefulness, known as the obesity hypoventilation syndrome. These patients usually also suffer from chronic obstructive pulmonary disease (COPD) and are thought to be at even higher risk than those with OSA alone [20].

The constellation of effects that OSA imparts on obese individuals is significant and certainly raises concerns when considering bariatric surgery on patients who suffer from it. The importance of thorough preoperative assessment and testing by a multidisciplinary team cannot be overstated as it can drastically affect outcomes in an already high-risk population.

Clinical Features

Unfortunately, many of the clinical symptoms of OSA are nonspecific, and this may lead to delayed diagnosis. Patients may be asymptomatic or experience symptoms at night, during wakefulness, or both. The classic presentation is loud snoring with periods of "snorting" that are usually witnessed by a partner, and this is typically accompanied by excessive daytime sleepiness and the need for naps. While less common, patients may give a history of conscious "gasping" or "choking" episodes while abruptly waking from sleep [21, 22]. Over a prolonged period of time, these symptoms can lead to frequent headaches, irritability, and depressed mood, all of which can negatively affect quality of life for the patient and those around them [23].

Diagnosis

The diagnosis of OSA is made by clinical history, physical examination, validated screening tools, and polysomnography (PSG). While the numerous screening tools available can be useful in establishing a diagnosis (Table 51.1) (Maintenance of Wakefulness Test, the Epworth Sleepiness Scale, the Berlin 60 questionnaire, and the STOP-BANG questionnaire), their sensitivities vary, and a more reliable diagnosis can be achieved by PSG [24]. For a PSG study, the patient is admitted overnight to a sleep study lab, and the number of apneic and hypopneic events per hour is quantified. To be defined as apnea during the study, there must be a complete cessation of upper airway flow; to be defined as a hypopneic event, there must be a 50–90 % decrease in flow and at least a 4 % decrease in oxygen saturation for over 10 s. From this data, an apnea-hypopnea index (AHI) is calculated and used to not only diagnose OSA but also to characterize the severity of disease [25].

Table 1. STOP-BANG scoring tool

Do you **S**nore loudly?	Yes/no
Do you often feel **T**ired, sleepy, or fatigued during the day?	Yes/no
Has anyone **O**bserved you stop breathing?	Yes/no
Have you been diagnosed with high blood **P**ressure?	Yes/no
BMI > 35?	Yes/no
Age > 50?	Yes/no
Neck circumference > 17″ (male), 16″ (female)?	Yes/no
Gender = male?	Yes/no
Three "Yes" responses place the patient in the category of suspected high risk of having OSA	

Modified from Frances Chung et al. A tool to screen patients for obstructive sleep apnea. Anesthesiology 2008; 108:812–21

Treatment

While surgical procedures aimed at increasing airway patency do exist, their efficacies vary and many are not validated in morbidly obese patients [26, 27]. For the purpose of this review, we will focus on the medical treatment of OSA.

Continuous positive airway pressure (CPAP) is currently the mainstay in treatment of both obese and nonobese OSA patients (Fig. 51.1). It delivers continuous airway pressure that keeps the upper airway open during sleep, and studies have shown that it improves OSA-related desaturation events, hypertension, and "sleepiness" in those with an established diagnosis of OSA. Conventional nasal CPAP masks can be difficult to tolerate for some patients, and compliance is a constant concern, but numerous types of masks exist, and some may be better tolerated than others. For patients with significant nasal dryness or obstruction, a CPAP facemask can be utilized to improve therapy [28–30]. While no clear consensus exists on the duration of CPAP therapy before considering surgery, the patient should be given ample time to adjust to the system before moving ahead with surgery [10].

Previously, there have been concerns regarding the postoperative use of CPAP and the risk of anastomotic leak after Roux-en-Y gastric bypass based on studies that reported increased complication rates in those using postoperative CPAP [31, 32]. Because of these concerns, some have suggested omitting positive airway pressure therapy in the immediate postoperative period to avoid adverse surgical events. The American Society for Metabolic and Bariatric Surgery (ASMBS) released their position statement in 2012 addressing this issue and concluded that there was no evidence that postoperative CPAP increased the risk of anastomotic leak and that the usage of CPAP immediately after bariatric surgery was appropriate if indicated for pulmonary concerns [10].

Postoperative Care of the Bariatric Patient with Sleep Apnea

The level of postoperative monitoring and care required will ultimately depend on numerous patient- (OSA severity, other comorbidities) and procedure-specific factors (type of surgery,

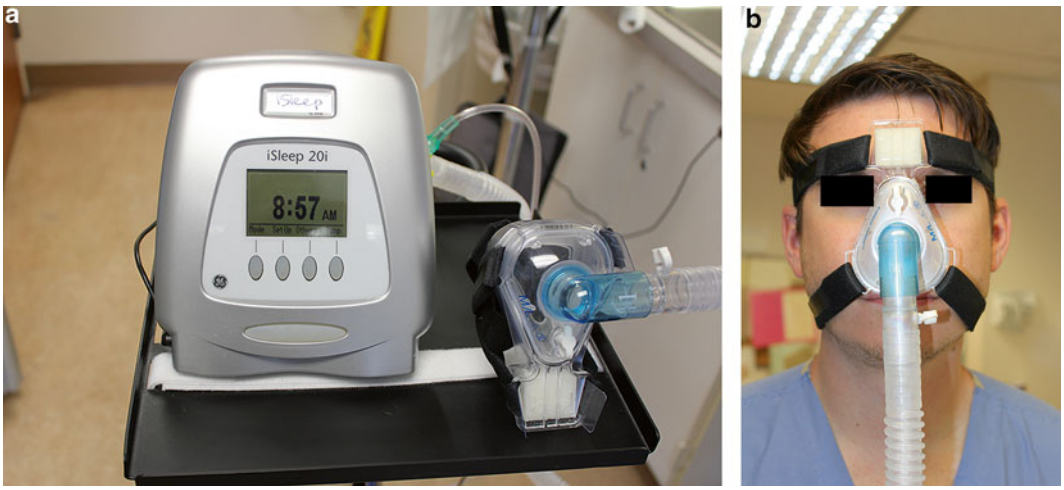

FIG. 1. (**a**) Continuous positive airway pressure (CPAP) device, with face mask (**b**). (GE Breas iSleep™ 20i self-adjusting CPAP; courtesy of GE Healthcare).

laparoscopic vs. open). Our patients are placed on continuous telemetry including pulse oximetry monitoring while on the surgical ward. Higher-risk patients such as those with severe OSA, numerous other comorbidities, superobesity, or advanced age may be better served with a brief ICU stay depending on the facility and airway expertise available at night. There are numerous published guidelines on the postoperative care of bariatric patients, and institutions vary regarding the protocols they incorporate into practice [33, 34]. The level of monitoring should be at the discretion of the surgeon in consultation with the medical consultants managing the patient. For instance, Grover et al. omitted intensive monitoring for their bariatric patients undergoing laparoscopic RYGB. Their cohort included over 200 patients with OSA, and they reported no increase in overall or pulmonary complications despite non-routine use of intensive monitoring [34]. There is consensus, however, that patients should continue their CPAP therapy postoperatively to avoid potential cardiopulmonary events in a patient already under considerable physiologic stress. Patients are encouraged to bring their own masks from home to ensure they have a properly fitting mask during their admission. It is also helpful to have experienced respiratory therapists who are comfortable with positive pressure therapy in morbidly obese patients. The surgeon and the multidisciplinary team should discuss the perioperative usage of CPAP at length with the patient prior to surgery.

Effect of Bariatric Surgery on Obstructive Sleep Apnea

Bariatric surgery is now considered to be the most effective way to achieve durable weight loss and has been shown to improve many obesity-related comorbidities like type 2 diabetes and metabolic syndrome [35–37]. Many studies have also shown that bariatric surgery is capable of improving or resolving OSA, which is not surprising given the fact that even modest weight loss can achieve some degree of improvement. Interestingly, many patients develop clinical improvement or resolution of symptoms of OSA after bariatric surgery, regardless of whether a normal BMI is achieved. In fact, 10–20 % weight loss has been associated with improvement of symptoms and a significant reduction in AHI [38]. It is important to note that not all causes of OSA are obesity related, and bariatric surgery may not improve symptoms of sleep apnea in all patients [39].

Marti-Valeri et al. reported prospective outcomes in 30 subjects who required CPAP (or BiPAP) therapy before RYGB surgery. At 1 year after RYGB, patients experienced significant weight loss and achieved a decrease in mean RDI assessed by PSG (63.6 ± 38.4 preoperatively, 17.4 ± 16.6 postoperatively; $p = 0.004$) [40]. Dixon and colleagues published their prospective randomized control trial assessing surgical (LAGB, $n = 30$) vs. conventional weight loss ($n = 30$) therapy for the treatment of OSA. At 2 years follow-up, the surgical cohort lost significantly more weight and achieved greater AHI reductions (reduction of 25.5 events/h vs. 14 events/h) than the conventional weight loss cohort [41]. Greenberg et al. performed a meta-analysis in 2009 looking at the effects of surgical weight loss on objective measures of OSA. Their analysis included 12 studies ($n = 342$ patients) that had polysomnography performed before and at least 3 months after bariatric surgery. The cohort achieved a 17.9 kg/m^2 reduction in BMI, which corresponded to a pooled cohort reduction of 38.2 hypopneic/apneic events per hour [2].

While many patients subjectively notice improvement in their sleep apnea after bariatric surgery and stop using their CPAP at home several months after surgery, we recommend that they continue to follow up with their pulmonologist to

have their CPAP titrated down during the rapid weight loss phase. Patients should also undergo a repeat PSG 6–12 months after surgery to determine the need for further CPAP therapy.

Conclusion

Obstructive sleep apnea is prevalent in the morbidly obese, and bariatric surgeons must be aware of the history and symptoms suggestive of OSA, as well as the evaluation and management of these patients. A multidisciplinary approach involving the patient, surgeon, anesthetist, medical specialists, respiratory therapists, and support staff is paramount if these patients are to achieve therapeutic success. Metabolic surgery can offer these patients durable weight loss and improvement or remission of OSA.

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