Anesthetic Implications of Obstructive Sleep Apnea in the Ambulatory Setting

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Abstract

The purpose of this review article is to summarize our current knowledge concerning the anesthetic management of patients with obstructive sleep apnea (OSA) in the ambulatory setting.
The pathophysiology, detection, and management of OSA are presented. Although minimal data exist to guide perioperative management in an evidence-based fashion, current guidelines and recommendations are discussed. Depending on the type of surgery, anticipated postoperative analgesic (opioid) requirement, severity of the OSA, associated comorbidities, and the resources of the facility, outpatient surgery may be imprudent.

Keywords: obstructive sleep apnea, ambulatory surgery, morbid obesity.

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Introduction

Although not all patients with obstructive sleep apnea (OSA) are obese nor do all obese patients have OSA, nonetheless a discussion of OSA would be incomplete without including some introductory remarks about obesity. Obesity (defined as a body mass index [BMI] > 30) is reaching epidemic proportions in the United States and has become a major public health hazard. Morbid obesity, defined as a BMI >35 or a weight that is twice ideal body weight (ideal body weight [kg] = height [cm] – 100), affects approximately 5% of Americans and creates notable problems for patients, surgeons, and anesthesiologists alike. Technical challenges abound when one is caring for a morbidly obese patient. Venous access may be very difficult to establish, and noninvasive blood pressure determination may be hampered by an improperly fitting cuff or one that takes too long to inflate. Mask ventilation may be extremely troublesome or impossible, and endotracheal intubation may be challenging. Additionally, patient positioning for surgical procedures often proves vexing, and optimal surgical exposure may prove elusive.

In addition to the technical challenges presented by the morbidly obese patient, the clinician is often confronted with a wide variety of associated medical problems that must be managed perioperatively. These may include diabetes mellitus, osteoarthritis, psychological disturbances, systemic and pulmonary hypertension, restrictive ventilatory dysfunction and hypoxemia, left ventricular and/or right ventricular failure, liver disease, an increased risk of aspiration owing to delayed gastric emptying time and hiatus hernia, hypercoagulabililty, wound infections, and OSA. Indeed, morbidity and mortality rates are high in morbidly obese patients primarily because of associated cardiovascular and respiratory abnormalities and their propensity for deep vein thrombosis and pulmonary embolism. Clearly, OSA plays an important role in contributing to the troubling morbidity and mortality rates encountered in the patient with morbid obesity.

This review article will focus on current knowledge and controversies surrounding the management of patients with OSA.

Sleep Apnea: Definitions and Demographics

Sleep patterns disturbed by snoring are thought to occur in approximately 25% of the population.[1] However, most patients who snore do not have apnea or associated episodes of notable hypoxemia. Nonetheless, OAS is a relatively common disorder among middle-aged adults, especially (obese) Americans. Obesity is a critical independent causative risk factor. The majority of people who have OSA are obese, and the severity of the condition seems to correlate with the patient's neck circumference.[2] In the minority of OSA patients who are nonobese, causative risk factors include craniofacial and orofacial bony abnormalities, nasal obstruction, and hypertrophied tonsils. Importantly, Young and colleagues [3] estimated that 93% of women and 82% of men with moderate to severe OSA have not been clinically diagnosed.

OSA is defined as cessation of airflow for >10 sec despite continuing ventilatory effort, occurring five or more times per hour of sleep, and is usually associated with a decrease in arterial oxygen saturation of >4%. Although this review will focus predominantly on OSA, it should be noted for the sake of completeness that the three types of sleep apnea are obstructive, central, and mixed. Central sleep apnea, much rarer than OSA, is also known as Ondine's curse, an allusion to the mythological water nymph who cursed her unfaithful husband to cease breathing if he ever fell asleep. Unlike OSA, respiratory efforts temporarily stop in central sleep apnea. Diagnosis is established definitively with polysomnography.

It is generally accepted that many patients with OSA have resultant pathologic daytime sleepiness associated with performance decrements. It has also been well established that patients with severe

apnea suffer major health consequences as a result of their condition. Yet, it remains somewhat controversial whether patients with less severe forms of this disease incur the same detrimental consequences, owing to methodological problems and failure to control for confounding factors. Thus, few absolute conclusions can be drawn at this time about the long-term consequences of mild to moderate OSA. However, findings from the Sleep Heart Health Study, [4] the Copenhagen City Heart Study,[5] and others [6] demonstrate a firm association between sleep apnea and systemic hypertension, even after other important patient characteristics, such as age, gender, race, consumption of alcohol, and use of tobacco products are controlled for.

Few definitive data exist to guide perioperative management of patients with OSA. It is not surprising that many anesthesiologists question whether OSA patients are appropriate candidates for ambulatory surgery. The risks of caring for these challenging patients in the ambulatory venue are further amplified by the unfortunate fact that 80 to 95% of people with OSA are undiagnosed;[3,7] they have neither a presumptive clinical and/or a sleep study diagnosis of OSA. This is concerning because these patients may suffer perioperatively from life-threatening desaturation and postoperative airway obstruction. Moreover, serious comorbidities may be present because prolonged apnea results in hypoxemia and hypercarbia, which can lead to increased systemic and pulmonary artery pressures and dysrhythmias. Cor pulmonale, polycythemia, and congestive heart failure may develop.

Pathophysiology and Therapy

Sleep apnea occurs when the negative airway pressure that develops during inspiration is greater than the muscular distending pressure. Obstruction can occur throughout the upper airway, above, below, or at the level of the uvula.[8,9] Because there is an inverse relationship between obesity and pharyngeal area, the smaller size of the upper airway in the obese patient causes a more negative pressure to develop for the same inspiratory flow. [9,10] Kuna has also postulated that there may be a neurological basis for the disease in that the neural drive to the airway dilator muscles is insufficient or not coordinated appropriately with the drive to the diaphragm. [9] Indeed, it has been hypothesized that OSA is a state-dependent disease possibly caused by complicated neuroanatomical interactions. During wakefulness OSA patients have increased basal genioglossus activity to compensate for their narrower, more collapsible airway. However, neural compensation for anatomic abnormalities that are operative during wakefulness is lost during sleep. [11] Isono has underscored that pharyngeal wall collapsibility is exacerbated by the reduced lung volumes associated with obesity. [12] The caudal tracheal traction that occurs during inspiration is reduced in obese, supine adults. This traction is thought to increase longitudinal tension of the pharyngeal airway wall, thereby stiffening the airway. [13] Isono, therefore, is emphatic that the safe management of OSA should focus on improving the pharyngeal anatomical imbalance and maintaining lung volume. [12]

Obstruction can occur during any sleep state, but is often noted during rapid eye movement (REM) sleep. Nasal continuous positive airway pressure (CPAP) can ameliorate the situation by keeping the pressure in the upper airway positive, thus acting as a "splint" to maintain airway patency and increasing the cross-sectional area. Interestingly, it has also been shown that CPAP reduces leptin levels and facilitates weight loss.

The site(s) of obstruction can be determined preoperatively by such techniques as magnetic resonance imaging, computed tomography studies, and intraluminal pressure measurements during sleep. [14] Some studies suggest that the major site of obstruction in most patients is at the oropharynx, but obstruction can also occur at the nasopharynx, the hypopharynx, and the epiglottis. [15] Obviously, if the surgery is designed to relieve obstruction at one area but pathology extends to other sites, [16] postoperative obstruction is not only possible but probable, especially when one allows for edema associated with airway instrumentation.

CPAP devices, at least until the recent past, were often not well tolerated by patients. However, many technological advances have been made with positive airway pressure devices, making these gadgets more easily tolerated. Additionally, weight loss may improve OSA. Recently atrial overdrive pacing has shown promising results in patients with central or OSA. [17] French investigators serendipitously observed that some patients who had received a pacemaker with atrial overdrive pacing to reduce the incidence of atrial dysrhythmias reported a reduction in sleep disorders after pacemaker implantation. These cardiologists, therefore, initiated a study to investigate the efficacy of atrial overdrive pacing in the treatment of sleep apnea symptoms in consecutive patients who required a pacemaker for conventional indications. They found that atrial pacing at a rate 15 beats per minute faster than the mean nocturnal heart rate resulted in a significant reduction in the number of episodes of both central and obstructive apnea. [17] Postulating that enhanced vagal tone may be associated with (central) sleep apnea, the investigators acknowledged, however, that the mechanism of the amelioration of OSA by atrial overdrive pacing is unclear. Moreover, whether these unexpected findings are germane to the sleep apnea patient with normal cardiac function is uncertain. Gottlieb [18] has tantalizingly suggested that a central mechanism affecting both respiratory rhythm and pharyngeal motor neuron activity would offer the most plausible explanation for the reported equivalence in the improvement of central and OSA during atrial overdrive pacing. Do cardiac vagal afferents also inhibit respiration? Perhaps identification of specific neural pathways might also advance efforts to develop pharmacologic treatment for sleep apnea.

A variety of surgical approaches to treating sleep-related airway obstruction are available. They include classic procedures, such as tonsillectomy, that directly enlarge the upper airway, as well as more specialized procedures to accomplish the same objective. Examples of the latter include uvulopalatopharyngoplasty (UPPP), uvulopalatal flap (UPF), uvulopalatopharyngoglossoplasty (UPPGP), laser midline glossectomy (LMG), lingualplasty (LP), inferior sagittal mandibular osteotomy and genioglossal advancement (MOGA), hyoid myotomy (HM) and suspension, and maxillomandibular osteotomy and advancement (MMO). Another approach is to bypass the pharyngeal part of the airway with a tracheotomy.

Although physicians and surgeons have been treating OSA for more than 25 years, a paucity of long-term, standardized results about the efficacy of different therapies are available. One recent report, however, suggests that at least 50% of patients with sleep apnea syndrome can be managed effectively with one or a combination of therapies. Nasal CPAP, tracheotomy, MMO, and tonsillectomy typically receive high marks for efficacy, [19] and a recent study of UPPP showed positive results that were maintained for a minimum of one year. [20] Another study, combining UPPP with genioglossus and hyoid advancement, reported encouraging results in patients with mild and moderate OSA and multilevel obstruction. [21] However, concern about the long-term results of laser-assisted uvulopalatoplasty (LAUP) for the management of OSA was recently voiced. [22] The response has been characterized as varied and

unpredictable. It appears that the favorable subjective short-term results of LAUP deteriorated in time. Postoperative polysomnography revealed that LAUP might lead to deterioration of existing apnea. These findings are probably related to velopharyngeal narrowing and progressive palatal fibrosis inflicted by the laser beam.

Should Patients with OSA Undergo Ambulatory Surgery?

There is serious and thoughtful ongoing debate about whether OSA patients should undergo surgery as outpatients. Clearly, there is no one-size-fits-all solution. [7] According to Guidelines published in 2006 by the American Society of Anesthesiologists (ASA), [23] when deciding a management strategy it is important to consider the patient's body mass index and neck circumference, the severity of the OSA, the presence or absence of associated cardiopulmonary disease, the nature of the surgery, and the anticipated postoperative opioid requirement. The extent of fat accumulation in the intraabdominal region, which is associated with the metabolic syndrome and the secretion of hormones and proinflammatory cytokines that may perhaps influence breathing in obese OSA patients, should also be considered. [24] Although multiple screening tests for OSA are available, such as the Berlin questionnaire, the STOP-Bang instrument, the ASA checklist, and the Kushida morphometric index, they are highly accurate for detecting only severe OSA, producing high false negative rates for identifying mild OSA. [25] Polysomnography is the gold standard – and a cumbersome and expensive one – for detecting and quantifying the severity of OSA. Absent polysomnography results, the clinician must rely heavily on the clinical history and physical examination.

It seems reasonable to expect that OSA patients without multiple risk factors who are having relatively noninvasive procedures (carpal tunnel repair, breast biopsy, knee arthroscopy, etc) typically associated with minimal postoperative pain may be candidates for ambulatory status. However, those individuals with multiple risk factors, or those OSA patients having airway surgery, most probably will benefit from a more conservative approach that includes postoperative admission and careful monitoring. [23] Indeed, the ASA Guidelines specifically state that adult airway surgery, tonsillectomy in children < 3 years of age, and laparoscopic surgery involving the upper abdomen are inadvisable outpatient procedures for patients with OSA. It is imperative to appreciate that OSA patients are exquisitely sensitive to the respiratory depressant effects of opioids. Additionally, the risk of prolonged apnea is increased for as long as one week postoperatively.

Is perioperative risk related to the type of anesthesia (general, regional, or monitored anesthesia care) administered? The limited evidence suggests that the type of surgery probably supercedes in importance the selection of anesthetic technique. Certainly, the use of regional anesthesia, although strongly recommended by the ASA, may not necessarily obviate the need for securing the airway, and may even require emergency airway intervention if excess sedative-hypnotics or opioids are administered, an intravascular injection inadvertently occurs, or if a high level of neuraxial blockade is obtained. Regardless of the type of anesthesia selected, sedation should be administered judiciously. CPAP or noninvasive positive pressure ventilation (NIPPV) should be administered as soon as feasible after surgery to patients who were receiving it preoperatively. The supine position should be avoided when possible during recovery. The sitting position allows for improved lung volumes, considered beneficial in terms of minimizing pharyngeal collapsibility. Moreover, it is important to be aware that the ASA Guidelines state OSA patients should be monitored postoperatively for 3 hours longer than usual, and for 7 hours after the last episode of obstruction or room air hypoxemia. Patients should be awake and alert, have an oxygen saturation within 2% of baseline, and have minimal pain and postoperative nausea/vomiting at the time of discharge. If the patient requires an oral opioid for analgesia, the effect of this medication should be observed for at least an hour before discharge

When confronted with an especially challenging OSA patient requiring general anesthesia, a judicious approach may include awake fiberoptic intubation, administering very low-dose, short-acting opioids, short-acting muscle relaxants, and a low solubility inhalational agent, as well as infiltrating the surgical site with a long-acting local anesthetic. Extubation should be performed only when the patient is without residual neuromuscular blockade and fully awake, leaving a tube changer or catheter until the patency of the airway is established.. Indications for admission include a difficult intraoperative or postoperative course, the requirement for parenteral opioids to manage pain postoperatively, and severe OSA with notable comorbidities. In the interest of patient safety, one should have a high index of suspicion and a low threshold for admitting patients. "When in doubt, admit." Obviously, this option is best implemented when the surgery is conducted in a hospital-based ambulatory facility. Hence, different criteria for patient and procedure selection may – or should – apply in free-standing facilities.

Summary

Anesthetic care of the OSA patient is especially challenging, and few definitive data are available to guide perioperative management. Recommendations are based more on expert opinion and consensus than on evidence. The anesthesiologist should begin by having a high index of suspicion for the diagnosis, and then seek to identify and quantify associated comorbidities. The major focus of the anesthesiologist of necessity must be on establishing and maintaining the airway, a challenge that will extend well into the postoperative period. Depending on the type of surgery, the anticipated amount of opioid required postoperatively to manage pain, and the patient's condition, outpatient surgery may not be prudent. The resources of the facility must also be factored into the decision whether to accept an OSA patient. The roles that effective communication, monitoring, vigilance, judgment, and contingency planning play cannot be overemphasized.

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