Obstructive Sleep Apnea, Anesthesia, and Ambulatory Surgery

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There are 3 different pharynxes (Figure 1). The nasopharynx, or velopharynx, is the airspace posterior to all of the soft palate; the oropharynx is the airspace posterior to the tip of the uvula to the tip of the epiglottis; and the laryngopharynx, or hypopharynx, is the airspace posterior to the tip of the epiglottis to the vocal cords.

The adult human is one of only a few animals to have a space between the uvula and the epiglottis. All other mammals, and even the newborn human, have an interlocking uvula and epiglottis. As the newborn grows, the larynx descends and the space between the uvula and epiglottis increases. As a result, only the adult human has an upper airway that is essentially a long, soft-walled tube with no bony support anteriorly or laterally. This long, soft-walled tube gives the adult human the potential to have obstructive sleep apnea (OSA).1

The tube remains open as a result of the action of muscles. The muscles that keep the upper airway open are as follows2:

- The tensor palatine retracts the soft palate away from the posterior pharyngeal wall, thereby maintaining the patency of the retropalatal nasopharynx.
- The genioglossus moves the tongue anteriorly to open the retroglossal oropharynx.
- The geniohyoid, sternohyoid, and thyrohyoid muscles move the epiglottis forward by tensing the hyoepiglottic ligament, thereby enlarging the retroepiglottic laryngopharynx.

When we sleep, muscle tone throughout the body is lost; the deeper the sleep, either natural or pharmacologically induced, the greater the relaxation. The 2 major stages of sleep are non–rapid eye movement (NREM) and rapid eye movement (REM). Within NREM sleep are 4 substages that represent progressive slowing of brain electrical activity. Deep and restorative sleep occurs during the deeper NREM and REM sleep stages, and it is nocturnal deep and restorative sleep that allows one to function the next day without requiring diurnal sleep.

During deep and restorative sleep, the pharyngeal muscles participate in the loss of muscle tone. The loss of pharyngeal muscle tone always causes some extent of pharyngeal collapse. If the loss of pharyngeal muscle tone and pharyngeal collapse is partial but sufficient to cause the inspired air to flutter around the uvula, tongue, or epiglottis, snoring and hypopnea (formally defined as a decrease in air flow more than 50% of awake value for more than 10 seconds) will result.3 If the loss of pharyngeal muscle tone and pharyngeal collapse leads to complete obstruction, the result will be silence and apnea.3 Apnea, defined as no air flow for longer than 10 seconds despite continuing ventilatory effort, and hypopnea during sleep are considered sleep-disordered breathing (Figure 2).

To survive each obstructive episode, the patient must have some sort of arousal. In the vast majority of instances, the period of arousal is brief. These periods of “mini-arousal” are expressed in the brain as bursts on an electroencephalograph, as extremity movement or turning, vocalization, or some combination of the 3.
Mini-arousal activates the pharyngeal muscles, which open the pharyngeal airway—often accompanied by a snorting noise. The reopened airway allows the patient to resume deep sleep, leading in turn to relaxation of the pharyngeal musculature, pharyngeal collapse, and pharyngeal obstruction. Thus, the cycle of deep sleep, pharyngeal obstruction, arousal, deep sleep, and so forth, repeats.

There are 4 mechanisms of arousal: During the first and second mechanisms, the duration of the apnea or the progression of hypopnea results in an increase in the magnitude of arterial hypoxemia and hypercapnia. Third, the chemical changes caused by increases in hypoxemia and hypercapnia induce an increased ventilatory effort. Finally, the increased ventilatory effort against an obstructed airway causes increased negative pressure in the airway. Any or all mechanisms together can increase neural traffic in the reticular activating system and arouse the individual.

Obviously, arousals from apneic states are necessary for survival. However, each arousal stimulates the sympathetic nervous system, which in turn causes systemic and pulmonary hypertension (Figure 3). Furthermore, patients with OSA are 5 to 9 times more likely to have metabolic syndrome, and vice versa. In time, hypertension in one or both circulations becomes fixed, leading to ventricular hypertrophy. Hypoxemia can cause myocardial ischemia, which can cause arrhythmias and sudden and unexpected death. The increased negative intrathoracic pressure can cause reflux from the positive-pressure stomach to the negative-pressure esophagus, increasing the incidence of gastroesophageal reflux disease. Data from studies with dogs suggest that mild degrees of nocturnal negative-pressure pulmonary edema may occur. Repetitive arousals fragment the sleep, robbing the patient of restorative sleep and resulting in somnolence during the day; a high risk for motor vehicle accidents; and personality, behavior, and cognitive changes. Finally, because they are prone to loud snoring and continuous movements during sleep, individuals with OSA often are undesirable bed partners and suffer from nocturnal social isolation.

**Conditions Associated With OSA**

The anesthesiologist must know the conditions associated with OSA because it is unreasonable to ask all patients questions that allow a presumptive clinical diagnosis of the condition. If a cause is present, such as obesity, or thick or fat neck, it is reasonable to investigate whether OSA exists.
Obesity. Obesity is by far the leading cause of OSA in the United States. Obesity is best expressed quantitatively as a body mass index (BMI) greater than 30 kg/m². BMI can be calculated as mass/height² = kg/m² or lb x 703/in², where underweight, normal weight, overweight, obesity, and morbid obesity equal less than 19, 19 to 24.9, 25 to 29.9, 30 to 34.9, and greater than 35, respectively.7

Obese patients are uniquely susceptible to OSA because there is an inverse relationship between pharyngeal area and obesity (weight/height).8 Studies using magnetic resonance imaging show that decreased pharyngeal area results from deposition of adipose tissue in the uvula, tonsils, tonsillar pillars, tongue, arypepiglottic folds, and the lateral pharyngeal walls,9 and the volume of fat deposited in these structures correlates well with the severity of OSA.10 From the sites of fat deposition, it is obvious that all 3 pharynxes are involved. The converse also is true: Weight loss results in a significant decrease in the severity of OSA.11 Thus, when an obese patient goes into a deep sleep and there is a given degree of loss of pharyngeal muscle tone and pharyngeal collapse, the more posterior the tongue, the greater the degree of pharyngeal obstruction.

Thick/Fat Neck. The patency of the soft-walled, collapsible pharynx is determined by the transmural pressure across the wall, which is the difference between extraluminal and intraluminal pressure. In obese patients, extraluminal pressure is increased by superficially located fat masses.12 Thus, when an obese patient goes into a deep sleep and there is a given degree of loss of pharyngeal muscle tone and pharyngeal collapse, the greater the amount of intraluminal fat, the greater the pharyngeal obstruction.

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Large Tongue (high oropharyngeal classification). For a given degree of loss of pharyngeal muscle tone and pharyngeal collapse, the larger the tongue, the greater the degree of pharyngeal obstruction.

Enlarged Tonsils. For a given degree of loss of pharyngeal muscle tone and pharyngeal collapse, the larger the tonsils, the greater the degree of pharyngeal obstruction.

Nasal Obstruction (any etiology). For a given degree of loss of pharyngeal muscle tone and pharyngeal collapse, the greater the degree of nasal obstruction, the greater the degree of pharyngeal obstruction.

If a cause of OSA is known to be present in a given patient, the anesthesiologists should ask questions to help confirm or exclude a presumptive clinical diagnosis of OSA.

OSA and Anesthesia: A Multidimensional Problem

The literature indicates that disastrous respiratory outcomes during the perioperative management of patients with OSA are a major and increasing problem for the anesthesia community.2,14-16 The disastrous outcomes are due to 1) failure to secure the airway during the induction of anesthesia, 2) respiratory obstruction...
soon after extubation, and 3) respiratory arrest after the postoperative administration of opioids and/or sedation to extubated patients. The growing OSA management problem is almost certainly fueled by the growing obesity epidemic; the large majority of these patients (70%-90%) are obese (BMI >30 kg/m²). In 1993, the number of people in the United States with clinically significant OSA was approximately 19 million. Given the increase in obesity, this number almost certainly is much higher in 2011. It is estimated that a large proportion of the general population, 24% of men and 9% of women, have mild OSA. Moderately severe OSA is present in approximately 11.4% of men and 4.7% of women. The prevalence among obese individuals is even higher, with reported rates at 50% of men and 40% of women. Furthermore, an estimated 82% of men and 93% of women with OSA remain undiagnosed. Among patients presenting for surgery, OSA is more prevalent than in the general population. Failure to manage the intubation, extubation, and pain of patients with OSA often results in brain damage or death.

PREOPERATIVE EVALUATION

The American Society of Anesthesiologists (ASA) Task Force on Sleep Apnea Guidelines are best understood if the underlying logic is well appreciated (Figure 4). The ASA advocates performing a complete formal preoperative review for OSA, including a review of the medical records, physical examination, sleep studies, and preoperative x-rays in certain cases. The causes and conditions associated with OSA must be known. If one or more causes (such as morbid obesity) are present in the patient, questions to ascertain the presumptive clinical diagnosis should be asked. If a presumptive diagnosis of OSA is made, the severity of the OSA must be determined. The severity of OSA will help determine perioperative risk, along with other factors including the invasiveness of anesthesia and surgery, and the anticipated requirements of opioids after surgery.

A significant number of patients arrive for surgery lacking a formal diagnosis of OSA. Therefore, the anesthesiologist must identify the patient with OSA in the preoperative setting. This can be performed by incorporating established screening assessments, such as the Berlin questionnaire, Flemons questionnaires, the ASA checklist, the STOP questionnaire and its STOP-BANG modification (Table). Most of the assessments include patient self-reporting of snoring, choking episodes, apneic episodes, arousal from sleep, daytime somnolence, and the presence of hypertension. They also vary rely on physical characteristics such as gender, BMI, neck and waist circumference, airway anomalies, and age.

The gold standard for OSA diagnosis is polysomnography (PSG), which reports an apnea-hypopnea index (AHI) as a measure of severity of disease. OSA is defined by PSG as the occurrence of at least 5 episodes of apnea or hypopnea in 1 hour, or an AHI of 5. Moderate and severe OSA is defined as an AHI greater than 15 and 30, respectively.

Figure 4. The logic of the ASA sleep apnea guideline.

The sensitivity of the screening tools discussed here ranges from 54% to 100% and varies such that the sensitivity generally increases with increasing AHI. In contrast, the specificity of these tools is less optimal, ranging from 38% to 60%, and decreases as the AHI increases. Importantly, an extremely sensitive test resulting in an excessive number of false-positives may lead to diminished cost-effectiveness. The ASA Task Force on Perioperative Management of Patients with Obstructive Sleep Apnea estimates the annual cost of implementing its guidelines is $80,000, with an additional annual cost to an outpatient facility of $50,000.

The tremendous backlog for PSG testing makes timely preoperative testing difficult. Portable home sleep testing (HST) should be considered, as it has the advantage of being administered at home, returned in a 24-hour time period for results, and has been validated against PSG with comparable sensitivity and specificity.

The utility of obtaining a formal preoperative diagnosis of OSA and subsequent positive airway pressure (PAP) therapy, in contrast to screening, is not yet clear. In 2 relevant studies, postoperative institution of PAP therapy failed as a result of patient noncompliance and the inability to prevent desaturation in the first 24 hours. However, in a recent study, Minokadeh and Davidson (unpublished data) showed increased postoperative PAP compliance when a diagnosis and therapy were instituted preoperatively. Preoperative patients were screened for OSA using the STOP questionnaire. Those who screened positive were given a multichannel sleep test to administer at home. Patients with an AHI greater than 15 were placed on an auto-titrating positive airway pressure (APAP) machine, with the expectation that they would learn to use it in the days prior to surgery and continue to use it throughout the first postoperative week. The preliminary conclusion is that patients with OSA can be screened, tested, and compliant with APAP for the first postoperative week.

The University of California, San Diego Medical Center
INTRAOPERATIVE MANAGEMENT

The use of a short-acting sedative, induction agents, and volatile anesthetics can be helpful. However, the practitioner must keep in mind that the termination of the clinical action of agents such as midazolam, fentanyl, propofol, and even sevoflurane and desflurane, is based on redistribution of the agent from rapidly perfused tissues rather than by metabolism of the drugs. The use of high doses of these agents may result in a significant tissue concentration that is only slowly cleared from the body, resulting in prolonged sedation.

Because opioids have the strongest respiratory depressant effect of all anesthetic agents, the use of remifentanil, which is rapidly metabolized by blood and tissue esterases, may be preferred over other relatively short-acting alternatives. The sedative dexmedetomidine (Precedex, Hospira) has a narcotic-sparing effect, causes minimal respiratory depression, and may be a useful adjunctive agent.

The endotracheal tube (ETT) is significantly more stimulating when compared with mask airway devices such as the laryngeal mask airway (LMA, LMA North America). As a result, the overall anesthetic requirement is likely to be greater with ETT-based anesthesia than when LMAs are used. Many OSA patients are obese, have difficult airways, and experience gastroesophageal reflux. The anesthesiologist must carefully consider the risks and benefits associated with using an LMA in this population of patients.

If airway trauma is a possibility, either because of the surgical procedure or a difficult intubation, administration of an intraoperative steroid may be warranted to reduce airway swelling.

When muscle relaxants are used, the practitioner must ensure that the agent has been fully reversed at the end of the surgical procedure and prior to extubation. Great care should be given to extubation of patients with OSA to ensure that their airway reflexes return with adequate tidal volume, respiratory rate, and acceptable concentrations of end-tidal carbon dioxide (etCO₂).

POSTOPERATIVE MANAGEMENT AND DISPOSITION

The primary challenge during the immediate postoperative period in the postanesthesia care unit (PACU) is the balance between pain control and adequate respiration. The initial use of short-acting IV narcotics for rapid pain control must ultimately shift to the use of longer-acting IV narcotics and, if hospital discharge is anticipated, oral pain medications. If appropriate, nonnarcotic analgesics such as ketorolac should be used early in the course of PACU treatment.

During the titration of pain control, the PACU staff should carefully observe the patient for respiratory events such as bradypnea, apnea, arterial oxygen desaturation, inability to wean from supplemental oxygen, and pain-sedation mismatch (ie, concurrent high pain and sedation scores). Some patients may require positive airway pressure (PAP) therapy in the PACU to achieve acceptable respiratory status (Figure 5).

Table. The STOP and STOP-BANG Questionnaires.

<table>
<thead>
<tr>
<th>The STOP and STOP-BANG questionnaires ask patients the following:</th>
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<tr>
<td>Do you Snore loudly?</td>
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<tr>
<td>Do you often feel Tired, fatigued, or sleepy during daytime?</td>
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<tr>
<td>Has anyone Observed you stop breathing during your sleep?</td>
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<tr>
<td>Have you ever been or are you now being treated for high blood Pressure?</td>
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<tr>
<td>Is your Body mass index greater than 35 kg/m²?</td>
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<tr>
<td>Are you over 50 years of Age?</td>
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<tr>
<td>Is your Neck circumference greater than 40 cm?</td>
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<tr>
<td>Are you male [Gender]?</td>
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<tr>
<td>High-risk OSA: STOP, yes to 2 or more questions. STOP-BANG, yes to 3 or more questions.</td>
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OSA, obstructive sleep apnea

has chosen to use the STOP questionnaire as the initial preoperative screening tool for OSA based on its simplicity of use, acceptable sensitivity and specificity profile, and the ease of conversion to the more sensitive STOP-BANG. Patients deemed to be high risk by these questionnaires in the preoperative setting are then flagged on the operating room schedule for appropriate considerations in intraoperative management and postoperative monitoring.

INTRAOPERATIVE MANAGEMENT

Virtually all IV and volatile anesthetic agents act as respiratory depressants and muscle relaxants. Therefore, it is prudent to minimize the use of these agents within the parameters of safe anesthetic practice. The anesthetic technique should prevent intraoperative awareness, maintain hemodynamic stability, and provide a quiet operative field and optimal postoperative pain control while ensuring the smallest overall cumulative anesthetic dose.

When a general anesthetic technique is mandated by the planned operative procedure, options exist that can reduce the amount of postprocedure burden of residual anesthetic.

The use of regional anesthesia where appropriate, either as the primary or as an adjunctive anesthetic technique, may reduce the incidence of perioperative respiratory complications in patients with OSA and should be strongly considered.28 When local anesthetic infiltration is possible, particularly at the beginning of the surgical procedure, the total anesthetic burden can be significantly reduced.

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Strong clinical data show that the presence of respiratory events in the PACU predict respiratory events in the post-PACU period.\textsuperscript{36,37} The ASA guidelines on OSA recommend PACU observation from 3 to 7 hours for patients without and with observed respiratory events in the unit, respectively.\textsuperscript{28}

If respiratory events do not occur, or if they occur but resolve during the observation period, the patient may be considered for discharge home or to an unmonitored bed in a surgical unit.

No concrete, evidence-based guidelines guarantee safe discharge to an unmonitored setting, so the practitioner must rely on experience and clinical judgment. The decision should be made with attention to the type of anesthesia the patient received, the type of surgery, the expected level of postoperative pain, the type of postoperative opioid prescribed, the patient’s age, and the sophistication of home support (Figure 5). As the consequences of an inappropriate discharge are potentially life-threatening, the default decision should be for a monitored hospital bed in borderline cases.

If respiratory events do not resolve or if the patient requires assistance to maintain acceptable respiratory status, the patient must be admitted to a hospital bed with continuously observed SpO\textsubscript{2} and etCO\textsubscript{2} monitoring. Capnometry can alert the providers to apnea or hypopnea in order to intervene before the late sign of oxygen desaturation and resultant cardiac arrest.

**Figure 5. University of California, San Diego Perioperative OSA Algorithm.**

BIPAP, bilevel positive airway pressure; CO\textsubscript{2}, carbon dioxide; CPAP, continuous positive airway pressure; OR, operating room; OSA, obstructive sleep apnea; PACU, postanesthesia care unit; SpO\textsubscript{2}, oxygen saturation as measured by pulse oximetry.
Conclusion

Perioperative management of OSA is a multidimensional problem that requires the joint efforts of the primary care physician, anesthesiologist, and surgeon. The preoperative clinic is the proper starting point to screen for the condition. A firm diagnosis or high index of suspicion will lead to thoughtful intraoperative and postoperative planning. Postoperative PACU respiratory monitoring will identify patients at increased risk for post-PACU respiratory events and desaturation. These high-risk patients must be admitted to a monitored hospital unit where pulse oximetry and capnometry can help to identify patients who require intervention, including PAP therapy. The implementation of appropriate perioperative protocols for the care of the patient with OSA is the best protection against the perioperative complications associated with this life threatening and unfortunately, common syndrome.

References