

Upper Airway Management of the Adult Patient with Obstructive Sleep Apnea in the Perioperative Period - Avoiding Complications

Clinical Practice Review Committee
American Academy of Sleep Medicine

Amy Lynn Meoli, MD¹; Carol L. Rosen, MD²; David Kristo, MD³; Michael Kohrman, MD⁴; Nalaka Gooneratne, MD⁵; Robert Neal Aguillard, MD⁶; Robert Fayle, MD⁷; Robert Troell, MD⁸; Ronald Kramer, MD⁹; Kenneth R. Casey, MD, MPH¹⁰; Jack Coleman, Jr., MD, FACS¹¹

¹St. John's Regional Medical Center, Joplin, MO; ²Case Western Reserve University School of Medicine, Department of Pediatrics, Rainbow Babies and Children's Hospital, Cleveland OH; ³Walter Reed Army Medical Center, Pulmonology/Critical Care, Washington, D.C.; ⁴University of Chicago, Pediatric Neurology, Chicago, IL; ⁵University of Pennsylvania, Division of Geriatric Medicine and the Center for Sleep and Respiratory Neurobiology, Philadelphia, PA; ⁶Methodist Hospital, Methodist Sleep Disorders Center, Memphis, TN; ⁷University of Texas Medical School at Houston, Park Plaza Hospital, Sleep Disorders Center, Houston, TX; ⁸Beauty by Design, Las Vegas, NV; ⁹Colorado Sleep Disorders Center, Englewood, CO; ¹⁰Sleepwell Diagnostic Center, South Jordan, UT; ¹¹Middle Tennessee Medical Center, Murfreesboro, TN

Purpose: To help practitioners avoid adverse perioperative events in patients with obstructive sleep-disordered breathing.

Reviewers: Members of the American Academy of Sleep Medicine's Clinical Practice Review Committee.

Methods: A search of MEDLINE database using MeSH terms apnea, obstructive sleep apnea and anesthesia was conducted in October 2001. This review focuses on articles published in English between 1985 and 2001 that pertain to non-upper airway surgery in obstructive sleep apnea patients.

Results and Conclusions: Scientific literature regarding the perioperative risk and best management techniques for OSAHS patients is scanty and of limited quality. There is insufficient information to develop an AASM standards of practice recommendation. Therefore, the Clinical Practice Review Committee (CPRC) used the available data to make this statement based upon a consensus of clinical experience and published peer-

reviewed medical evidence. Important components of the perioperative management of OSAHS patients include a high degree of clinical suspicion, control of the airway throughout the perioperative period, judicious use of medications, and appropriate monitoring. Further research is needed to define the magnitude of risk and optimal perioperative care.

Synonyms: Obstructive Sleep Apnea Hypopnea Syndrome (OSAHS), Obstructive Sleep Apnea Syndrome (OSAS), Sleep Disordered Breathing (SDB), and Obstructive Apnea-Hypopnea Syndrome (OAHS).

Key Words: Obstructive Sleep Apnea; Sleep Disordered Breathing; Apnea; Anesthesia; Perioperative

Citation: Report of the Clinical Practice Review Committee, American Academy of Sleep Medicine. Upper airway management of the adult patient with obstructive sleep apnea in the perioperative period - avoiding complications. *SLEEP* 2003;26(8):1060-5.

1.0 INTRODUCTION

OBSTRUCTIVE SLEEP APNEA HYPOPNEA SYNDROME (OSAHS) IS A PREVALENT CONDITION THAT IS THOUGHT TO RESULT FROM DECREASED UPPER AIRWAY PATENCY IN SLEEP COMPARED TO WAKEFULNESS. In obstructive sleep-disordered breathing, the airway completely (apnea) or partially (hypopnea) occludes despite continued respiratory effort. Arousals from sleep temporarily restore upper airway patency, only to be followed by a repetitive cycle of airway collapse and arousal. This phenomenon produces sleep fragmentation and can lead to significant nocturnal hypoxemia. Young and colleagues reported that the estimated prevalence of sleep-disordered breathing, defined as an apnea-hypopnea index of five obstructed events per hour of sleep or higher, was 9% for women and 24% for men between the ages of 30 and 60.¹ OSAHS may produce significant complications including an increased risk of hypertension,² cardiac arrhythmias,³ myocardial infarction,⁴ and stroke.^{5,6}

Apart from the risks of OSAHS itself, patients with disordered breathing during sleep may also be at risk of complications related to anesthesia and postoperative analgesia. Most published reports and reviews focus on patients undergoing surgical treatments specifically intended to treat OSAHS; much less is known about the perioperative management of OSAHS patients related to non-upper airway surgery.^{7,8,9,10,11,12,13,14}

The risks associated with OSAHS may be due to the sleep-disordered breathing and associated pathophysiologic processes, or to difficult airway control (e.g., related to obesity or abnormal anatomy), associated comorbidity (e.g. hypoventilation, cardiovascular vulnerability, etc.) or other causes. Several recent reviews note that the role of OSAHS as a risk factor for anesthetic morbidity and mortality is considerable but not well defined.^{15,16,17,18} Furthermore, no consensus exists regarding optimal perioperative management of patients with OSAHS.

Sometimes the anesthesia care provider's first introduction to a patient occurs immediately prior to surgery. Without the availability of a quick screening test or a reliable clinical profile assessment, practitioners need to consider both the risk factors for and the perioperative management of potential sleep-disordered breathing in each patient. Unfortunately clinical presentation alone may be a poor predictor of the presence of OSAHS.¹⁹ The goals of this review are to synthesize available data that may help to identify patients at risk for OSAHS and to suggest anesthetic techniques and perioperative management that may minimize complications.

2.0 METHODS

A literature search using Medline was conducted using the terms *apnea*, *OSAHS* and *anesthesia* during October, 2001. The search was limited to articles published in English between 1985 and 2001. Articles identified were reviewed and additional, pertinent publications from bibliographies of selected references were also included. The literature review focused on the perioperative risk and management strategies in patients undergoing surgery other than that directed at treatment of

Address correspondence to: Address correspondence to: Clinical Practice Review Committee care of American Academy of Sleep Medicine One Westbrook Corporate Center, Ste 920, Westchester, IL 60154; <http://www.aasmnet.org/positionpapers.htm>

OSAHS. Data was summarized and considered by members of the Clinical Practice Review Committee (CPRC). The CPRC is a multidisciplinary AASM committee formed to develop statements regarding clinical issues that have too little scientific data to support a Standards of Practice guideline. A consensus of members was used to review available scientific evidence and formulate the recommendations made in this article. Relevant outside experts were consulted as needed. **The results in this paper are based on review of available data and expert opinion, but are not, in most cases, evidence based.**

3.0 OSAHS AND ANESTHESIA: FACTORS CONTRIBUTING TO PERIOPERATIVE RISK

The incidence of perioperative complications in OSAHS patients undergoing “non apnea” surgery is unknown. Several factors may contribute to the development of complications, including: 1) increased upper airway instability related to anesthetic agents and narcotic analgesics, 2) cardiopulmonary effects of SDB, 3) reduced functional residual capacity and oxygen reserve resulting from obesity, 4) reduced ventilatory drive resulting from anesthetic agents. Although the impact of general anesthesia on sleep is poorly understood, several characteristics of sleep change significantly in the perioperative period.^{20,21} Many patients lose sleep perioperatively because of anxiety, the disease process requiring operation, pain, circadian rhythm alterations, and nursing activity. Because sleep deprivation exacerbates sleep-disordered breathing, these patients may have more severe apnea as they begin to pay back the sleep debt accumulated perioperatively.¹⁷ Anesthetic and narcotic agents increase the tendency for upper airway collapse. These agents also impair normal arousal mechanisms, thereby worsening apnea severity.²² In a study of 10 young, healthy volunteers (five men) with body weights no more than 20 % greater than ideal, magnetic resonance imaging showed a significant reduction in the minimum anterior-posterior distance at the level of the soft palate in the supine position during propofol anesthesia.²³ No significant change occurred in the minimum diameter at the level of the tongue base or epiglottis. In contrast, lateral radiographs in 29 elderly men without known OSAHS demonstrated significant posterior displacement of the soft palate, tongue base and epiglottis during apnea following thiopentone anesthesia.²⁴ Methodological differences could explain the conflicting findings. Both groups were believed to be free of OSAHS; it is unknown if similar changes would occur in patients with sleep-disordered breathing. In a study of orthopedic patients, Gupta and colleagues demonstrated that the post-operative period, especially the first 24 hours, is particularly hazardous due to the effects of anesthetic agents, pain medications, and adverse positioning.²⁵ Although necessary after some procedures, supine positioning is associated with more frequent obstructive respiratory events during sleep. The relationship between apnea severity and the magnitude of sedation risk is not well defined. In one study of OSAHS patients, a preoperative apnea index over 70 events per hour and minimum oxygen saturation of less than 80% were identified as risk factors for complications, as was the intraoperative use of narcotics.²⁶ Another study demonstrated a positive correlation between OSAHS severity and complication rate.²⁷ Apnea frequency and oxygen saturation nadir were significant predictors of complications in OSAHS patients undergoing upper airway surgery.²⁸ On the other hand, for the orthopedic patients described above, even mild OSAHS posed an increased perioperative risk.²⁵ Thus, while severity of sleep-disordered breathing may correlate with perioperative risk, in some patients, even mild SDB confers significant risk, perhaps because of positioning and analgesia.

4.0 OSAHS AND ANESTHESIA-PATIENTS WITH DIAGNOSED OBSTRUCTIVE SLEEP APNEA

Preoperative evaluation should include assessment of the likelihood of OSAHS. Patients with previously diagnosed OSAHS should be identified, and a copy or results of diagnostic sleep studies should be incorporated in the medical record as verification of the diagnosis. Although

the apnea-hypopnea index, oxygen saturation nadir, associated arrhythmias and sleep architecture, and the effect of body position (25) all provide important information about the nature severity of OSAHS, it is not clear that these measures predict perioperative complications. A level of apnea severity that may not otherwise be significant may become dangerous under the influence of anesthetic agents or analgesics, or with edema of the airway from intubation or surgical alteration.^{11,25} Most patients who are diagnosed with OSAHS are treated with nasal continuous positive airway pressure (CPAP). However, some will have been treated with upper airway surgery, oral appliances or other modalities; or will have refused therapy.

The effective level of CPAP pressure and the need for supplemental oxygen are often established during diagnostic sleep studies and may be used as initial therapy in postoperative patients who have not suffered perioperative cardiorespiratory complications. If anesthesiologists feel uncomfortable with interpreting the results of diagnostic sleep studies, they should consider consulting physicians experienced in the management of patients with OSAHS. Consideration should be given to “medical-alert” jewelry for this condition such as used for diabetes mellitus and heart disease. Patients should be questioned about treatment recommendations and compliance with OSAHS treatment. Patients who have previously undergone surgical treatment for SDB should be evaluated for recurrence since there is a significant tendency for relapse of sleep-disordered breathing after some surgical approaches.²⁹ The continued presence or recurrence of OSAHS symptoms after any treatment modality should be assessed, as well as changes in medications or body weight since the last evaluation. A patient without symptoms who is compliant with treatment can be managed as outlined below. If symptoms and signs of OSAHS persist despite treatment or if the patient has not been receiving therapy, consultation with a sleep specialist is appropriate. If surgery cannot be postponed, these patients may require adjustment of CPAP pressures or even empiric CPAP (*vide infra*, Section 5.4) to eliminate upper airway obstruction when the endotracheal tube is removed.

5.0 OSAHS AND ANESTHESIA: PATIENTS WITH UNDIAGNOSED OBSTRUCTIVE SLEEP APNEA

5.1 Symptoms of Obstructive Sleep Apnea

It is likely that the majority of individuals with OSAHS have not yet been diagnosed.³⁰ Many patients with OSAHS are undiagnosed because they are unaware of the condition or have not sought medical attention. The problem of indentifying sleep apnea is further compounded by the fact that the symptoms of sleep-disordered breathing can be nonspecific, and may be attributed to other causes or remain unmentioned by the patient unless directly questioned. The anesthesiologist may be the first health professional to inquire about sleep and breathing and therefore has an important role in identifying these patients to prevent both short and long-term complications.³² Numerous reviews of the symptoms of OSAHS are available.^{33,34,35} Classic findings include loud and habitual snoring, with or without observed apnea, non-restorative sleep, excessive daytime sleepiness, and a narrow upper airway. Gastroesophageal reflux disease and hypertension are often associated with OSAHS.^{36,37} Having a bedroom companion or another observer of the patient’s sleep present during the preoperative interview may help to uncover many OSAHS findings. A questionnaire may also be useful to save time and to insure that consistent and complete histories are taken (see Appendix A). However, the absence of these findings does not eliminate the possibility of OSAHS. This disorder occurs in non-obese patients, women and in patients with otherwise atypical presentations. It should be noted that the tools suggested here are presented as examples and have not been validated regarding sensitivity or specificity. The individual who is symptomatic but undiagnosed with OSAHS is generally easily recognized. Of particular concern is the undiagnosed individual who is relatively asymptomatic but has associated features that suggest increased perioperative risk, such as obesity or pharyngeal anatomic abnormalities. The rate of development of complications in such patients has not been ade-

quately investigated.

Since OSAHS cannot be excluded on the basis of history and physical alone³⁸ and since clinical screening does not exclude the diagnosis of OSAHS, healthcare providers must maintain a high degree of suspicion and vigilance for OSAHS. In some instances, one must presume the presence of sleep apnea and treat the patient with the same precautions as the individual who has an established diagnosis. In selected cases, it may be judicious to postpone elective surgery until evaluation and effective treatment of coexisting cardiorespiratory disease and sleep-disordered breathing can be undertaken.¹⁴

5.3 Signs of Obstructive Sleep Apnea

The majority of OSAHS patients snore, and many are obese (see Appendix B). A Body Mass Index (BMI) > 25 kg/m² and/or a neck circumference of over 17 inches for men or 16 inches for women have predictive value for the presence of OSAHS.^{39,40,41} Obstructive sleep apnea is more prevalent in men and in post-menopausal women.⁴² Enlarged nasal turbinates, a narrowed mandible or maxilla, retrognathia, macroglossia, and enlarged tonsils and adenoids may be predisposing conditions.⁴³ Anesthesiologists use methods such as the Mallampati score to help gauge ease of intubation. A high Mallampati score predicts difficulty with intubation and should increase the suspicion of OSAHS,⁴⁵ but in one study a high Mallampati score did not differentiate obese patients with symptoms of sleep disordered breathing from those without.⁴⁶ Despite the clinical stereotype, not all OSAHS patients are overweight, middle-aged men.

5.4 Suspected Obstructive Sleep Apnea

If significant sleep apnea is suspected, preoperative evaluation and treatment may be the ideal course to optimize patient safety. If a critical surgical problem exists, the relative risk and benefit of not proceeding immediately with the surgery must be weighed. If the situation permits, a patient suspected of having severe OSAHS should be evaluated prior to surgery by a physician trained in the diagnosis and treatment of sleep apnea.

The ideal management strategy for the suspected but undiagnosed OSAHS patient who requires emergency surgery is not established. It is reasonable to manage any patient suspected of having OSAHS in an anticipatory manner, and the possibility of OSAHS should be considered in virtually any patient who presents difficulty with intubation.^{47,48} Any patient who presents an intubation challenge should be monitored closely for manifestations of airway instability postoperatively. Intraoperative precautions and concerns regarding injudicious use of sedating and analgesic medications are equally important as with patients known to have OSAHS. Empiric CPAP in the postoperative period may be helpful, but this can be difficult because of uncertainty about the acceptance and efficacy of its use in patients who are naïve to it. Experience suggests that CPAP may be poorly tolerated if the patient has no previous experience with this therapy. Mask fitting, assessing the need for humidification, and establishing optimal pressure settings are important issues in the setting of urgent need for airway stabilization, and staff inexperienced in the use of CPAP could increase the possibility of poor tolerance. So-called "auto-titrating" CPAP machines may be useful in this setting, but they should only be used with appropriate patient observation and monitoring.⁴⁹

6.0 CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP)

If tolerated, CPAP is the most efficacious immediate, noninvasive treatment for OSAHS and should be used in the perioperative period at pressures known to be effective in eliminating upper airway obstruction in the particular patient.^{12,14,51} In a study of anesthetized healthy volunteers, nasal CPAP increased pharyngeal volume derived using MRI.²³ Preoperative CPAP use may also lessen upper airway edema.^{14,52} If a known OSAHS patient has had effective CPAP pressure determined in

the remote past, evaluation by their sleep specialist prior to surgery is advisable to determine if treatment can be optimized. Education of ancillary personnel regarding the importance of CPAP use and the need for effective monitoring techniques for OSAHS patients is critical, particularly since CPAP pressure requirements may change after surgery or with the use of anesthetic, sedative and analgesic medications. Furthermore, patients may be incapable of using CPAP without assistance in the postoperative period. Patients may also be reluctant to use CPAP because of pain, nausea, or other discomfort. CPAP can be used in the presence of a nasogastric tube. For surgical procedures that require nasal packing, a full face mask may be needed. For those who fail CPAP therapy perioperatively, a nasal airway and oxygen supplementation may be useful.²³

7.0 ANESTHESIA AND PERIOPERATIVE MANAGEMENT OF DIAGNOSED AND UNDIAGNOSED OBSTRUCTIVE SLEEP APNEA

7.1 Preparation for Intubation

The primary goal in the care of the sleep apnea patient is maintenance of constant airway control either by the patient or by the anesthesiologist. Many authorities suggest that an antireflux agent and antisialagogue should be used and administered well enough in advance to be in effect during intubation.⁵³ Unsupervised preoperative sedation should be avoided. At the time of induction, the airway is best controlled using an oropharyngeal airway that is of the proper size and correctly positioned to hold the base of tongue out of the airway. If the proper size is not available, a nasopharyngeal airway may be used but it must be long enough to extend into the retroglottic portion of the hypopharynx. A three to five minute period of pre-oxygenation is useful in order to decrease the rate of oxyhemoglobin desaturation should intubation prove difficult. Two-person ventilation, one for jaw positioning and mask seal and the other for ventilation, may be needed to mask ventilate an obese patient.⁵⁴ Another consideration is the use of a laryngeal mask airway (LMA).⁵⁵ This device, when properly positioned, can provide a more stable airway than an oropharyngeal airway and may allow better ventilation with higher tidal volumes. It can also be used for later endotracheal intubation if the LMA is deemed inappropriate for the entire case.⁵⁶

7.2 Intubation

In addition to the standard procedures of intubation, there should be careful attention given to potential occlusion of the upper airway in patients with OSAHS. The dose and selection of pharmacologic agents used during the intubation may be important in determining airway stability.^{22,23} Even brief periods without control of the airway by either the patient or the anesthesia provider must be avoided. Intubation over a fiberoptic scope with or without mild sedation should be considered if there is any doubt about the ability to intubate the patient. Considerable care should be exercised in paralyzing an OSAHS patient for intubation. If the patient can be easily ventilated with a mask after initial induction, then a paralyzing agent may be used, preferably a short acting agent such as succinylcholine. In these patients all such drugs should be administered by titration until the desired effect is achieved.⁵⁷ While muscle paralysis may facilitate intubation, the patient can experience dangerous levels of oxygen desaturation before recovery of spontaneous breathing.^{18,57} In the event of unsuccessful intubation, alternative methods of securing the airway must be immediately available, including mask ventilation, an esophageal obturator tube, transtracheal jet ventilation, and equipment for tracheotomy. A surgeon should always be immediately available and prepared to perform an emergency tracheostomy if necessary. However, a surgical airway may be difficult to achieve, especially in obese patients. Other modes of re-establishing airway control may need to be pursued and should be available.

7.3 Type of Anesthetic Agent

The choice between local anesthesia with or without sedation, region-

al anesthesia with or without sedation, or general anesthesia is important. In terms of airway safety, local anesthesia without sedation may be preferable. Even so, the use of analgesics in the post-operative period might create an airway problem. Sedation may be more dangerous for OSAHS patients than general anesthesia because the sedated patient's ability to protect his or her own airway may be dangerously compromised. With general anesthesia administered with proper precaution during induction and awakening, the airway is "controlled" during the procedure. There is controversy regarding the use of epidural opiates in patients with OSAHS. Respiratory depression was reported in a 65 year old man, later diagnosed with central sleep apnea, eight hours after epidural administration of morphine.⁷ Another report exists of epidural analgesia used postoperatively without complication in an OSAHS patient.⁵⁸ Some authorities regard this to be a preferred strategy for management of postoperative pain. Others point out that epidural opiates may significantly depress the patient's respiration and normal arousal mechanisms.^{59,60}

7.4 Extubation

Extubation is another critical time in the management of the known or suspected OSAHS patient because the potential for loss of airway control is again present. "Deep extubation" is not an option for the apnea patient. The hazards include not only loss of airway control but also the risk of postobstructive pulmonary edema as the patient generates negative pressure trying to breathe against a closed glottis or collapsed airway. Adequate muscular tone of the upper airway should be present before the endotracheal tube is removed. Therefore, the patient must be sufficiently awake prior to extubation. The presence of purposeful movement, recovery of neuromuscular integrity demonstrated by neuromuscular blockade monitoring, sustained head lift for a minimum of five seconds and adequate voluntary tidal volume are helpful criteria in determining safety for extubation. Removal of the endotracheal tube should take place in the operating room, recovery room, or special care unit so that control of the airway, if lost, can be reestablished immediately. Immediate maximal head of bed elevation may reduce upper airway collapse, although this may not be sufficient in more severe OSAHS. An appropriately sized oropharyngeal or nasopharyngeal airway should be immediately available. Application of a vasoconstricting nasal spray can improve the nasal caliber as well as reduce the risk of epistaxis during nasal airway placement. A nasal airway placed prior to extubation may facilitate post-extubation nasal ventilation and CPAP application, especially in those with compromised nasal airway or difficult intubation.

Intraoperative narcotic use should be titrated carefully to achieve pain control without respiratory or upper airway compromise. The use of local anesthetic blocks at the end of surgery may be very useful to minimize need for systemic medications. However, local or topical anesthesia may not be appropriate in the upper airway since it has been shown in non-surgical patients to block airway mechanoreceptors that contribute to the arousal stimulus, an important defense mechanism in OSAHS.⁶¹ Narcotic reversal agents should be available but used with extreme caution in these patients because the duration of action may be less than longer-acting narcotic agents, and the patient may have a reduced sensorium and airway instability after the reversal has worn off. Nasal CPAP should be available for use in the recovery room, even if the patient is not currently using this treatment at home.

8.0 THE POSTOPERATIVE PERIOD

8.1 General Principles

The first 24 postoperative hours are probably the most critical time. However, deaths from complications in OSAHS patients have occurred beyond 24 hour postoperatively, perhaps as the accumulated effects of sleep deprivation, including REM sleep rebound, become important.^{62,63} Because of this extended postoperative risk, care should be taken in

selecting patients for outpatient procedures. Patients known to have mild OSAHS are candidates depending upon the procedure performed and the presence of comorbid disease. Patients with pre-existing tracheostomies may also do well with outpatient procedures. Analgesic dosing must be carefully titrated to ensure that adequate pain relief is given without compromising upper airway muscle tone. It may not be appropriate to use patient controlled analgesia (PCA) in OSAHS patients without carefully establishing dosing limits related to airway and respiratory stability. Potential synergistic effects of medications should always be considered, especially with regards to central nervous system depression. Sedative-hypnotics, anxiolytics, and other sedating medications should be used with caution. Patient monitoring is important to gauge medication effect. Nasal CPAP can provide vital airway support, even in OSAHS patients who have not been using it at home.^{12,14,50} Prescribed CPAP pressures should be initiated and the patient should be monitored for evidence of impaired gas exchange and upper airway obstruction. Continuous pulse oximetry and heart rate monitoring with preset alarms should be used in the immediate postoperative period. If there is clinical evidence of snoring or obstructive apnea while CPAP is being administered, pressures may need to be increased to eliminate upper airway obstruction.

8.2 Generic Patient Care Protocols.

Individual institutional patient care protocols should be examined to determine appropriateness for the sleep apnea patient. If the generic protocol does not provide adequate levels of postoperative monitoring, individual alterations in care are appropriate.

8.3 Transfer of Care

It is critical that all medical and nursing staff who participate in the care of the postoperative patient are aware of problems identified during the pre-operative assessment and during intraoperative support. Relay of information such as intraoperative medication use may be uniquely valuable in the OSAHS patient to determine the appropriate level of postoperative monitoring.

9.0 CONCLUSIONS

OSAHS poses a significant challenge to perioperative care. Sleep-disordered breathing increases the risk for anesthetic and sedative complications, including life threatening cardiorespiratory complications. A systematic approach to identify patients with OSAHS is needed in order to provide safe perioperative care. The important concepts include a high index of suspicion, constant control of the airway, judicious use of medications, and proper monitoring of OSAHS patients. However, details regarding patient management are not fully investigated. Appropriate study is needed to refine these precautions. The recommendations presented here are based upon clinical experience and expert opinion supported by the limited peer-reviewed medical literature available. An integrated, team approach to perioperative management of the OSAHS patient is important.

ACKNOWLEDGEMENT

The Clinical Practice Review Committee acknowledges the American Sleep Apnea Association for bringing this topic to the attention of the American Academy of Sleep Medicine.

REFERENCES

1. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med* 1993; 328:1230-5.
2. Lavie P, Herer P, Hoffstein V. Obstructive sleep apnoea as a risk factor for hypertension: population study. *BMJ* 2000; 320:479-82.
3. Harbison J, O'Reilly P, McNicholas WT. Cardiac rhythm disturbances in the obstructive sleep apnea syndrome: effects of nasal continuous positive airway pressure. *Chest* 2000; 118:591-5.
4. Peker Y, Hedner J, Kraiczi H, Loth S. Respiratory disturbance index: an independent

- predictor of mortality in coronary artery disease. *Am J Respir Crit Care Med* 2000; 162:81-6.
5. Harbison JA, Gibson GJ. Snoring, sleep apnoea, and stroke: chicken or scrambled egg? *QJM* 2000; 93:647-54.
 6. Peker Y, Hedner J, Norum J, Kraiczki H, Carlson J. Increased incidence of cardiovascular disease in middle-aged men with obstructive sleep apnea: a 7-year follow-up. *Am J Respir Crit Care Med*;2002;166(2):159-65.
 7. Lamarche Y, Martin R, Reiher J, Blaise G. The sleep apnea syndrome and epidural morphine. *Can Anaesth Soc J* 1986;33(2):231-3.
 8. Wehner RJ, Pierson DJ. The Pickwickian syndrome: a special challenge for the anesthesiologist. *AANA J* 1977;45(1):57-61.
 9. Feinberg GL. Obesity: class IV anesthesia risk. *N Y State J Med* 1971;71(8):2200-1.
 10. Neuman GG, Baldwin CC, Petrini AJ, Wise L, Wollman SB. Perioperative management of a 430-kilogram (946-pound) patient with Pickwickian syndrome. *Anesth Analg* 1986;65(9):985-7.
 11. Gentil B, Lienhart A, Fleury B. Enhancement of postoperative desaturation in heavy snorers. *Anesth Analg* 1995;81:389-92.
 12. Rennotte MT, Baele P, Aubert G, Rodenstein DO. Nasal continuous positive airway pressure in the perioperative management of patients with obstructive sleep apnea submitted to surgery. *Chest* 1995;107(2):367-74.
 13. Jarrell L. Preoperative diagnosis and postoperative management of adult patients with obstructive sleep apnea syndrome: a review of the literature. *J Perianesth Nurs* 1999;14(4):193-200.
 14. Mehta Y, Manikappa S, Juneja R, Trehan N. Obstructive sleep apnea syndrome: anesthetic implications in the cardiac surgical patient. *Journal of Cardiothoracic and Vascular Anesthesia* 2000;14(4):449-53.
 15. Loadsman JA, Hillman DR. Anaesthesia and sleep apnoea. *Br J Anaesth* 2001; 86:254-66.
 16. Benumof JL. Obstructive sleep apnea in the adult obese patient: implications for airway management. *J Clin Anesth.* 2001; 13:144-56.
 17. Cullen DJ. Obstructive sleep apnea and postoperative analgesia - a potentially dangerous combination. *J Clin Anesth.* 2001; 13:83-85.
 18. Boushra NN. Anaesthetic management of obstructive sleep apnea patients. *Can J Anaesth* 1996; 43:599-616.
 19. Viner S, Szalai JP, Hoffstein V. Are history and physical examination a good screening test for sleep apnea? *Ann Intern Med* 1991; 115:356-359.
 20. Aurell J, Elmquist D. Sleep in the surgical intensive care unit: continuous polygraphic recording of nine patients receiving postoperative care. *BMJ Clin Res Ed* 1985; 290:1029-32.
 21. Rosenberg J, Rosenberg-Adamsen S, Kehlet H. Post-operative sleep disturbances: causes, factors and effects on outcome. *Eur J Anaesthesiol* 1995; 10(Suppl):28-30.
 22. Robinson RW, Zwillich CW, Bixler EO, Cadioux RJ, Kales A, White DP. Effects of oral narcotics on sleep-disordered breathing in healthy adults. *Chest* 1987; 91:197-203.
 23. Mathru M, Esch O, Lang J, Herbert ME, Chaljub G, Goodacre B, et al. Magnetic resonance imaging of the upper airway: effects of propofol anesthesia and nasal continuous positive airway pressure in humans. *Anesthesiology* 1996;84(2):253-5.
 24. Nandi PR, Charlesworth CH, Taylor SJ, Nunn JF, Doré CJ. Effect of general anaesthesia on the pharynx. *Br J Anaesth* 1991;66(2):157-62.
 25. Gupta R, Parvizi J, Hanssen A, Gay P. Postoperative complications in patients with obstructive sleep apnea undergoing hip or knee replacement: a case-control study. *Mayo Clin Proc* 2001; 76:897-905.
 26. Riley RW, Powell NB, Guilleminault C, Pelayo R, Troell RJ, Li KK. Obstructive sleep apnea surgery: risk management and complications. *Otolaryngol Head Neck Surg.* 1997; 117:648-652.
 27. Manjula V, Klowden AJ, Joseph, NJ, Raman NV, Salem MR. "Does the severity of sleep apnea hypopnea syndrome increase the risk of perioperative complications?" Scientific paper presentation at the annual meeting of the American Society of Anesthesiologists October 17, 2000 in San Francisco, CA.
 28. Esclamado RM, Glenn MG, McCulloch TM, Cummings CW. Perioperative complications and risk factors in the surgical treatment of obstructive sleep apnea syndrome. *Laryngoscope* 1989;99(11):1125-9.
 29. Levin BC, Becker GD. UPPP for snoring: long-term results. *Laryngoscope* 1994; 104:1150-2.
 30. Young T, Evans L, Finn L, Palta M. Estimation of the clinically diagnosed proportion of sleep apnea syndrome in middle aged men and women. *Sleep.* 1997; 20:705-706.
 31. Rosen R, Mahowald M, Chesson A, Doghranju K, Goldberg R, Moline M, et al. The taskforce 2000 survey on medical education in sleep and sleep disorders. *Sleep* 1998; 21:235-238.
 32. Eastwood PR, Szollosi I, Platt PR, Hillman DR. Comparison of upper airway collapse during general anesthesia and sleep. *Lancet* 2002; 359:1207-9.
 33. Schäfer H, Ewig S, Hasper E, Lüderitz B. Predictive diagnostic value of clinical assessment and nonlaboratory monitoring system recordings in patients with symptoms suggestive of obstructive sleep apnea syndrome. *Respiration* 1997; 64:194-199.
 34. Tami TA, Duncan HJ, Pflieger M. Identification of obstructive sleep apnea in patients who snore. *Laryngoscope* 1998; 108:508-513.
 35. Dealberto MJ, Ferber C, Garma L, Lemoine P, Alperovitch A. Factors related to sleep apnea syndrome in sleep clinic patients. *Chest* 1994; 105:1753-1758.
 36. Suganuma N, Shigedo Y, Adachi H, Watanabe T, Kumano-Go T, Terashima K, et al. Association of gastroesophageal reflux disease with weight gain and apnea, and their disturbance on sleep. *Psychiatry Clin Neurosci* 2001; 55:255-6.
 37. Logan AG, Perlikowski SM, Mente A, Tisler A, Niroumand M, Leung RS, et al. High prevalence of unrecognized sleep apnea in drug-resistant hypertension. *J Hypertens* 2001;19:2271-7.
 38. Hoffstein V, Szalai JP. Predictive value of clinical features in diagnosing obstructive sleep apnea. *Sleep* 1993; 16:118-22.
 39. Hoffstein V, Mateika S. Differences in abdominal and neck circumferences in patients with and without obstructive sleep apnoea. *Eur Resp J.* 1992; 5:377-81.
 40. Katz I, Stradling J, Slutsky AS, Zamel N, Hoffstein V. Do patients with sleep apnea have thick necks? *Am Rev Respir Dis* 1990; 141(5 Pt 1):1228-31.
 41. Kushida CA, Efron B, Guilleminault C. A predictive morphometric model for the obstructive sleep apnea syndrome. *Ann Int Med* 1997;127:181-7.
 42. Bixler EO, Vgontzas AN, Lin HM, Ten Have T, Rein J, Vela-Bueno A, et al. Prevalence of sleep-disordered breathing in women: effects of gender. *Am J Respir Crit Care Med* 2001;163(3):597-8.
 43. Schellenberg JB, Maislin G, Schwab RJ. Physical findings and the risk for obstructive sleep apnea. The importance of oropharyngeal structures. *Am J Respir Crit Care Med* 2000; 162(2 pt 1):740-8.
 44. Rombaux P, Bertrand B, Boudevyns A, Deron P, Goffart Y, Hassid S, et al. Standard ENT clinical evaluation of the sleep-disordered breathing patient; a consensus report. *Anesth Analg* 2002;94(3):732-6.
 45. Brodsky JB, Lemmens HJ, Brock-Utne JG, Vierra M, Saidman LJ. Morbid obesity and tracheal intubation. *Anesth Analg* 2002;95(3):783.
 46. Biddle C. Comparative aspects of the airway during general anesthesia in obese sufferers of sleep apnea and matched controls. *Adv Prac Nurs Q* 1996;2(3):14-9.
 47. Hiremath AS, Hillman DR, James AL, Noffsinger WJ, Platt PR, Singer SL. Relationship between difficult tracheal intubation and obstructive sleep apnea. *Br J Anaesth* 1998; 80:606-11.
 48. Benumof, JL. "The ASA difficult airway algorithm: new thoughts and consideration", Refresher course presented at the annual meeting of the American Society of Anesthesiologists October 9, 1999 in Dallas, TX.
 49. Berry RB, Parish JM, Hartse KM. The use of auto-titrating continuous positive airway pressure for treatment of adult obstructive sleep apnea. *An American Academy of Sleep Medicine Review.* *Sleep* 2002; 25:148-73.
 50. Powell NB, Riley RW, Guilleminault C, Nino Murcia G. Obstructive sleep apnea, continuous positive airway pressure, and surgery. *Otolaryngol Head Neck Surg* 1988;99(4):362-9.
 51. McConkey P. Postobstructive pulmonary oedema - a case series and review. *Anaesth Intensive Care* 2000;28:72-76.
 52. Ryan F, Lowe AA, David LI, Fleetham JA. Magnetic resonance imaging of the upper airway in obstructive sleep apnea before and after chronic nasal continuous positive airway pressure therapy. *Am Rev Respir Dis* 1991;144:939-44.
 53. Dodds C, Ryal DM. Tonsils, obesity and obstructive sleep apnea. *Br J Hosp Med* 1992;47(1):62-6.
 54. Benumof, JL. Management of the difficult airway. *Anesthesiology* 1991, 75:1087-1110.
 55. Biro P, Kaplan V, Bloch KE. Anesthetic management of a patient with obstructive sleep apnea syndrome and difficult airway access. *J Clin Anesth* 1995;7(5):417-21.
 56. Benumof, JL. Laryngeal mask airway and the ASA difficult airway algorithm. *Anesthesiology* 1996; 84:686-699.
 57. Benumof, JL, Dagg R, Benumof R. Critical hemoglobin desaturation will occur before return to an unparalyzed state following 1 mg/kg intravenous succinylcholine. *Anesthesiology* 1997; 87:979-982.
 58. Pellicchia DJ, Bretz KA, Barnette RE. Postoperative pain control by means of epidural narcotics in a patient with obstructive sleep apnea. *Anesth Analg* 1987;66:280-2.
 59. Ostermeier AM, Roizen MF, Hautkappe M, Klock PA, Klafra JM. Three sudden postoperative respiratory arrests associated with epidural opioids in patients with sleep apnea. *Anesth Analg* 1997; 85:452-60.
 60. Knill Clement JL, Thompson WR. Epidural morphine causes delayed and prolonged ventilatory depression. *Can Anaesth Soc J* 1981; 28:537-43.
 61. Berry RB, Kouchi KG, Bower, JL, Light RW. Effect of upper airway anesthesia on obstructive sleep apnea. *Am J Respir Crit Care Med* 1995; 151:1857-1861.
 62. Rosenberg J, Rasmussen GI, Wøjedemann KR, Kerkeby LT, Jørgensen LN, Kehlet H. Ventilatory pattern and associated episodic hypoxaemia in the late postoperative period in the general surgical ward. *Anaesthesia* 1999; 54:323-328.
 63. Knill RL, Moote CA, Skinner MI, Rose EA. Anesthesia with abdominal surgery leads to intense REM sleep during the first postoperative week. *Anesthesiology* 1990; 73:52-61.
 64. Flemons, WW, Whitelaw WA, Brant R, Remmers JE. Likelihood ratios for a sleep apnea clinical prediction rule. *Am J Respir Crit Care Med* 1994; 150:1279-1285.
 65. Gyulay S, Olson LG, Hensley MJ, King MT, Allen KM, Saunders NA. A comparison of clinical assessment and home oximetry in the diagnosis of obstructive sleep apnea. *Am Rev Resp Dis* 1993; 147:50-53.

APPENDIX A

Questionnaire for exploring OSAHS symptoms

People tell me that I snore.	Y	N
I wake up at night with a feeling of shortness of breath or choking.	Y	N
People tell me that I gasp, choke or snort while I am sleeping.	Y	N
People tell me that I stop breathing while I am sleeping.	Y	N

I awake feeling almost as or more tired than when I went to bed.	Y	N
I often awake with a headache.	Y	N
I often have difficulty breathing through my nose.	Y	N
I fight sleepiness during the day.	Y	N
I fall asleep when I relax before or after dinner.	Y	N
Friends, colleagues or family comment on my sleepiness.	Y	N

These questions regarding snoring and breathing arrests during sleep suggestive of OSAHS were derived from several trials.^{32,33,34,64,65}

APPENDIX B

High Risk

Male
 BMI>25 kg/m²
 Neck circumference
 >17 inches in men
 >16 inches in women
 Habitual snoring/ gasping noted by bed partner
 Daytime sleepiness
 Hypertension

Low Risk

No snoring
 Premenopausal
 Thin