

Obstructive Sleep Apnea Syndrome (OSAS) in Children

BY KAREN A. BROWN, MD

Obstructive sleep apnea syndrome (OSAS) is a sleep-disordered pattern of breathing characterized by periodic, partial, or complete obstruction of the upper airway during sleep. In children, the airway obstruction occurs primarily during active rapid eye movement (REM) sleep. Although OSAS occurs at all ages – from infancy to adulthood^{1,2} – the focus of this issue of *Anesthesiology Rounds* is on pediatric OSAS.

There are several hypotheses to explain the propensity for pharyngeal collapse during sleep in OSAS.

The anatomic hypothesis considers the upper airway to be “a collapsible tube in a bony box.” The size of the confining skeletal structure can be increased with airway maneuvers that extend the neck and advance the mandible.^{3,4} The volume of soft tissue, particularly the volume of the tongue and lateral pharyngeal walls that lie within the pharynx, correlates with a diagnosis of OSAS.⁵ Encroachment on the pharyngeal lumen by excess adipose tissue or lymphoid tissue underscores the association between obesity and adenotonsillar hypertrophy, respectively, and OSAS.⁶⁻⁹ The influence of soft tissue on pharyngeal caliber is evident by the fact that the apnea hypopnea index (AHI) decreases in conditions of microgravity.¹⁰

In addition to limitations in pharyngeal caliber, the upper airway in patients with OSAS is inherently more collapsible.¹¹⁻¹⁴ Sensory blockade of the upper airway with local anesthesia unmasks this collapsibility and is associated with a marked decrease in the cross-sectional area of the pharynx.¹³ The application of continuous positive airway pressure (CPAP) is invaluable both to increase pharyngeal caliber and decrease collapsibility of the upper airway.¹¹ Some of the beneficial effects of CPAP may, in part, reflect changes in lung volume and its influence on upper airway collapsibility.¹⁴

In normal subjects, subatmospheric pressure must be applied to the upper airway to collapse it; this pressure is referred to as the “closing pressure.”^{11,15} In patients with OSAS, the pharyngeal airway collapses at supra-atmospheric pressure during sleep and the closing pressure of the pharyngeal airway linearly correlates with severity of OSAS. The negative airway pressure reflex occurs when negative pressure is applied to the pharynx and results in reflex stimulation of the upper airway dilator muscles.^{16,17} Reflex activation of upper airway dilators by negative pressure is a major mechanism safeguarding pharyngeal caliber.¹⁵

The vagal tone hypothesis is a second hypothesis to explain upper airway collapsibility and decreased pharyngeal caliber during sleep. Central muscarinic blockade at the hypoglossal nucleus in an experimental rat model was recently reported to augment genioglossus function,¹⁸ suggesting that cholinergic mechanisms may underlie OSAS. In patients with sick sinus syndrome, increasing the basal heart rate is reported to decrease the AHI during sleep.¹⁹

Surface forces: The upper airway collapses during both obstructive and central apnea. Therefore, surface forces may play a role in upper airway patency. Administering agents that reduce surface tension (eg, a surfactant) into the nasopharynx is reported to decrease the AHI during sleep.²⁰

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The inflammatory hypothesis: Nasal exudates in children undergoing tonsillectomy have been assayed for inflammatory mediators. Surprisingly, children with OSAS have a higher leukotriene content than those with chronic tonsillitis. This has led to an inflammatory hypothesis for OSAS, which suggests that the recurrent vibratory stimulus associated with snoring results in upregulation of leukotrienes in pharyngeal lymphoid tissue.²¹

BACKGROUND

Skeletal hypotonia and atonia during sleep, particularly REM sleep, promote the collapse of the upper airway, resulting in a pattern of cyclic obstructive sleep apnea. In children, an apnea index of >1 event per hour and desaturation of <92% during sleep are statistically aberrant and support the diagnosis of OSAS.^{22,23} Overt apnea may not be present, but sustained periods of persistent obstructive hypoventilation may be and are considered an “OSAS equivalent” in children. This pattern is often referred to as the upper airway resistance syndrome.^{24,25} The prevalence of OSAS according to sleep laboratory diagnostic criteria is 8-fold higher than the prevalence of overt disease, at least in the adult population.¹

Episodes of obstructive apnea (OA) are terminated with electroencephalographic (EEG) arousal, which causes sleep fragmentation and decreases active REM sleep to < 20% of total sleep time (at least in adults). However, sleep “architecture” in children with OSAS has a normal sleep-stage distribution.²⁶ Daytime sleepiness is less common in OSAS children than in OSAS adults.²⁴ Obstructive apnea may be accompanied by desaturation and the saturation nadir is inversely correlated with the AHI.^{27,28} Consequently, OSAS results in a trilogy of:

- sleep fragmentation
- nocturnal intermittent hypoxia
- episodic hypercapnia.

THE EFFECTS OF OSAS

The trilogy described above produces a spectrum of diseases that affect multiple organ systems, including the cardiovascular, neurocognitive, and endocrine systems. OSAS has a negative impact on quality of life, somatic growth, cardiovascular health, neurocognitive function, and behaviour. Quality of life is diminished to levels seen in pediatric cancer, asthma, and juvenile rheumatoid arthritis.²⁹ Because OSAS disrupts the circadian release of growth hormone and because growth hormone secretion is dependant on sleep, OSAS may have a negative impact on somatic growth.³⁰ Both “failure to thrive” and obesity may be present. In fact, failure to thrive is a common presenting complaint.²⁵ In addition, sleep deprivation alters ghrelin and leptin secretions, driving an appetite for high-caloric foods, therefore,

promoting obesity.³¹ At the present time, 14% of children are considered obese – representing a 2-fold increase in childhood obesity during the last 25 years – and the incidence reaches 21% in minority group Americans.³² One-third of obese children have OSAS^{7,9} and severity correlates with degree of obesity since nadir saturation and the AHI both correlate with weight/height ratios.³³

Pediatric OSAS is associated with neurocognitive dysfunction. Grade school children in the lowest 10% for school performance have a higher risk for sleep-disordered breathing. Adenotonsillectomy (T&A) improves school performance. Intermittent hypoxia is associated with long-term sequelae on hippocampal functions such as learning and memory. The arousal index has a significant effect on prefrontal cortical functions influencing behaviour and attention.³⁴⁻³⁶

Cardiovascular disease is associated with OSAS. Sleep debt increases sympathetic tone and cortisol levels, promoting cardiovascular disease.³⁰ OSAS in children is associated with both systemic³⁷ and pulmonary hypertension.^{38,39} In addition to right ventricular dysfunction, decreased left ventricular (LV) function and eccentric LV hypertrophy have recently been reported in severe pediatric OSAS.^{40,41} Longitudinal studies suggest that the neuroendocrine disturbances associated with childhood OSAS are antecedents for adult cardiovascular disease, diabetes, and obesity.

INCIDENCE OF OSAS AND RISK FACTORS

The incidence of OSAS in children is 1% to 3%^{23,24} but, in the preadolescent population, the incidence may be as high as 10% to 12%. There are no gender differences in the incidence of pediatric OSAS; however, African Americans have a 3-fold higher incidence of OSAS.⁴²⁻⁴⁴ Former premature infants comprise a subgroup of high-risk OSAS patients.³⁹ In children, adenotonsillar hypertrophy is the leading cause of OSAS and for the majority, T&A is curative.^{45,46}

Severe OSAS is defined by an AHI >10 events/hour and/or a saturation nadir < 80%, and is associated with important cardiovascular sequelae. These sequelae include pulmonary and systemic hypertension, both right and LV dysfunction,^{40,41} recurrent pulmonary aspiration,⁴⁷ and abnormalities in ventilatory control,⁴⁸ such as an increased sensitivity to opioid respiratory depression.⁴⁹

There is a growing body of literature reporting that there is an increased perioperative risk associated with a diagnosis of OSAS in both adults⁵⁰ and children.⁵¹⁻⁵³ The risk of post-T&A respiratory morbidity in the general pediatric population is 1%.⁵⁴⁻⁵⁶ A diagnosis of OSAS increases this risk by at least 20-fold.^{28,51-53,57-59} Studies in postoperative risk report that an AHI >10 events/hour and a saturation nadir

<80% are values that predict increased risk for postoperative respiratory morbidity.^{28,50-52} Children with severe OSAS who experience profound nocturnal desaturation during sleep are at particular risk.^{51,53,59}

Perioperative risk is the product of independent risk factors. An associated medical condition is an independent risk factor for post-T&A respiratory complications.^{28,51,52} Medical conditions associated with OSAS include prematurity,³⁹ craniofacial anomalies,^{60,61} asthma,⁶ and obesity.^{6,7,9} Forty percent of morbidly obese children electively admitted to a pediatric intensive care unit (PICU) experienced respiratory complications post-T&A that required major respiratory support, including CPAP, intubation, and ventilation.⁶² A young child with severe OSAS and an associated medical condition is at high risk for post-T&A respiratory complications.^{52,59}

DIAGNOSIS OF OSAS

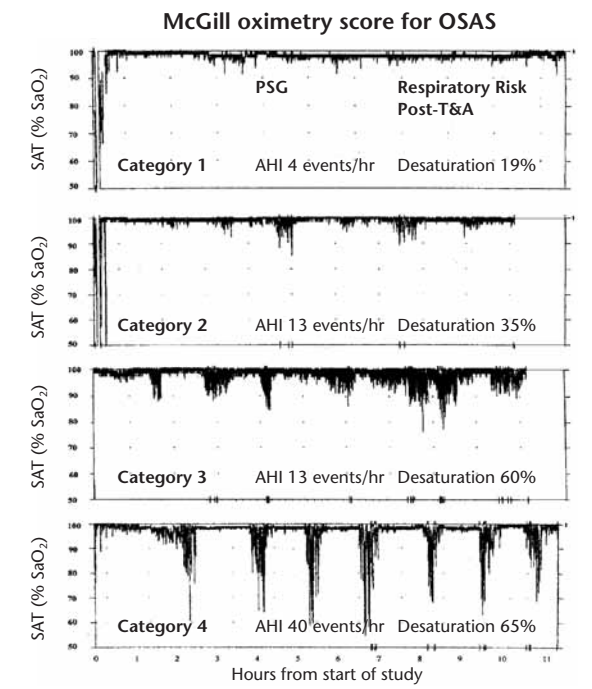
Establishing a diagnosis of OSAS on clinical grounds alone is problematic and only a minority of children currently undergo testing for OSAS prior to T&A.⁶³ A diagnosis based on clinical scores alone correlates poorly with polysomnography (PSG), which is considered the gold standard.^{25,64-67} At a minimum, the anesthetic preoperative examination should include the following questions:

- Does your child snore or have noisy breathing at night and, if yes, is it loud?
- Does your child stop breathing while sleeping?
- Have you ever shaken your child to restart his breathing?
- Are you fearful when watching your child sleep?
- Does your child use mouth breathing during the day?

A positive response to these questions correlates with positive findings on PSG. The OSAS-18 clinical score, which was developed in 60 non-obese children, is a recent attempt to diagnose OSAS by history.²⁹ It assesses the frequency of symptoms known to be associated with OSAS, including sleep disturbance, physical symptoms, emotional distress, daytime dysfunction, and parental concern. When applied to a nonobese, pediatric population, there was a high correlation between the total score and the Respiratory Distress Index, analogous to the AHI, obtained during a 90-minute nap-PSG, and the size of the adenoids and tonsils.

Our research group^{53,68} and others⁵⁰ have focused on the diagnostic potential of oximetry to stratify OSAS severity because oximetry is potentially widely available and preoperative oxygen desaturation during sleep correlates with the AHI and provides a good predictor of perioperative risk.^{28,53} We have developed the McGill oximetry score (Figure 1). At least 3 clusters of desaturation that are <90% during sleep have a positive predictive value for a diagnosis of OSAS in otherwise healthy children.

FIGURE 1: The McGill oximetry categories are distinguished by the depth of the oxygen desaturation during sleep



Category 4, 3 and 2 demonstrate desaturation <80%, <85% and <90%, respectively. Category 1 does not exclude a diagnosis of OSAS. The correlates of category 4,3,2 and 1 with the Apnea Hypopnea Index (AHI) are 40, 13, 13 and 4, respectively. The prevalence of post-T&A desaturation for categories 4,3,2 and 1 were 65%, 60%, 35% and 19%, respectively. Major airway complications only occurred with category 4.

IMPLICATIONS FOR ANESTHESIA

There is a differential sensitivity of respiratory muscles to anesthetic agents so that diaphragmatic function is preserved relative to that of the upper airway dilators.^{69,70} As a result, anesthesia mimics the skeletal hypotonia present during sleep. Whether by blockade of sodium channels with local anesthesia,^{13,71} stimulation of γ -aminobutyric acid (GABA) receptors by sedative/general anesthetic agents,^{70,72-74} or blockade of nicotinic cholinergic neuromuscular⁶⁹ and central¹⁸ receptors, the end result is inhibition of the upper airway dilator muscles that promotes upper airway obstruction.

There may be an "ideal" OSAS anesthetic to minimize the risk of postoperative morbidity. Given the critical role of the upper airway dilators in OSAS, general anesthesia with a secured airway may be safer than deep sedation and a natural airway. The risk of postoperative desaturation may be decreased if surgery is performed in the morning.⁷⁵

CPAP is extremely useful for managing the airway in a child with OSAS.² However, since the closing pressure correlates with the severity of OSAS, this implies that the nasopharynx of children with severe OSAS may not open even with high levels of CPAP and intravenous induction may be preferable. Administration of intraoperative atropine⁵⁹ decreases risk and may involve cholinergic mechanisms that,

increasingly, are recognized to play a role both in sleep regulation⁷⁶ and the function of the genioglossus musculature.¹⁸ In children, an augmentation in muscle activity of the upper airway dilators, principally the genioglossus muscle, is usually sufficient to terminate an OA without full EEG arousal.⁴⁶ Impaired function of the upper airway dilators may contribute to respiratory morbidity following T&A.⁷⁷

Children with OSAS demonstrate a blunted response to hypercarbia and greater respiratory depression with opioids.^{48,49} Following T&A, morphine consumption is decreased in children who demonstrate recurrent episodic desaturation during sleep;⁷⁸ therefore, the child with severe OSAS may require less opioid for analgesia. Model analysis of the analgesic and respiratory properties of morphine in healthy adult volunteers suggests a common μ -opioid receptor subtype. Although respiratory depression occurs at subanalgesic morphine concentrations, the gain in analgesic effect was greater at higher concentrations.⁷⁹

Increased opioid sensitivity in children with severe OSAS is not a small issue, given the widespread use of codeine as a home analgesic,⁸⁰ the introduction of scheduled codeine regimes following T&A,⁸¹ evidence of prolonged respiratory depression following morphine administration,⁷⁹ and the lack of preoperative screening for nocturnal desaturation in the day-surgery population.⁶³ Morphine-sparing adjuncts, including dexamethasone⁸² and acetaminophen,⁸³ may be useful. The anti-inflammatory properties of dexamethasone may confer additional benefits, given the inflammatory hypothesis of OSAS.²¹ Sensory nerve blockade, including glossopharyngeal nerve block, should be used with caution in severe OSAS.^{13,71}

Two prospective studies report that the majority of desaturations that complicate T&A in the postoperative period are associated with obstructive apneas.^{57,77} Nasal secretions may be copious⁷⁷ and the resultant nasal obstruction may limit the utility of CPAP following T&A. Respiratory distress may present at a time remote from surgery (eg, after midnight) and obstructive apneas are common.^{57,75,77} Supportive measures in the postoperative period may require the insertion of nasal airways, CPAP support, reintubation, ventilation, and administration of furosemide, salbutamol, racemic epinephrine, heliox, and dexamethasone.^{28,51,52,59,62} In many hospitals, this level of pediatric respiratory support is only provided in an intensive care setting. Therefore, children with severe OSAS are best cared for in a hospital setting capable of this level of monitoring and treatment.

TABLE 1: Criteria for the diagnosis of pediatric OSAS and prediction of post-T&A respiratory morbidity. There are higher AHI and lower saturation nadir thresholds to predict post-T&A risk.

	Diagnostic criteria ^{22,23}	Predictors of post-T&A respiratory risk ^{51,52,59}
AHI (#/hr)	>1 (mild) >5 (severe)	>10
Saturation nadir (%)	< 92	< 80

AHI = apnea hypopnea index

CONCLUSIONS

The implications of OSAS in the management of children undergoing T&A is sobering, since it affects 3% of children, a prevalence similar to asthma and diabetes. A diagnosis of OSAS identifies a patient population at increased risk of perioperative complications at a time when the majority of pediatric T&As are performed during day surgery programs⁸⁰ and the most common indication for T&A is obstructive breathing.⁶³

Although a diagnosis of OSAS *per se* is an exclusion for day surgery, it is, in fact, the stratification of OSAS severity that is important in predicting perioperative risk. Indeed, the criteria for prediction of perioperative risk and diagnosis of OSAS differ (Table 1). It is the subset of children with moderate-to-severe OSAS that poses the greatest challenge to anesthesiologists. Given the extensive body of literature reporting significant pathophysiology and perioperative morbidity in children with OSAS, it is imperative that anesthesiologists review the management of these children.

The *Practice Guidelines on Perioperative Management of Patients with Obstructive Sleep Apnea*, which was recently presented by members of the American Society of Anesthesiologists Task Force at the May Society for Ambulatory Anesthesia (SAMBA) meeting, proposes a scoring system to stratify perioperative risk. The scoring system considers patient factors, surgical procedure, and analgesic practice, namely the use of opioids, in evaluating operative risk. The Practice Guidelines proposed by this Task Force, when approved by the American Society of Anesthesiologists this fall, will enable the diagnosis of OSAS on clinical grounds alone. It is acknowledged that clinical scores will overestimate the true prevalence of OSAS. Greater specificity can only be achieved with testing, including oximetry,⁶⁸ cardiorespiratory studies^{84,85} and, of course, formal PSG.

Finally, as anesthesiologists, we are uniquely positioned to assess the collapsibility of the air-

way during anesthesia and sedation. Excessive respiratory depression during anesthesia and opioid administration may indicate an altered CO₂ responsiveness that is suggestive of OSAS. The importance of the diagnostic role of anesthesiologists in evaluating the behavior of the airway during anesthesia has been recently discussed.⁸⁶

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