CME Perioperative Management of Children with Obstructive Sleep Apnea

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Obstructive sleep apnea syndrome (OSA) affects 1%–3% of children. Children with OSA can present for all types of surgical and diagnostic procedures requiring anesthesia, with adenotonsillectomy being the most common surgical treatment for OSA in the pediatric age group. Thus, it is imperative that the anesthesiologist be familiar with the potential anesthetic complications and immediate postoperative problems associated with OSA. The significant implications that the presence of OSA imposes on perioperative care have been recognized by national medical professional societies. The American Academy of Pediatrics published a clinical practice guideline for pediatric OSA in 2002, and cited an increased risk of anesthetic complications, though specific anesthetic issues were not addressed. In 2006, the American Society of Anesthesiologists published a practice guideline for perioperative management of patients with OSA that noted the pediatric-related risk factor of obesity, and the increased perioperative risk associated with adenotonsillectomy in children younger than 3 yr. However, management of OSA in children younger than 1 yr-of-age was excluded from the guideline, as were other issues related specifically to the pediatric patient. Hence, many questions remain regarding the perioperative care of the child with OSA.

In this review, we examine the literature on pediatric OSA, discuss its pathophysiology, current treatment options, and recognized approaches to perioperative management of these young and potentially high-risk patients.

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Management of the child known to have or is suspected of having obstructive sleep apnea syndrome (OSA) is a challenge for the anesthesiologist. Children with OSA have recurrent episodes of partial or complete airway obstruction during sleep, resulting in hypoxemia, hypercapnia, and sleep disruption, and approximately 1%–3% of all children are thought to have OSA.[1–5] Children who carry the diagnosis of OSA usually have been evaluated by an otolaryngologist, pulmonologist, or sleep specialist, often in preparation for adenotonsillectomy. However, children scheduled for other types of surgical procedures may not be as thoroughly evaluated; OSA in these patients may be missed by surgeons or primary care providers.[6–8]

Anesthesiologists are aware of the concerns in caring for children diagnosed with OSA, and 82% of surveyed anesthesiologists reported that guidelines would assist them in caring for them.[9] The American Society of Anesthesiology (ASA) Task Force on Perioperative Management of Patients with OSA suggests a scoring system to estimate perioperative risk based on severity of sleep apnea, invasiveness of procedure, and postoperative opioid requirement. This task force also supports postoperative admission for children <3 yr-of-age who undergo adenotonsillectomy. However, medical evidence is lacking as to whether a monitored bed is required for some patients with OSA. Because specific guidelines are lacking regarding such patients, local institutional policies often lead to controversies among anesthesiologists, surgeons, and third-party payers regarding the need for, and level of, postoperative monitoring.[10]

This review will summarize what is currently known about childhood OSA, including diagnosis, treatment, and strategies for perioperative care of these patients.

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DIAGNOSIS

OSA presents differently in young children than it does in teenagers and adults (Table 1). Adults and teenagers with OSA are often obese and have daytime somnolence; younger children may have normal weight or failure to thrive and behavior disorders such as hyperactivity, attention problems, and enuresis.[11,12] Both adult and pediatric patients have an increased risk for complications during or after surgery.[10]

Table 1. Childhood Versus Adult Obstructive Sleep Apnea Syndrome Features

	Children	Adults
Presentation		
Age	2–6-yr peak	Increased elderly
Gender	Male = female	Males > females
Obesity	Few	Most
Tonsils and adenoids	Often enlarged	Rarely enlarged
Daytime sleepiness	Less common than in adults but can be seen	Common
Sleep		
Obstruction	Obstructive apnea or hypoventilation	Obstructive apnea
Sleep architecture	Usually normal	Decreased delta and REM
Arousals with obstruction	May not be seen	At end of each apnea
Treatment		
Surgical	Definitive therapy in most patients	Minority of cases with inconsistent results
Medical (positive airway pressure)	Selected patients	Most common therapy

Adapted from Sterni and Tunkel, Pediatr Clin North Am 2003;50:427–43.
REM = rapid eye movement.

A history of snoring is a sensitive, though not specific, symptom that supports the diagnosis of OSA.[13] Primary snorers are those who snore but have no obstructive apnea, gas exchange abnormalities, or multiple arousals on polysomnography (sleep study). Approximately 10% of children have primary snoring.[14–17] Primary snoring does not seem to progress to OSA and may resolve over time,[13,18,19] and although generally considered benign, it may be associated with neurobehavioral changes, such as attention disorders, mild cognitive problems, and anxious or depressive symptoms.[20]

OSA in children may manifest with obstructive apneas or obstructive hypoventilation (OH).[21] Children with OH exhibit snoring with continuous partial upper airway obstruction during sleep, leading to paradoxical respiratory effort, hypercarbia, and hypoxemia in some.[22] As many as 40% of snoring children who are referred to a sleep clinic or otolaryngologist will have OSA,[1–5] and children with OSA almost always snore and have increased respiratory effort during sleep.[3,23,24]

Screening children for OSA is recommended by the American Academy of Pediatrics as part of routine health maintenance. Evaluation should be pursued for children with a history of nightly snoring. Although several pediatric studies were unable to show that questionnaires differentiated those with OSA from those with primary snoring,[3,5,25–29] two independent studies of Chinese children reported validated questionnaires for OSA.[30,31] Direct observation of a child having apnea and labored respirations during sleep may support the diagnosis of OSA. However, parents may not be able to distinguish primary snoring from obstructive snoring; as well, obstructive episodes that occur during rapid eye movement (REM) sleep may go unwitnessed.[32] Videotapes, audiotapes with pulse oximetry, questionnaires, and daytime nap studies may be used but do not exclude OSA when negative.[3,5,31,33–40] Unattended home polysomnography might prove to be a useful testing modality,[41,42] but validation of these studies is needed.

Table 2. Components of Polysomnography Recommended by The American Thoracic Society

Respiratory effort—assessed by abdominal and chest wall movement
Airflow at nose, mouth, or both
Arterial oxygen saturation
End-tidal CO_2 or transcutaneous CO_2 (recommended specifically for pediatric polysomnography to detect hypoventilation)
Electrocardiograph
Electromyography (tibial) to monitor arousals
Electroencephalography, electrooculography, and electromyography for sleep staging

From Standards and indications for cardiopulmonary sleep studies in children. American Thoracic Society. Am J Respir Crit Care Med 1996;153:866–78, with permission.

The "gold standard" for making the diagnosis of OSA in children is use of overnight polysomnography. However, there are significant differences in the criteria for the performance, scoring, and interpretation of pediatric versus adult polysomnograms; for complete efficacy, it is essential that polysomnography laboratories have experience in the performance and interpretation of these studies in children.[43] Use of sedatives and sleep deprivation is not recommended.[44,45] If sufficient sleep and REM time are captured, a single overnight study is usually adequate.

Obstructive events in children with OSA occur primarily during REM sleep,[46] although adult patients exhibit non-REM preponderance or equal REM and non-REM obstruction.[47] Obstruction is thought to worsen over the course of a night, however, evaluation of upper airway muscles in children has not shown clinically significant muscle fatigue.[48] The effect of the perioperative period on this process has not been adequately studied. Postoperative REM rebound may worsen OSA in some patients.[47,49]

The components of polysomnography recommended by the American Thoracic Society are listed in Table 2. Respiratory events that may be seen during

Table 3. Respiratory Events that can be Seen During Polysomnography

Event	Definition
Central apnea	Pause in airflow with absent respiratory effort, scored when >20 s or two missed breaths and a >3% drop in oxygen saturation
Obstructive apnea	>90% reduction of airflow despite continuing respiratory effort, scored when event lasts at least two missed breaths in children
Obstructive hypopnea	>50% reduction of airflow with associated with respiratory effort, scored when at least two missed breaths and >3% drop in oxygen saturation or arousal
Mixed apneas	≥90% reduction in airflow, lasting at least two missed breaths, and containing absent respiratory effort initially (a central apneic pause), followed by resumption of respiratory effort without a resumption of airflow (an obstructive apnea)
Obstructive hypoventilation	End-tidal CO_2 >50 mm Hg for >25% of the total sleep time with paradoxical respirations, snoring, and no baseline lung disease

polysomnography are listed and defined in Table 3 and depicted in Figure 1.

Often, obstructive apneas and hypopneas identified during a sleep study are combined to provide the apnea–hypopnea index (AHI), defined as the number of discrete obstructive events per hour. Many sleep laboratories will report an AHI or a respiratory disturbance index (RDI) that will include the number of all scored respiratory events (including central apneas) per hour. Indices that combine central and obstructive events cannot be used to diagnose OSA in children, as normal children have more frequent central apneas when compared with adults. Only obstructive event indices should be used to identify pediatric OSA.[50,51]

Data correlating polysomnography parameters with clinical outcomes in children are lacking, and there are no standard guidelines for classifying the severity of OSA in children. Polysomnography data from nonsnoring children have defined OSA as more than one obstructive event per hour.[37,52,53] It is important to note that the scoring does not consider the length of time of the obstructive event. The AHI may be misleadingly low in children who have OH rather than discrete apnea. OH is not scored as an event but diagnosed as shown in Table 3. Thus, children with OH may have significant disease with a low AHI if the periods of OH are few but long. For this reason, other factors must be considered. In our Pediatric Sleep Laboratory, we classify the severity of OSA based on total clinical picture, number of obstructive events per hour, duration of elevated end-tidal CO_2, and frequency and severity of oxygen desaturation. We classify OSA as severe if the patient has an AHI of ≥10/h because of the increased risk of respiratory compromise after adenotonsillectomy.[54] Oxygen saturation nadir <80% is also suggestive of severe disease and postadenotonsillectomy respiratory morbidity[55–58] Suggested guidelines for assessing severity of OSA based on polysomnography are listed in Table 4.

PATHOPHYSIOLOGY

The essential feature of OSA in children is increased upper airway resistance during sleep.[59] Adenotonsillar hypertrophy, allergic rhinitis, turbinate hypertrophy, deviated septum, and maxillary constriction cause airway narrowing in children.[60] Enlarged tonsils can lead to collapse of the hypopharynx at the level of the soft palate because of posterior displacement of the tongue and descent of the tonsils.[61] Although enlarged tonsils and adenoids are clearly an important risk factor in children,[62] there is no absolute correlation between the size of the tonsils and adenoids and the presence of OSA.[63–66] Enlarged soft tissues from obesity or lymphoid tissues contribute to OSA in many children. Other factors include abnormal central arousal threshold, abnormal bony anatomy, disordered neural control of airway caliber or sensation, and decreased pharyngeal tone that may be seen in certain types of cerebral palsy.[67] Dynamic airway narrowing or collapse occurs at multiple sites in children with OSA.[59] Thus, OSA is often a multifactorial disorder with overlapping influences that together predispose the patient to obstructed breathing (Fig. 2).[22]

Genetics affect the risk of developing OSA. The incidence of OSA is higher in first-degree relatives of index patients with OSA.[68] Infants of families with multiple members affected by sudden infant death syndrome, apparent life-threatening event, and OSA are more likely to have OSA than those in families with only one case of sudden infant death syndrome or apparent life-threatening event; these infants were found to have OSA in their first year of life.[69]

Craniofacial abnormalities with altered airway anatomy are associated with abnormal breathing function.[70] The combination of enlarged tonsils and craniofacial abnormalities (Table 5) can predict development of OSA.[71,72]

Obesity is an important and increasingly common risk factor for OSA in children. Deposition of adipose tissue around the upper airway and external compression from the excess soft tissue around the neck and jaw lead to upper airway narrowing. Decreased chest wall compliance and upward displacement of the diaphragm by the obese abdomen when the individual is supine lead to smaller lung volumes during sleep, decreased oxygen stores, and increased risk of desaturation with obstructive events.[73] Obese children continue to have an increased risk of continuing OSA after adenotonsillectomy.[74]

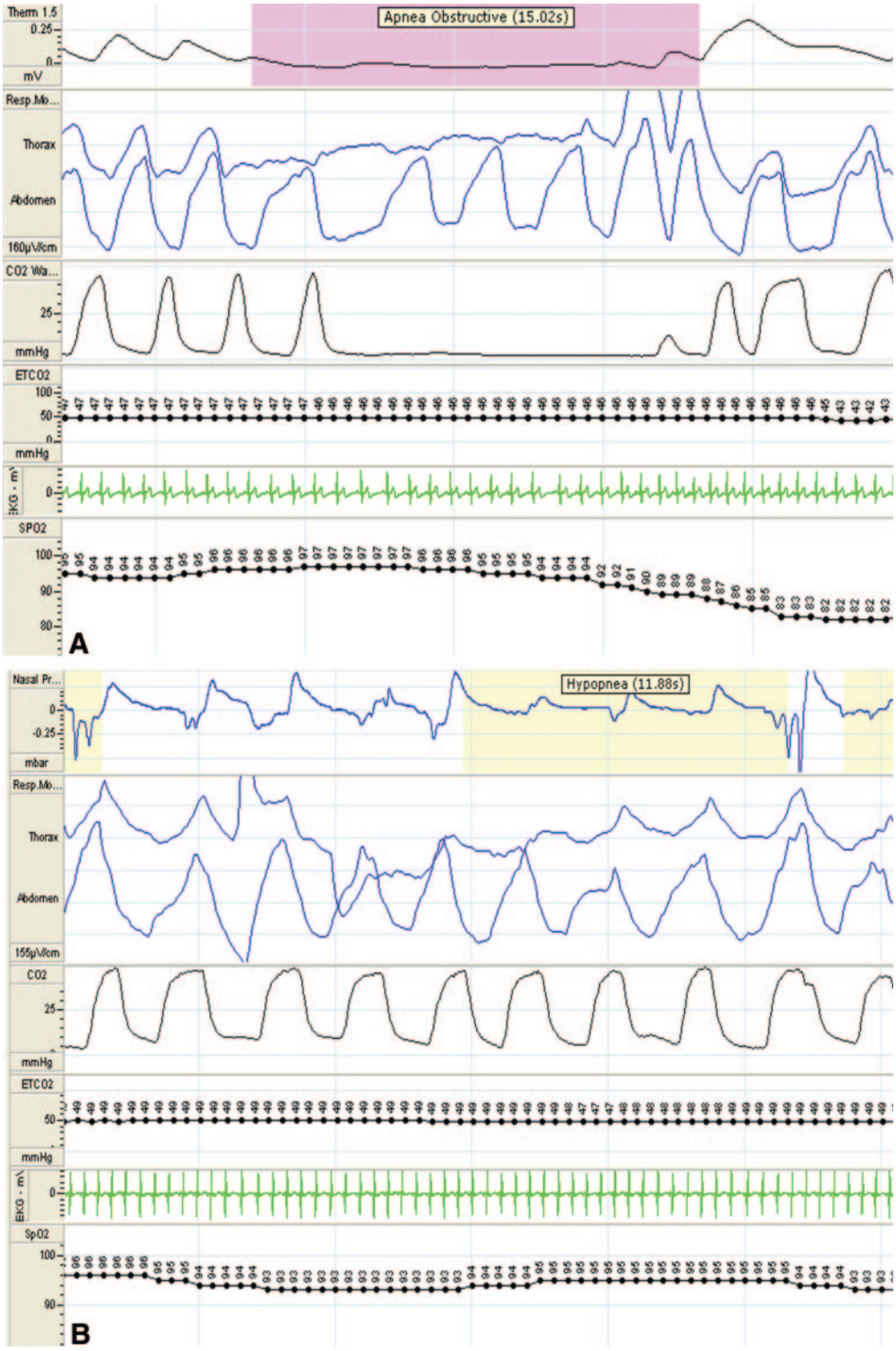

Figure 1. Typical polysomnographic recordings demonstrating: (A) Obstructive apnea. Note how the nasal thermistor and expired CO_2 tracings flatten while paradoxical respiratory efforts occur. The event is accompanied by a decrease in arterial oxygen saturations. (B) Obstructive hypopnea. The hypopnea is characterized by a decrease in nasal pressure signal associated with continuing paradoxical respiratory effort and desaturation. (C) Central apnea is distinguished from obstructive apnea by the absence of respiratory effort, which leads to the loss of airflow/pressure signals. This tracing shows one central apnea episode.

Congenital syndromes carry an increased risk of OSA (Table 6). Patients with Trisomy 21 may be predisposed to OSA,[75–78] particularly those with midface and mandibular hypoplasia, a small upper airway combined with relatively large and medially positioned tonsils, macroglossia, glossoptosis, increased secretions, obesity, and generalized hypotonia.[79]

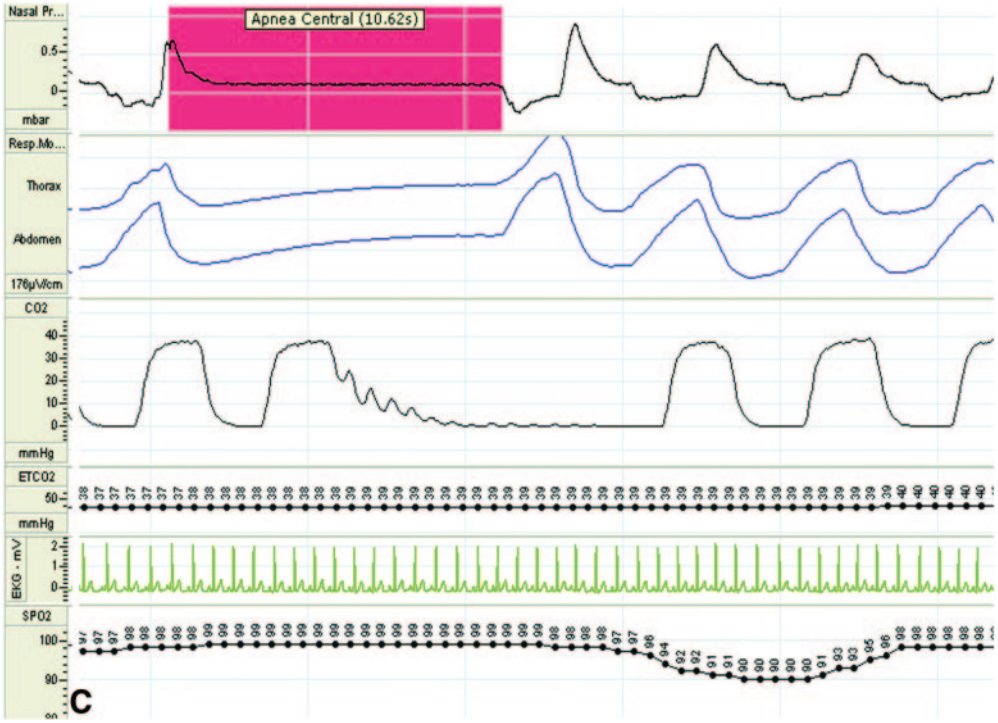

Figure 1. Continued.

Chronic OSA can have both reversible and irreversible consequences for the airway. Children with OSA have increased pharyngeal collapsibility and lose normal neuromotor responses to hypercarbia and negative pressure.[22,80,81] This increased collapsibility occurs with even mild OSA and may predispose these patients to more severe pharyngeal collapse later in life.[81]

Reversible and irreversible effects on the cardiovascular system are also possible. Chronic obstructed breathing leads to chronic hypoxemia, hypercarbia or both and, if left untreated, can eventually lead to pulmonary hypertension.[23,24,70] Pulmonary hypertension develops from vasoconstriction of the pulmonary arterial vessels in response to the chronic nocturnal hypoxemia, hypercarbia, and acidosis that accompany severe untreated OSA.[82–86] In a series of 92 children with adenotonsillar hypertrophy, 3.3% developed pulmonary hypertension that was reversed with adenotonsillectomy.[87] Cardiovascular morbidity is associated with endothelial dysfunction. Gozal et al.[88] showed that soluble CD40 ligand levels (sCD40L triggers inflammatory and procoagulant states) were elevated in children with polysomnography-proven OSA; tonsillectomy produced both significant improvements in AHI and sCD40L in all children except those with significant family histories of cardiovascular diseases.

Cardiac dysfunction associated with OSA may be manifested by structural and functional changes of both ventricles. Right ventricular dysfunction develops from chronically elevated pulmonary pressure and negative intrathoracic pressure created by breathing against a partially closed upper airway over time.[70] If pulmonary hypertension is left untreated, cor pulmonale is eventual and is diagnosed by echocardiographic findings of right ventricular hypertrophy, ventricular enlargement, pulmonary and tricuspid valve insufficiency, decreased ejection fraction, and dilation of the pulmonary artery.[70,89,90] In time, right atrial pressure increases, and decreased venous return to the heart results in peripheral edema, hepatic congestion, and ascites. Fortunately, right ventricular dysfunction and cor pulmonale may be reversible with surgical treatment of OSA.[83,86,89]

Although right ventricular dysfunction is classic, biventricular hypertrophy can develop. It is more likely to be seen in patients with severe OSA, but has been reported in patients with only mild OSA.[91] Children with OSA show signs of enhanced sympathetic activity,[92] autonomic dysfunction[93], and endothelial dysfunction.[88] Systemic and diastolic hypertension or a trend toward higher arterial blood pressures has been documented in children with OSA.[24,94–96] Higher blood pressures, especially at night, have been associated with increased severity of OSA that is particularly related to desaturation events,[97,98] even though the blood pressures reported might not have been high enough to require treatment with antihypertensives. Other studies show evidence of endothelial dysfunction with OSA but no hypertension.[88] In adults, it has been shown that intermittent hypoxia, not hypercapnia, is the critical stimulus for OSA-associated sympathetic activation, endothelial dysfunction, oxidative stress and inflammation, which produce cardiovascular dysfunction.[99]

Right ventricular dysfunction and overload leads to bowing of the ventricular septum and subsequent

Table 4. A Severity Ranking System Based on Polysomnography

	Apnea-hypopnea index	Oxygen saturation nadir
Normal	0–1	>92
Mild OSA	2–4	
Moderate OSA	5–9	
Severe OSA	>10	<80

Peak $ETco_2$ values and percent of time spent with $ETco_2$ >50 mm Hg should also be considered when assessing severity.

OSA = obstructive sleep apnea syndrome.

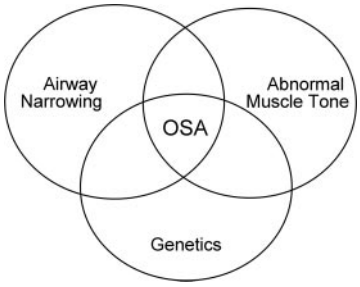

Figure 2. Pathophysiology of pediatric obstructive sleep apnea syndrome. The different influences on the airway are additive. Adapted from Marcus, Respir Physiol 2000;119:143–54.

Table 5. Facial and Airway Features Suggestive of Obstructive Sleep Apnea

Small triangular chins
Retro-position of the mandible
Steep mandibular plane
High palate
Long, oval-shaped face
Long soft palate
Large tonsils in association with the above facial features

Adapted from Guilleminault et al., Pediatrics 1996;98:871–82; Guilleminault et al., Otolaryngol Head Neck Surg 2007;136:169–75.

Table 6. Some Congenital and Medical Conditions Associated with Obstructive Sleep Apnea Syndrome

Achondroplasia
Apert syndrome
Beckwith–Wiedemann syndrome
Cerebral palsy
Choanal stenosis
Cleft palate patients after repair
Crouzon syndrome
Cystic hygroma
Down syndrome
Hallermann–Streiff syndrome
Hypothyroidism
Klippel–Feil syndrome
Mucopolysaccharidosis
Obesity
Osteopetrosis
Papillomatosis (oropharyngeal)
Pierre Robin syndrome
Pfeiffer syndrome
Pharyngeal flap surgery
Prader–Willi syndrome
Sickle cell disease
Treacher–Collins syndrome

From Sterni and Tunkel, Pediatr Clin North Am 2003;50:427–43, with permission.

increased left end-diastolic pressure that can result in pulmonary edema and pulmonary parenchymal damage.[70,100,101] Chronic hypoxia is an independent risk factor for the development of left ventricular hypertrophy,[91] which is a known risk factor for future cardiovascular disease.[102,103] Severe OSA doubles the risk of congestive heart failure in adult patients.[104] Patients with OSA can also experience postobstructive pulmonary edema due to either acute airway obstruction and generation of marked negative inspiratory pressures or in the relief of significant chronic airway obstruction. In both cases, physical damage to pulmonary capillaries, release of vasoactive mediators, and hydrostatic forces that result in fluid transudation to the pulmonary parenchyma may occur.[105]

Impaired growth has been seen in some children with OSA[23–25] and has been thought to be related to increased work of breathing during sleep.[106] These children have been shown to have impaired secretion of nocturnal growth hormone.[107] Improved growth has been reported after treatment with adenotonsillectomy.[106–109]

TREATMENT

Adenotonsillectomy is the treatment of choice for most children with OSA. Improvement in sleep-related airway obstruction and quality-of-life measures is estimated to occur in more than 75% of children after adenotonsillectomy.[110,111] However, persistent respiratory abnormalities may be seen in obese children and children with the most severe cases of OSA.[74,112] Suen et al.[26] suggested that a RDI >19.1 may be predictive of persistent OSA after adenotonsillectomy.

The range of surgical techniques for adenotonsillectomy reflects attempts to reduce the considerable postoperative discomfort and minimize the risk of hemorrhage after surgery. The use of radiofrequency volume reduction or powered intracapsular tonsillectomy can reduce perioperative pain by avoiding trauma to surrounding peritonsillar tissues. These procedures provide relief of disordered breathing in children with OSA, and recovery is more rapid than with total tonsillectomy techniques[113–115]; however, there is a risk of regrowth of tonsillar tissue.[116]

Children with abnormal craniofacial anatomy or abnormalities of neuromotor tone may require additional treatment of persistent OSA, including pharyngeal surgery, craniofacial surgery, and even tracheostomy.[117] Uvulopalatopharyngoplasty has been used for treatment of persistent OSA in children with neuromotor disease (such as cerebral palsy) or with craniofacial anomalies (such as those seen with Trisomy 21). Uvulopalatopharyngoplasty includes resection of the uvula, part of the soft palate and tonsillar pillars, with the goal of reducing upper airway obstruction at the level of the palate and oropharyngeal and nasopharyngeal levels. Tongue reduction procedures have been used in syndromic children with

obstructive macroglossia, and flap takedown can be performed for children with sleep-related airway obstruction after pharyngeal flap surgery for velopharyngeal insufficiency.[118] Craniofacial procedures, such as mandibular distraction/advancement, genioglossus advancement, and midfacial advancement, have been used to treat OSA that results from craniofacial structural abnormalities.[119]

Noninvasive nasal positive-pressure ventilation is a common medical treatment for OSA in children. Continuous positive airway pressure (CPAP) delivers a constant pressure to the airway; bi-level positive airway pressure (BiPAP) applies pressure that decreases during exhalation. The positive pressure mechanically stents the airway open and leads to improved functional residual capacity.[120] Both CPAP and BiPAP are used safely and successfully for children who have contraindications to adenotonsillectomy, persistent OSA after adenotonsillectomy, minimal adenotonsillar tissue, or prefer nonsurgical interventions.[120–127] The level of positive pressure required to eliminate obstructive apneas and normalize ventilation and night-time oxygen saturation must be determined in the sleep laboratory. Serial evaluation and adjustment of CPAP is required for growing children, as their pressure requirements change with time.[121,126] Complications of CPAP and BiPAP are usually minor and include local discomfort or irritation from poor mask fit, eye irritation, conjunctivitis, congestion, and skin ulceration. Children using noninvasive ventilation should have regular assessment of facial development. Midfacial hypoplasia has been reported with long-term use.[128] Regular evaluation of mask fit can help to avoid these difficulties. Infrequently, hypoventilation can be seen; BiPAP with a back-up rate can be used.[122] Pneumothorax and clinically significant reductions in cardiac output have not been reported in children treated for OSA.[121] The greatest limitation to the use of noninvasive positive-pressure ventilation in children is poor compliance.[121,125]

Nocturnal oxygen supplementation has been used as a temporary treatment for patients with significant hypoxia associated with OSA until definitive therapy can be provided.[121,129] In most cases, sleep-disordered breathing is not worsened. However, in some patients, supplemental oxygen may suppress the hypoxic ventilatory drive and worsen hypercapnia.[121] Nocturnal oxygen therapy for children with OSA should be initiated only under monitored conditions, including assessment of CO_2 exchange.

The use of orthodontic devices in the treatment of OSA in children seems promising. Orthodontic maxillary expansion can improve sleep-related airway obstruction in children with narrow palates,[72,130,131] but further studies are necessary to define the indications, proper candidates, and effectiveness of orthodontic treatment in the care of children with OSA.

Table 7. Key Questions to Ask Parents

Does your child have difficulty breathing during sleep?
Have you observed symptoms of apnea?
Have you observed sweating while your child sleeps?
Does your child have restless sleep?
Does your child breathe through his/her mouth when awake?
Are you worried about your child's breathing at night?
Do you have any family history of obstructive sleep apnea, sudden infant death syndrome, or apparent life-threatening events?
Does your child have behavioral problems?

Adapted from Li et al., Pediatr Pulmonol 2006;41:1153–60; Brouilette et al., J Pediatr 1984;105:10–14; Messner, Otolaryngol Clin North Am 2003;36:519–30; McNamara and Sullivan, J Pediatr 2000;136:318–23; Whiteford et al., Arch Dis Child 2004;89:851–5.

ANESTHETIC MANAGEMENT

Children with OSA present for all types of surgical and diagnostic procedures. Although the most studied procedure for this patient population is adenotonsillectomy, there is evidence that perioperative complications are increased in OSA patients after all types of surgery.[132]

Preoperative Assessment

Primary care providers do not routinely screen patients for OSA, nor do they demonstrate adequate knowledge about pediatric sleep disorders.[6–8] The prudent anesthesiologist should screen patients beginning with the question: Does your child snore? A history of nightly snoring is a sensitive (91%) but not completely specific (75%) marker of OSA.[32] If a patient regularly snores, additional focused questions may help to clinically identify those with OSA (Table 7), especially in patients with known OSA risk (Table 6).

Children <1-yr-of-age comprise a special group of patients that has not been adequately studied, but there is OSA in this age group. Presenting signs are snoring, apnea, failure to thrive, developmental delay, and recurrent respiratory infections.[133] It is not clear if infants are in a subset of OSA with more severe disease, more comorbidities, or genetic predisposition. Some infants studied at 2-mo-of-age showed resolution of OSA between 6 and 12-mo-old.[69]

The physical examination should include an airway assessment: nasal anatomy, ability to breathe through the nose, presence of elongated facies, oral aperture size, mandibular size, intermaxillary distance, thyromental distance, tonsillar size, tongue volume, body habitus, and Mallampati score.[134] Guilleminault et al.[71] showed increased risk of sleep-disordered breathing in children with the physical features listed in Table 5. The examination of the patient should also include an assessment of muscle tone, handling of oral secretions, and observation of facial malformations. When history, physical examination, and sleep laboratory data are combined, risk assessment is more accurate.[56]

Most children presenting for adenotonsillectomy do not need cardiac evaluation. However, adult patients with multiple episodes of severe hypoxemia,

Table 8. Clinical Features that Predict Respiratory Compromise After Adenotonsillectomy and, in Some Cases, Persistent Obstructive Sleep Apnea

Severe obstructive sleep apnea on polysomnography
History of prematurity, especially with respiratory disease
Age <3 yr
Morbid obesity
Nasal problems (deviated septum, enlarged turbinates)
Mallampati score 3 or 4
Neuromuscular disorders/disordered pharyngeal tone
Genetic or chromosomal disorders
Craniofacial disorders
Enlarged lingual tonsils
Upper respiratory infection within 4 wk of surgery
Cor pulmonale
Systemic hypertension
Marked obstruction on inhalational induction
Disordered breathing in the postanesthesia care unit
Difficulty breathing during sleep
Growth impairment due to chronic obstructed breathing

Adapted from Blum and McGowan, Paediatr Anaesth 2004;14:75–83; Guilleminault et al., Otolaryngol Head Neck Surg 2007;136:169–75; Gerber et al., Arch Otolaryngol Head Neck Surg 1996;122:811–14; McGowan et al., Pediatr Pulmonol 1992;13:222–6; Fricke et al., Pediatr Radiol 2006;36:518–23.

defined as oxygen saturation <70%, are at risk of left ventricular dysfunction[135] and childhood OSA is associated with hypertension and arterial blood pressure dysregulation.[136,137] Patients with cardiac involvement are at increased risk of perioperative cardiopulmonary complications.[70,82,83] Although the available data are somewhat limited in the pediatric population, we recommend cardiac evaluation for any child with signs of right ventricular dysfunction, systemic hypertension, or multiple episodes of desaturation below 70%. Electrocardiogram and chest radiograph are not sensitive evaluation tools; echocardiography is recommended.[102,138] Routine blood gas analysis is not recommended, but a basic metabolic panel can identify a patient with compensatory metabolic alkalosis in response to chronic hypercarbia, and a hemoglobin level may identify the patient with severe chronic hypoxemia.[139]

Preoperative CPAP has been used to reduce the postoperative complication rate and increase airway patency in adult OSA patients,[140,141] and may be beneficial for certain pediatric patients.[121] In our practice, children with very severe OSA who are at risk for persistent OSA (Table 8) and those with cardiovascular complications from OSA are considered for preoperative CPAP/BiPAP therapy. Effective CPAP/BiPAP therapy may improve pulmonary hypertension and reduce the patient's surgical risks.[142] This therapy is usually initiated by a pediatric pulmonologist, and the pressures required to treat the patient's OSA are determined in the sleep laboratory. The child's preoperative CPAP/BiPAP regimen can also be used in postoperative care, and the patient's response to adenotonsillectomy and need for long-term CPAP can be determined several weeks postoperatively.

Intraoperative Management

Studies that compare one anesthetic to another for adenotonsillectomy are few, and no technique is preferred. Sedative and anesthetic medications alter the CO_2 response curve, theoretically placing OSA patients at higher risk of sedation and anesthesia-induced respiratory complications.[143] Patients with OSA rescue themselves during obstructive episodes by arousal from sleep, but sedatives or residual anesthetics may make it impossible for patients to arouse themselves during obstructive episodes. Consequently, short-acting anesthetics should be chosen.

The use of sedatives in pediatric OSA patients has not been well studied, and the few studies that exist do not include control groups. Preoperative administration of midazolam 0.5 mg/kg to 70 children undergoing adenotonsillectomy for OSA (diagnosed as severe in 40% of subjects by polysomnography) resulted in two children having respiratory events; one had a self-limited desaturation event before surgery, and one had postoperative obstruction with desaturation, requiring a nasal airway.[144] In another small series, patients with Trisomy 21 were successfully sedated for magnetic resonance imaging studies with dexmeditomidine and ketamine without airway instrumentation.[79] Sedatives used before the induction of general anesthesia may delay emergence in patients, especially for short cases.[145,146] Without more evidence, we conclude that patients with OSA can receive sedatives but require monitoring until recovery can be demonstrated.

There is no consensus regarding a best anesthetic induction strategy in OSA, but common sense dictates that some patients with OSA require approaches different from those used with normal patients. Children with altered bony anatomy or syndromes are at higher risk for having a difficult airway. Induction of anesthesia with volatile anesthetics results in airway collapse from relaxation of the genioglossus muscle, thus placing the OSA patient at high risk for airway obstruction.[147–149] Positioning in an upright or lateral position, use of a jaw thrust maneuver, delivery of positive pressure by face mask and placement of an oral airway may aid in relieving the obstruction.[150,151] In cases when the patient is only partially anesthetized and suffering from airway collapse, an airway device may not be tolerated. If the patient remains in a state of obstruction, desaturation ensues. Severe airway obstruction in a spontaneously breathing patient may result in a very high negative inspiratory force generated although the patient is inhaling against the collapsed pharynx or closed glottis; the increased pulmonary blood flow and pulmonary microvascular pressure that ensues can result in postobstructive pulmonary edema.[70,117] IV induction can be used to rapidly induce a deep plane of anesthesia ready for airway instrumentation. This technique may be preferable for patients with very severe OSA. Children with simple adenotonsillar hypertrophy with normal

body habitus and without maxillofacial malformations are often easily mask-ventilated once an oral airway is placed, and endotracheal intubation is likewise usually straightforward. Children with OSA in addition to craniofacial abnormalities or other significant airway disorders must be evaluated for potential difficult intubation. A recent study of 40,000 adults showed OSA to be an independent predictor of impossible mask ventilation (Kheterpal S et al. Incidence, predictors, and management of impossible mask ventilation; A review of 40,000 anesthetics, 2008 ASA annual meeting, abstract A1243). Whether this association applies to the pediatric population with OSA is not known.

Laryngeal mask airways are used by some anesthesiologists for adenotonsillectomy,[152–154] but studies are lacking regarding their use in patients with OSA for this procedure. They can also be used in OSA patients with bony abnormalities for the purpose of managing a difficult airway.[155,156]

Upon completion of the procedural anesthetic, patients should be awake and have adequate strength to maintain the upper airway before tracheal extubation. We do not recommend deep extubation; patients with severe OSA and those with comorbidities are at risk of persistent OSA after surgery (Table 8). Extubation should take place in a controlled environment with appropriate personnel and equipment available; once stable, the extubated patient can be transported to the appropriate care unit. Before extubation, we sometimes place nasal airways in patients with severe OSA. The child who continues to have significant obstructive episodes after extubation can be positioned in the lateral decubitus or prone position to help relieve the obstruction. CPAP or BiPAP can be used to assist ventilation and relieve airway collapse. The placement of a nasal airway before extubation might be considered in more severe cases. Reintubation may be required in an occasional patient.

Efforts are made to reduce the risk of postoperative vomiting and pain after adenotonsillectomy. A study that compared anesthetic techniques using sevoflurane and propofol with muscle relaxant and fentanyl showed no statistical difference in postoperative vomiting.[157] Steroids have been shown to improve postoperative oral intake and reduce pain and vomiting.[158–161] A dosing study of IV dexamethasone for adenotonsillectomy showed that low dose (0.0625 mg/kg) is just as effective as high dose (1 mg/kg) in reducing postoperative pain and vomiting.[162] In another study, adult tonsillectomy patients who were treated with steroids had less pain, less nausea, and vomiting and improved healing as compared with those who were not treated.[163] Although nasal steroids have been used to treat pediatric OSA, with improvement in sleep-disordered breathing,[164] no studies have examined the use of nasal steroids to reduce perioperative swelling.

A review and meta-analysis of antiemetics showed that antiserotonergic drugs and dexamethasone were most effective for reducing postoperative vomiting after tonsillectomy. A randomized, double-blind study comparing the antiemetic effects of metoclopramide (0.5 mg/kg) versus ondansetron (0.1 mg/kg) in children after tonsillectomy showed ondansetron to be superior.[165,166] Antibiotics are often given to children perioperatively to reduce postoperative complications of adenotonsillectomy. A meta-analysis examining the use of antibiotics for these patients showed an associated 1-day reduction in time required for return to normal oral intake.[167]

Postoperative Management and Analgesia

Children with OSA usually need pain medication after surgery, yet chronic hypoxemia renders them more susceptible to the respiratory depressant effects of opioids.[168,169] OSA patients have been shown to have a higher incidence of apnea after administration of 0.5 μg/kg of fentanyl, and diminished minute ventilation during spontaneous ventilation under general anesthesia with inhaled anesthetics, when compared with non-OSA patients.[170] OSA patients may also be more sensitive to the analgesic effects of opioids. Children with oxygen saturation nadir of $<$85% on polysomnography required half the morphine dose as those with less desaturation to achieve the same level of analgesia.[171] If high doses of opioids are required for an OSA patient after a surgical procedure, intensive postoperative cardiopulmonary monitoring must be considered. When possible, regional anesthesia and/or analgesia should be used. For short procedures, one approach is to minimize opioids intraoperatively and then titrate them to effect when the child is awake, tracheally extubated, and in a monitored setting.[70] Less painful procedures may only require non-opioid analgesics, such as acetaminophen or nonsteroidal antiinflammatory drugs (NSAIDS).

Methods for managing posttonsillectomy pain without opioids have been studied. Surgical options to spare the tonsillar bed have been shown to reduce the need for pain medications.[172,173] Mixed results have been reported with the use of local anesthetic infiltration to the tonsillar bed, and this strategy carries the risk of intravascular injection.[174,175] One group reported administering ketamine to the tonsillar region, finding it to be an effective analgesic with no sedating side effects in patients without OSA.[176,177] Tramadol, a synthetic selective mu_1 agonist, is an alternate medication for moderate pain management with potentially less respiratory depression, but it is not currently approved by the Food and Drug Administration for pediatric patients in the United States. In an Australian study, tonsillectomy patients with moderate OSA who received morphine had a significantly higher risk of desaturation than those given Tramadol, although they were more comfortable.[178] The use of NSAIDS in posttonsillectomy patients has been avoided because of reports of associated postoperative bleeding[139,179];

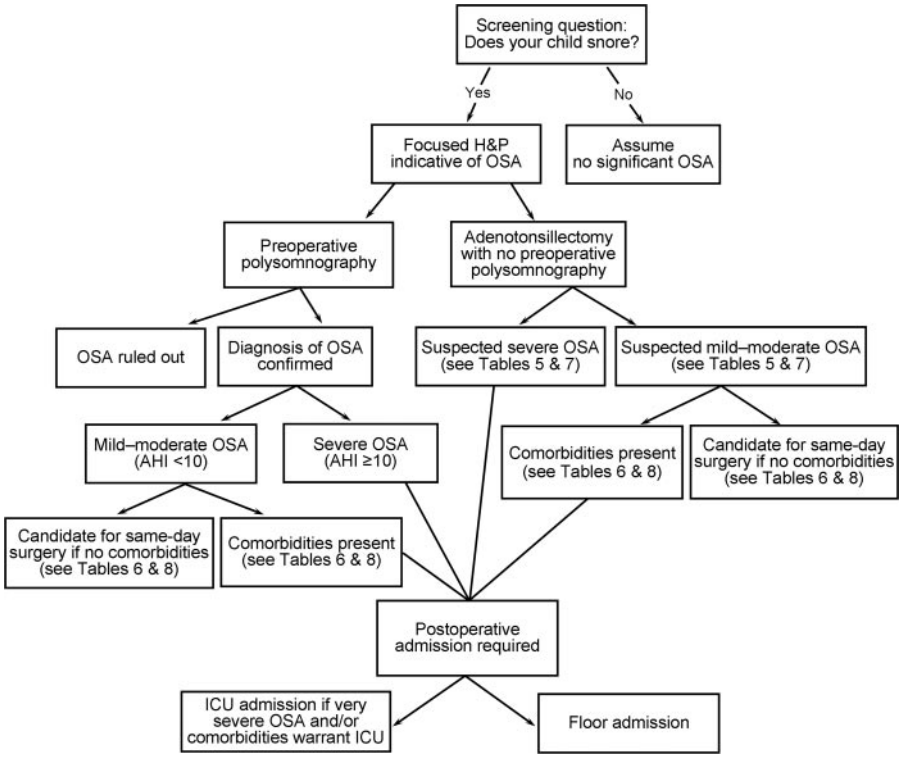

Figure 3. Algorithm for risk assessment and disposition planning.

however, a systematic review did not find an increased risk of reoperation for bleeding and, additionally, found less vomiting when NSAIDS were part of the analgesic regimen.[180] A different meta-analysis showed that a significant risk of perioperative bleeding is associated with the use of aspirin for analgesia but no increased risk with other NSAIDS.[181] Furthermore, a large retrospective review on the subject concluded that ibuprofen should be used for the relief of pain after adenotonsillectomy.[182] Others have written that the use of NSAIDS after attainment of hemostasis is reasonable, given the evidence and pharmacokinetics of the nonaspirin NSAIDS.[183] Consequently, the bulk of evidence supports the use of nonaspirin NSAIDS for postoperative analgesia.

Numerous authors have demonstrated that pediatric patients with OSA are at increased risk for postoperative respiratory complications.[17,56,184–186] Rates of complications range from 6.4% to 27% but depend on age, severity of OSA, uniformity of diagnosis, and comorbidities.[17,57,186,187] Children <3-yr-of-age have twice the risk of children who are 3–6 yr of age.[186] Children with OSA had a 23% rate of respiratory difficulties after adenotonsillectomy, with the greatest risk seen in children <3 yr and those with preoperative RDI >10.[54] Nursing intervention was required to treat complications in 60% of children with severe OSA; complications included oxygen desaturation <90%, increased work of breathing and changes on a chest radiograph (edema, atelectasis, infiltrate, pneumothorax, pneumomediastinum, or pleural effusion).[55,57,187] Other complications associated with severe OSA include laryngospasm, apnea, pulmonary edema, pulmonary hypertensive crisis, pneumonia, and perioperative death.[54,56,188,189] Although adenotonsillectomy improves most patients, children with disorders of pharyngeal tone or craniofacial anatomy may have residual airway obstruction during the postoperative period and require close observation to assess the need for intervention.[134] The surgeon and anesthesiologist must agree on the discharge plan (Fig. 3).

We recommend that polysomnography be performed to help determine the postoperative disposition of patients with OSA. Without objective evidence of the severity of OSA, patients cannot be discharged with confidence. Polysomnography is essential for patients with comorbidities and high-risk features (Table 8). Children with OSA who are identified as high risk for respiratory compromise require overnight inpatient monitoring after surgery in a setting where signs of respiratory depression and airway obstruction can be recognized and prompt intervention can occur.[54,120,190] Two study groups have reported the onset of respiratory compromise during sleep ≥5 h postoperatively in children with OSA, and those with severe OSA had significantly more overnight obstructive episodes on the first postoperative night when compared with children who had mild OSA.[187,191] Because REM rebound is a possibility beyond the first postoperative night,[192] careful thought must also be given to whether it is safe to discharge patients with severe OSA on day 2, especially if opioids are needed to control pain.[171] Postoperative intensive care unit admission is reserved for very severe OSA, very young children and those with comorbidities that cannot be managed on the floor (Fig. 3).

Table 9. American Society of Anesthesiologists Risk Assessment, Scoring System

	Points
Severity of sleep apnea	
None	0
Mild	1
Moderate	2
Severe	3
Invasiveness of surgery and anesthesia	
Superficial surgery/local anesthesia and no sedation	0
Superficial surgery/moderate sedation or general anesthesia	1
Peripheral surgery with regional anesthesia and moderate sedation	1
Peripheral surgery with general anesthesia	2
Airway surgery with moderate sedation	2
Major surgery/general anesthesia	3
Airway surgery/general anesthesia	3
Requirement for postoperative opioids	
None	0
Low-dose oral opioids	1
High-dose oral, parenteral, or neuraxial opioids	3
Total score	

This table is a possible format developed by the ASA Task Force. It has not been subjected to prospective study, especially in the pediatric population. Risk stratification was determined by a panel of experts; a score of 4 suggests possible increased risk, and scores of ≥5 suggest significantly increased risk. From Gross et al., Anesthesiology 2006;104:1081–93, with permission.

Patients with mild-to-moderate obstructive disease (defined as AHI <10) and no comorbidities can usually be discharged home the same day if they are >3-yr-of-age. Polysomnography performed on otherwise healthy children with mild OSA on the first night after adenotonsillectomy showed that the number of apnea events decreased, and oxygen saturation during sleep improved immediately after surgery.[139,193] If a child with OSA is to be discharged on the day of surgery, an early morning operative time has been recommended by some,[191] and the ASA practice guidelines suggest monitoring patients longer than those without OSA.[10] We recommend a 2-h minimum postanesthesia care unit stay. Research is greatly needed in this area.

Determining which patients are at high risk for postoperative respiratory complications is a challenge because polysomnography is not a standard preoperative test for patients with suspected OSA, even though it is recommended by the American Thoracic Society and the American Academy of Pediatrics.[120,194] One study showed that <12% of school-aged children who underwent adenotonsillectomy for OSA had prior polysomnography.[195] Furthermore, it is clear that although sleep studies may be improved by tonsillectomy, they do not return to normal immediately after surgery, and certain patients continue to be at high risk of perioperative complications.[187] The ASA OSA Task Force attempted to use a scoring system identify patients at highest risk (Table 9); however, this system uses scoring of the severity of sleep apnea, which may not be available because of lack of polysomnography testing.[10]

CONCLUSIONS

Patients with significant OSA are clearly at higher anesthetic risk in the perioperative period than are patients with normal upper airways. Anesthesiologists should routinely screen patients for snoring, airway dysfunction, airway anatomic disorders and other coexisting diseases that can increase risk from OSA in the postoperative period. Rational, safe, cost-effective decision making for individual patients hinges on accurate risk assessment in the preoperative period. Preoperative diagnostic techniques that are affordable and readily available are needed. Anesthesiologists, along with pulmonologists and otolaryngology surgeons, should strive to develop and evaluate ways of identifying children at high risk to determine safe disposition in the postoperative period.

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