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Obstructive sleep apnea syndrome in children: controversies in diagnosis and treatment

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Obstructive sleep apnea syndrome (OSAS) is a common medical problem in adults that is increasingly recognized in children. It occurs at all ages, from newborns to teens. Several studies indicate prevalence rates of approximately 2% [1–4], with the peak prevalence from 2 to 8 years of age, when tonsils and adenoids are largest in relation to the upper airway size [5]. OSAS is characterized by prolonged partial upper airway obstruction or intermittent complete pharyngeal obstruction that disrupts normal ventilation and sleep continuity [6]. If unrecognized and untreated, OSAS can lead to neurobehavioral, growth, and cardiovascular sequelae in childhood [7–13].

The American Academy of Pediatrics recently published a clinical practice guideline for the diagnosis and management of OSAS in the otherwise healthy child; the reader is strongly encouraged to review the publication [9]. Key recommendations from the guideline are summarized in **Box 1**. The technical report that accompanies the guideline provides the reader with a comprehensive, evidence-based review of the literature up through the year 2000 [10]. In brief, this guideline advises pediatricians and parents that snoring may signal OSAS, which is a serious condition with important negative sequelae, that polysomnography is currently the most effective means to diagnose OSAS, and that surgical treatment is effective in 75% to 100% of children.

The goals of this article are to (1) highlight areas of controversy and uncertainty that limit the development of more definitive standards of practice, (2) up-

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Box 1. Summary of the clinical practice guidelines for the diagnosis and management of childhood obstructive sleep apnea syndrome in the otherwise healthy child

All children should be screened for snoring
Complex high-risk patients should be referred to a specialist
Patients with cardiorespiratory failure cannot wait for elective evaluation
Diagnostic evaluation is useful in discriminating between primary snoring and OSAS
Adenotonsillectomy is the first line of treatment for most children; continuous positive airway pressure is an option for children who are not candidates for surgery or who do not respond to surgery
High-risk patients should be monitored as inpatients postoperatively
Patients should be re-evaluated postoperatively to determine whether additional treatment is required

From American Academy of Pediatrics. Clinical practice guidelines: diagnosis and management of childhood obstructive sleep apnea syndrome. Pediatrics 2002;109(4):704–12; with permission.

date the reader on several newer publications relevant to diagnosis and treatment of childhood OSAS, and (3) identify areas for future clinical research. These knowledge gaps in the diagnosis and treatment of childhood OSAS present important pediatric clinical or health services research opportunities. Key areas of controversy or uncertainty are listed in [Box 2](#) and detailed in the following discussion.

Epidemiology of obstructive sleep apnea syndrome: prevalence and risk factors

Knowing the prevalence and risk factors for childhood OSAS is key to assessing the magnitude of the public health impact and identifying children at highest risk for developing OSAS and benefiting from effective treatment. Prevalence estimates for childhood OSAS have varied from 0.7% to 2.9%. Available epidemiologic data for sleep-disordered breathing (SDB) in children have been limited and are derived mainly from community surveys using two-stage sampling approaches that objectively measure SDB—only subsamples of high- and low-risk children based on symptoms [1–3,14,15]. Because parent

Box 2. Key areas of controversy or uncertainty in childhood obstructive sleep apnea syndrome*Epidemiology*

- How prevalent is OSAS?
- Which high-risk groups should be targeted for screening and treatment?

Clinical morbidity and outcomes

- Which adverse health outcomes are causally associated with OSAS?
- Is primary snoring really “benign?”

Diagnosis

- What is the best way to diagnose childhood OSAS?
- Which physiologic measurements (eg, oxygen saturation, apnea or hypopneas, carbon dioxide levels, arousals, or sleep architecture) have the greatest contribution to the diagnosis of OSAS?
- Which physiologic measurements are the best predictors of adverse outcomes?
- Which sensors provide the most reliable estimates of those physiologic measures?

Treatment

- What threshold of respiratory disturbance should be treated?
- How effective are the treatments?
- Which patient subgroups have the highest risk for incomplete resolution of OSAS after adenotonsillectomy and should be targeted for re-evaluation?
- How reversible are the negative health outcomes associated with OSAS?

reports cannot predict obstructive sleep apnea in children reliably, prevalence estimates from small subsamples of only “symptomatic” children may be unreliable [16,17]. One study in which all children had objective testing for SDB was not optimal for prevalence estimation because it included a broad age range of 2 to 18 years and included related family members [3]. In a new, more

definitive, community-based study that used objective criteria to measure SDB in 850 participants, the estimated prevalence rate in 8- to 11-year-old children was 2.2% [4].

Although childhood OSAS is commonly associated with adenotonsillar hypertrophy, it is not caused by enlarged tonsils and adenoids alone because children with OSAS do not have obstruction during wakefulness, adenotonsillar size does not correlate with OSAS, and a small percentage of children with adenotonsillar hypertrophy—but without other known risk factors for OSAS—are not cured by adenotonsillectomy [6]. Other risk factors for childhood OSAS include positive family history of OSAS, obesity, African-American race, sinus problems, and persistent wheeze [3]. In contrast to OSAS in adults, male gender is not a risk factor for childhood OSAS in prepubertal children [3,4,6]. Obese boys older than 15 years are more likely to be habitual snorers, however [18]. Of note, prematurity has been newly identified as a significant independent risk factor for SDB in a community-based sample of 8- to 11-year-old children [4]. Former preterm children were three to five times more likely to have SDB than full-term children. This study confirmed the increased vulnerability of African-American children, who were four to six times more likely to have SDB compared with white children [4]. Another recent study showed that African-American children were less likely to have undergone tonsillectomy or adenoidectomy but were more likely to have SDB after those surgeries [19]. Because preterm children are at increased risk for developmental delays and African-American children are at higher risk for socioeconomic disadvantage, these two groups may be more vulnerable to negative health outcomes associated with untreated OSAS and may benefit from more intensive screening for OSAS by primary care providers.

Clinical morbidity and negative health outcomes in childhood obstructive sleep apnea syndrome: what is causal and which are associations? Is primary snoring truly “benign”?

Better characterization of the clinical morbidity and health outcomes attributable to childhood OSAS is the next area of challenge. Although childhood OSAS is associated with important sequelae (eg, neurobehavioral problems, growth inhibition, and cardiovascular complications), the prevalence of these complications is unclear because of the lack of population-based cohort studies [10]. Most published articles on the complications of OSAS were reports of retrospective case series or prospectively collected—but uncontrolled—data that compared measures before and after surgical intervention. For neurobehavioral complications of OSAS, cross-sectional studies suggest a nearly threefold increase in behavioral problems and neurocognitive dysfunction in children with SDB [10].

Because most of these studies did not definitively separate children with primary snoring (believed not to require treatment) from children with OSAS [16], the true prevalence of behavioral and learning problems in children with OSAS

compared with primary snoring is unclear. Conversely, increasing evidence suggests that so-called “primary snoring,” even in the absence of well-defined OSAS, may not be benign. Supporting that view, several recent studies suggested that snoring is associated with adverse behavioral and learning outcomes. Using parent-reported questionnaire data, investigators found that children with lower academic performance in middle school were more likely to have snored during early childhood and to have required adenotonsillectomy for snoring compared with their better performing classmates [8]. The study also suggested that there may be critical periods in development when OSAS should be treated to reverse or prevent negative health outcomes. Two recent studies of children seen at community-based pediatrics clinics suggested that snoring and other symptoms of SDB were associated with inattention, hyperactivity, and conduct problem [11,13]. The causal relationship between OSAS and inattention or hyperactivity remains unclear in these cross-sectional studies. Finally, the precise mechanism for the daytime neurobehavioral morbidity is uncertain. Although excessive daytime sleepiness plays a predominant role in the daytime neurocognitive dysfunction in OSAS in adults, nocturnal sleep fragmentation and daytime sleepiness have not been predominant features of childhood OSAS [7,16,20]. Investigators found an increased propensity for daytime sleep (as suggested by shortened daytime sleep latencies) in a clinical sample of children with OSAS diagnosed by polysomnography compared with control children. The increased sleepiness correlated with increasing apnea or arousal indices, degree of nighttime hypoxemia, and body mass index [7].

In terms of other types of health outcomes, three new studies suggested that SDB negatively impacts health-related quality of life. In an observational genetic-epidemiologic cohort study that included 298 pediatric participants, SDB was associated with lower levels of two generic health-related quality-of-life measures, overall physical health, and bodily pain [21]. Two additional studies that used OSAS-specific quality-of-life measures found improvements after adenotonsillectomy in convenience samples of snoring, symptomatic children who were referred for surgical intervention at tertiary pediatric otolaryngologic practices. Validity and generalizability of these findings are limited, however, given the highly selected patients, few of whom had polysomnographic confirmation of OSAS, and other study design issues [22,23].

Although failure to thrive is a well-known complication of OSAS, especially in younger syndromic children [24], the prevalence of this complication has not been assessed systematically. With increased recognition of childhood OSAS and earlier identification of less severe cases, the incidence of the complication may be declining. Case series suggest that OSAS has an inhibitory effect on growth and that growth, especially weight gain, accelerates after surgery for OSAS even in obese children [25,26]. There have been conflicting data as to whether this effect is attributable to increased metabolic expenditures associated with OSAS [27–29]. A recent study found that growth hormone secretion (as measured by circulating insulin-like growth factor [IGF-1] and IGF binding protein 3 concentrations) was impaired in children with both OSAS and primary snoring. These levels increased

significantly in children with OSAS after surgical treatment [28]. Of note, the primary snorers showed a similar target height deficit and retarded bone age as children with OSAS, which further supports the concept of long-term abnormalities in growth regulation among primary snorers.

OSAS is associated with increased risk for systemic hypertension and cardiovascular morbidity in adults [30,31], but evidence for cardiovascular complications in childhood OSAS is more limited. Case series of childhood OSAS patients have shown increased diastolic blood pressure, autonomic alterations, ventricular hypertrophy and dysfunction, and pulmonary hypertension [12,32]. One recent study found abnormal left ventricular geometry in 15% of children with primary snoring and 39% of children with OSAS [12].

In summary, the evidence for adverse neurobehavioral, growth, and cardiovascular outcomes provide a clear rationale for treatment of OSAS, and newer studies that identify abnormalities in primary snoring place these children at risk for adverse health outcomes. The next key challenge and controversy is how best to define the spectrum of OSAS in children.

Diagnosis of childhood obstructive sleep apnea syndrome: which physiologic measures, sensors, and thresholds indicate disease? If polysomnography is the “gold standard,” what role do alternative diagnostic strategies play?

Snoring and obstructive apnea are the two extremes in the broad spectrum of upper airway resistance, narrowing, and collapse. Exactly where the transition from normal to pathologic condition occurs on that continuum is uncertain. The definition of what constitutes a pathologic condition in the snoring child has not been determined. Although methodologic standards and normal ranges for respiratory indices during sleep have been published [10,17,33,34], methods and interpretation vary. Current normative standards for polysomnographic determination of OSAS have been chosen on the basis of statistical distribution of data. Whether those standards have validity as predictors of the clinically important sequelae has not been established. The severity of the polysomnographic findings seems to be an important predictor of complications in the immediate postoperative period for adenotonsillectomy [10].

Overnight polysomnography performed in a sleep laboratory is currently the diagnostic test of choice for OSAS in children [9,10]. Although OSAS is one of the most common indications for adenotonsillectomy and contributes to the performance of approximately 274,000 procedures per year in children younger than age 15 [35], this clinical activity has not been accompanied by the implementation of standardized approaches for collecting, recording, and interpreting overnight sleep and breathing data in children. A wide interlaboratory variation in summary measures of the number of respiratory disturbances (apnea and hypopneas) during sleep is largely attributable to poor standardization of measuring techniques and a lack of consensus for defining apneas and hypopneas [36–38]. In adults, the variability in respiratory event identification and threshold

definitions resulted in marked differences in the absolute respiratory disturbance index level and prevalence estimates of SDB [39].

Similar analyses have been performed in children [40]. Median respiratory disturbance indices varied by more than 20-fold for definitions using the more liberal criteria for event definition (all respiratory events; ie, central or obstructive events and hypopneas with no requirement for associated desaturation) to the most conservative definition (using obstructive apneas only or obstructive apnea and hypopneas that required a 5% associated desaturation) [40]. Prevalence estimates for SDB based on respiratory disturbance indices that included central apneas were 40% to 140% higher than those that excluded central apneas [40]. These different approaches for quantifying respiratory disturbance index contribute to substantial variability in identification and classification of SDB in children and lead to discrepant estimates of its presence and severity. Standardized approaches for characterizing the respiratory disturbance index are needed to compare data from different laboratories and populations, ensure consistent case finding, assess accurately the public health impact of SDB in children, and determine the outcome of interventions. The role for newer technologies, such as the nasal pressure cannula to measure airflow during partial obstruction in OSAS [41] or faster, more artifact-resistant oximeters, currently is being assessed in children [42] and must be considered in that standardization process.

Although overnight polysomnography in the sleep laboratory is the current “gold standard” for diagnosis of childhood OSAS, especially for children, this testing is intrusive and costly and may adversely affect sleep quality. Only a limited number of sleep laboratories are skilled in evaluation of children, and there are few sleep medicine specialists with diagnostic expertise in children. These limited resources are barriers to efficient diagnosis of OSAS in children. The development of simple and less costly alternatives for diagnostic testing is highly desirable, as are simpler screening tests before full polysomnography. The current evidence for alternative diagnostic strategies for OSAS has been reviewed comprehensively in the technical report that accompanies the American Academy of Pediatrics guidelines [10].

In brief, strong evidence exists that clinical evaluation has unacceptably low sensitivity and specificity rates for predicting OSAS. Other strategies, including home audiotaping and videotaping, to supplement the clinical assessment are inadequately investigated. Pulse oximetry and daytime nap polysomnography seem to have high specificity and low sensitivity rates, which means that positive results are probably true but negative results must be confirmed using overnight polysomnography. The comparability of home and sleep laboratory overnight polysomnography seems promising, but additional studies using commercially available equipment in representative patient populations are needed for confirmation. Rigorous systematic assessment (reliability, accuracy, and cost effectiveness) of alternative and simplified diagnostic strategies for OSAS in children is critical in an attempt to extend clinical diagnostic services to children in the most efficient manner. Alternative diagnostic strategies are crucial to future randomized, controlled trials to determine an outcomes-based respiratory disturbance

threshold for treatment of OSAS, evaluate better the effectiveness of the various treatment modalities, and assess reversibility of adverse outcomes.

Treatment of childhood obstructive sleep apnea syndrome: what respiratory disturbance threshold should be treated? How effective are the treatments? Which patient groups need reassessment after adenotonsillectomy? How reversible are the negative health outcomes?

The evidence for the association of childhood OSAS with immediate or possibly long-term health consequences, especially behavioral and learning problems, impaired growth, and cardiovascular morbidity, is a strong rationale for treatment. Despite decades of treating children with OSAS, only limited information is available on the long-term consequences of pediatric OSAS and the degree of improvement in OSAS after intervention. The reader is referred to a recent comprehensive and evidence-based review of the current treatment modalities for childhood OSAS [32]. Although there is a strong consensus that adenotonsillectomy is the first-line treatment for childhood OSAS [9,10], the overall efficacy of this surgical intervention is uncertain given the limitations of the study designs in the currently available literature. Of note, when parent reports of postoperative symptoms serve as the main outcome measure for surgical efficacy, improvement or cure rates occur in 97% of patients. In contrast, when the outcome includes overnight respiratory measures, the cumulative cure rate falls to 80% [32]. This level of postoperative persistence of OSAS seems higher than expected. It is important to recognize, however, that studies of adenotonsillectomy efficacy that included objective pre- and postoperative respiratory measures likely targeted a different clinical population of children who were treated at tertiary medical centers, were more severely affected, and had other risk factors for incomplete resolution of OSAS after surgical intervention. Objective measurement of adenotonsillectomy effectiveness in more routine clinical practices has yet to be evaluated.

Finally, tracheotomy was once was the only surgical alternative when adenotonsillectomy failed. Currently, various surgical techniques have been advocated in addition to adenotonsillectomy. In one large series that included 70 children with various conditions who were treated with individualized surgery, tracheotomy was avoided in 90%, the respiratory disturbance index fell dramatically, and oxygenation improved [43]. The heterogeneity of the underlying conditions that may benefit from the various surgical approaches precludes any general recommendations, however, and must be individualized to optimize outcomes and avoid tracheotomy [44].

Of nonsurgical therapies, there are only case reports of pediatric experience with pharmacologic agents used in adults as palliative or second-line treatment of OSAS and insufficient evidence to support their use [32]. Systemic corticosteroids were ineffective in a single childhood OSAS study [45]. Two studies with intranasal topical corticosteroids indicated some partial and temporary benefit,

but rate of OSAS recurrence after discontinuation has yet to be determined, and larger cohort studies are needed [46,47]. Supplemental oxygen as a temporizing measure was evaluated in two small studies [48,49]. Although it improved some respiratory measures beyond oxygenation in a small proportion of children, it must be instituted with caution because two children developed significant hypoventilation [48,49].

Continuous positive airway pressure is a second-line treatment for childhood OSAS that is unresolved after adenotonsillectomy or for children who are not candidates for that surgery [9,10]. Clinicians should recognize that the patient-equipment interface and adequate family training are keys to the success of this therapy and can be determined only on an individual basis. Studies have demonstrated feasibility and efficacy if an appropriate patient-equipment interface is successfully established with behavioral training and family support, but compliance remains a challenge [50–53]. Identification of patients or families who are most likely to be compliant and achieve long-term benefit from this therapy has yet to be established.

Summary

Pediatricians and pediatric sleep medicine specialists have come a long way in understanding diagnosis and management of OSAS since the original reports describing OSAS in children. Although we have come a long way in understanding the risk factors, symptoms, signs, and potential adverse consequences of childhood OSAS, it is important to integrate knowledge with the unique aspects of individual patients and families who present for care. The following two case vignettes illustrate that integration in clinical practice.

Case 1

An 11-year-old obese African-American girl is still sleepy despite undergoing an adenotonsillectomy 4 months ago for snoring, difficulty breathing at night, witnessed apnea, and enuresis. She still takes a nap almost every day after school, and her sleepiness interferes with her school performance (she is failing several classes). Although she has some residual snoring after surgery, the apneas and enuresis disappeared. She recently was diagnosed with ADHD and received methylphenidate. Her mother stopped the medication because she was worried about her daughter taking chronic medication. The child goes to bed at 9:00 PM and falls asleep quickly but is difficult to wake up for school at 7 AM. Past medical history includes seasonal nasal allergies controlled with nasal corticosteroids. Several adult relatives in the family (including mother and father) are obese and snore. A maternal grandfather and two paternal uncles have OSAS that is treated with continuous positive airway pressure. Except for the child's obesity (body mass index 33 kg/m²), her physical examination was unremarkable.

What should you do?

The child has many risk factors for childhood OSAS, including African-American race/ethnicity, obesity, nasal airway inflammation, and a family history of OSAS. Her obesity is a risk factor for incomplete resolution of her OSAS. A repeat overnight polysomnography to look for residual OSAS and associated sleep disruption is indicated, despite parental reports of improved nocturnal symptoms. What is striking in this clinical scenario, however, is the daytime sleepiness and deteriorating school performance in the face of 10 hours of sleep at night and the marked clinical improvement in her nocturnal respiratory symptoms. At this point, the clinician should consider other explanations for the excessive daytime sleepiness, including narcolepsy that presents in childhood.

In this case, the repeat polysomnography showed only light, intermittent snoring, an apnea-hypopnea index of two events per hour, and an arousal index of nine per hour. The degree of daytime dysfunction in this child is out of proportion to polysomnographic findings. The multiple sleep latency “nap” test scheduled the morning after the polysomnography showed a mean sleep latency of 4 minutes (abnormally short) and sleep-onset REM periods in three of the four naps. The child has narcolepsy that requires long-term stimulant therapy for management. It is not uncommon for narcolepsy to be misdiagnosed as inattention, learning disabilities, poor sleep habits, or psychiatric disorders. The take-home message is that unexplained excessive sleepiness, even in a snoring child, may be the hallmark of childhood narcolepsy. Weight management should be a long-term goal.

Case 2

The patient is a 6-year-old boy with loud snoring, restless sleep, enuresis, moderate adenotonsillar hypertrophy, severe ADHD symptoms, and poor academic performance in whom pharmacotherapy is being considered. The child has snored since infancy, and the parents are aware of the association between ADHD-like symptoms and unrecognized OSAS. They hope that polysomnography will confirm OSAS that can be “cured” by adenotonsillectomy and help avoid the need for stimulant medication.

The child’s bedtime is 9 PM, but bedtime resistance is a long-standing problem. The child “needs” a television in his bedroom and a parent to lay down with him to fall asleep. He often does not fall asleep until 11 PM and experiences multiple night wakings, sometimes prolonged, and waking that resolves when his mother lies down with him in his bed. It is hard to wake him up by 6:30 AM to get him on the bus by 7 AM, and he is often irritable during the day. The mother is exhausted and at her “wit’s end” with his nighttime and daytime behaviors, which she attributes to school-related stress. Past medical history includes a 30-week preterm birth with a relatively uncomplicated neonatal course and discharge on a home cardiorespiratory monitor at 2 months of age. Family history is positive for

paternal enuresis that resolved at age 12 and ADHD. The mother is obese, snores, has daytime fatigue, and is suspected of having OSAS but has not been diagnosed. The child's body mass index is 15 kg/m². Except for 3+ (on a scale of 1+ to 4+) tonsil size, and mouth breathing, his examination is normal.

The child underwent overnight polysomnography with thermistry as the main airflow sensor. Loud snoring was confirmed, with a respiratory disturbance index of two events per hour (most with arousal), 3% desaturation index of ten per hour, and no saturation values below 92%. The periodic limb movement index was five per hour without EEG arousals (mostly REM related). The arousal index was 12 per hour. Most of the sleep time was in the prone or side position, with no REM supine sleep time recorded. The conservative otorhinolaryngologist is reluctant to operate on a child with such minimal polysomnographic findings and looks to the pediatrician for guidance.

How should the pediatrician interpret this clinical scenario in light of the polysomnographic findings?

The boy clearly has risk factors, symptoms, and signs of OSAS (family history, history of prematurity, snoring, restlessness, enuresis, daytime neurobehavioral problems, adenotonsillar hypertrophy), but many other sleep-related issues require attention. Specifically, the clinical history suggests insufficient sleep for age, inadequate sleep hygiene, and a sleep-onset association disorder. The long-standing habitual co-sleeping may be related to increased maternal vigilance, because the child was a preterm infant on a home cardiorespiratory monitor. Insufficient sleep may be a major contributor to the child's mood and behavioral problems, independent of a diagnosis of ADHD. A child with ADHD may be even more vulnerable to the effects of insufficient sleep. Conversely, SDB also may contribute to the daytime dysfunction. The polysomnography, as performed, may have underestimated the degree of SDB because there was no REM supine time and because thermistry—rather than a nasal pressure sensor—was used to measure airflow. Even if the level of SDB is accurate, it is unclear whether habitual snoring is truly benign. At 6 years of age, 10% of children experience enuresis, and a positive family history is common in those children. Finally, ADHD and childhood OSAS are common conditions and may coexist. This former preterm infant is at increased risk for both conditions.

In the author's opinion, the optimal management approach should include (1) adenotonsillectomy with reassessment of daytime functioning and consideration of ADHD treatment if the same neurobehavioral symptoms persist, (2) increase in total sleep time (at least 10 hours) by improving sleep hygiene (television out of the bedroom) and using behavioral management strategies to help manage the bedtime resistance and night wakings (suspected sleep-onset association disorder), and (3) consideration of behavioral and conditioning management strategies for enuresis, if desired by child and family, after addressing the first and second problems.

Summary

After more than two decades of experience with increasing recognition, diagnosis, and management, many knowledge gaps must be filled. The key areas of controversy or uncertainty will continue to stimulate future research initiatives.

Future directions for research on childhood obstructive sleep apnea syndrome

- Refine prevalence estimates and risk factor data
- Perform longitudinal studies to clarify the relationship among risk factors, exposures (primary snoring or levels of SDB) and the subsequent development of OSAS and negative health outcomes
- Develop an evidence-based consensus report on measurement techniques for clinical research and care to be used in the “gold-standard” polysomnography that takes into account the newer technologies for diagnosis of OSAS in children
- Develop and evaluate less invasive, low-cost, high-sensitivity, and high-specificity diagnostic methods for OSAS in children
- Establish multicenter studies to identify the respiratory disturbance index and threshold at which OSAS treatment is indicated
- Assess the long-term efficacy of adenotonsillectomy, continuous positive airway pressure, and other OSAS treatments
- Establish multicenter studies to identify which patients are most likely to benefit from treatment and which patients are most at risk for inadequate treatment after adenotonsillectomy

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