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Diagnosis and Management of Childhood Obstructive Sleep Apnea Syndrome

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Pediatrics; originally published online August 27, 2012;
DOI: 10.1542/peds.2012-1672

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TECHNICAL REPORT

Diagnosis and Management of Childhood Obstructive Sleep Apnea Syndrome

abstract

FREE

OBJECTIVE: This technical report describes the procedures involved in developing recommendations on the management of childhood obstructive sleep apnea syndrome (OSAS).

METHODS: The literature from 1999 through 2011 was evaluated.

RESULTS AND CONCLUSIONS: A total of 3166 titles were reviewed, of which 350 provided relevant data. Most articles were level II through IV. The prevalence of OSAS ranged from 0% to 5.7%, with obesity being an independent risk factor. OSAS was associated with cardiovascular, growth, and neurobehavioral abnormalities and possibly inflammation. Most diagnostic screening tests had low sensitivity and specificity. Treatment of OSAS resulted in improvements in behavior and attention and likely improvement in cognitive abilities. Primary treatment is adenotonsillectomy (AT). Data were insufficient to recommend specific surgical techniques; however, children undergoing partial tonsillectomy should be monitored for possible recurrence of OSAS. Although OSAS improved postoperatively, the proportion of patients who had residual OSAS ranged from 13% to 29% in low-risk populations to 73% when obese children were included and stricter polysomnographic criteria were used. Nevertheless, OSAS may improve after AT even in obese children, thus supporting surgery as a reasonable initial treatment. A significant number of obese patients required intubation or continuous positive airway pressure (CPAP) postoperatively, which reinforces the need for inpatient observation. CPAP was effective in the treatment of OSAS, but adherence is a major barrier. For this reason, CPAP is not recommended as first-line therapy for OSAS when AT is an option. Intranasal steroids may ameliorate mild OSAS, but follow-up is needed. Data were insufficient to recommend rapid maxillary expansion. *Pediatrics* 2012;130:e714–e755

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KEY WORDS

adenotonsillectomy, continuous positive airway pressure, sleep-disordered breathing, snoring

ABBREVIATIONS

AAP—American Academy of Pediatrics
ADHD—attention-deficit/hyperactivity disorder
AHI—apnea hypopnea index
AT—adenotonsillectomy
BP—blood pressure
BPAP—bilevel positive airway pressure
CBCL—Child Behavior Checklist
CPAP—continuous positive airway pressure
CRP—C-reactive protein
ECG—electrocardiography
HOMA—homeostatic model assessment
HS—habitual snoring
IL—interleukin
OSAS—obstructive sleep apnea syndrome
PAP—positive airway pressure
PSG—polysomnography
PT—partial tonsillectomy
QoL—quality of life
RDI—respiratory distress index
SDB—sleep-disordered breathing
SES—socioeconomic status
Spo_2—oxygen saturation
URI—upper respiratory tract infection

(Continued on last page)

INTRODUCTION

This technical report describes in detail the procedures involved in developing the recommendations for the updated clinical practice guideline on childhood obstructive sleep apnea syndrome (OSAS).[1]

The clinical practice guideline is primarily aimed at pediatricians and other primary care clinicians (family physicians, nurse practitioners,

and physician assistants) who treat children. The secondary audience for the guideline includes sleep medicine specialists, pediatric pulmonologists, neurologists, otolaryngologists, and developmental/behavioral pediatricians.

The primary focus of the committee was on OSAS in childhood.[2] The committee focused on otherwise healthy children who had adenotonsillar hypertrophy or obesity as underlying risk factors. Complex populations, including infants <1 year of age and children who had other medical conditions (eg, craniofacial anomalies, genetic or metabolic syndromes, neuromuscular disease, laryngomalacia, sickle cell disease), were excluded because these patients will typically require subspecialty referral.

Two professional studies recently published related guidelines: the American Academy of Otolaryngology–Head and Neck Surgery[3] and the American Academy of Sleep Medicine.[4] These guidelines have similar recommendations to many of the recommendations in the American Academy of Pediatrics (AAP) guideline.

The recommendations in this statement do not indicate an exclusive course of treatment. Variations, taking into account individual circumstances, may be appropriate.

METHODS

Literature Search

A literature search was performed that included English-language articles, children and adolescents aged 1 through 17.9 years, and publication between 1999 and 2008. Animal studies, abstracts, letters, case reports, and reviews were excluded. The Medical Subject Heading terms that were used in all fields were snoring, apnea, sleep-disordered breathing (SDB), sleep-related breathing disorders, upper airway resistance, polysomnography (PSG), sleep study, adenoidectomy, tonsillectomy, continuous positive airway pressure (CPAP), obesity, adiposity, hypopnea, hypoventilation, cognition, behavior, and neuropsychology. Search engines used were PubMed, Scopus, Ovid, PsycINFO, EBSCO (including Health Source [Nursing], Child Development and Adolescent Studies), and CINAHL. Articles covering special populations (eg, infants aged <1 year, those with craniofacial anomalies or syndromes) were excluded during the title and abstract reviews.

Titles and available abstracts of articles found by the literature search were reviewed by the committee members in several rounds (see Results). In the first round, duplicates and erroneous hits from the literature search were excluded. In the second round, titles were reviewed for relevancy by 2 committee members. Articles with relevant titles were then reviewed by 2 reviewers each, on the basis of the abstract. Because of the large number of remaining articles, text-mining (Statistica, StatSoft version 9; StatSoft, Inc, Tulsa, OK) was performed on the method section of the articles to reduce the large amount of articles for the final step of quality assessment. Text-mining is the combined, automated process of analyzing unstructured, natural language text to discover information and knowledge that are typically difficult to retrieve.[5]

Unfortunately, text-mining revealed that few articles reported research methods, such as the study design (eg, clinical case series, retrospective, observational, clinical experiment), blinding of the assessment, and recruitment and/or scoring, that could have been applied for further selection. A manual screening of the questionable articles after text-mining resulted in a pool of 605 articles. The committee decided on a final round of title selection; that is, each member was assigned a random batch of articles and selected titles based on relevance with respect to the guideline categories. These remaining articles were each reviewed and graded by a committee member, as detailed here. Because of the large volume of articles requiring detailed evaluation, some committee members recruited trainees and colleagues to assist them in the performance of these reviews, under their supervision. Jason Caboot, June Chan, Mary Currie, Fiona Healy, Maureen Josephson, Sofia Konstantinopoulou, H. Madan Kumar, Roberta Leu, Darius Loghmanee, Rajeev Bhatia, Argyri Petrocheilou, Harsha Vardhan, and Colleen Walsh participated. A literature search of more recent articles (2008–2011) was performed by individual committee members, per guideline category, and discussed during the committee meeting.

As would be expected from any panel of experts in a field, some of the citations were the work of the panel members. For this reason, a varied panel, including general pediatricians, pulmonologists, otolaryngologists, and sleep medicine physicians, was arranged to provide balance. For initial guideline drafts, committee members were assigned sections of the report that were not directly in their area of research, and the evidence, search results, and conclusions thereof were discussed by all committee members at a face-to-face meeting. Subsequent drafts of the guidelines and technical report were reviewed by all committee members.

Quality Assessment

The previous literature review form[6] was modified to include the evidence grading system developed by the American Academy of Neurology for the assessment of clinical utility of diagnostic tests (Table 1).[7] A specific customized software (OSA Taskforce;

TABLE 1 Evidence Grading System[7]

Level	Description
I	Evidence provided by a prospective study in a broad spectrum of persons who have the suspected condition, by using a reference (gold) standard for case definition, in which the test is applied in a blinded fashion, and enabling the assessment of appropriate test of diagnostic accuracy. All persons undergoing the diagnostic test have the presence or absence of the disease determined. Level I studies are judged to have a low risk of bias.
II	Evidence provided by a prospective study of a narrow spectrum of persons who have the suspected condition, or a well-designed retrospective study of a broad spectrum of persons who have an established condition (by gold standard) compared with a broad spectrum of controls, in which the test is applied in a blinded evaluation, and enabling the assessment of appropriate tests of diagnostic accuracy. Level II studies are judged to have a moderate risk of bias.
III	Evidence provided by a retrospective study in which either persons who have the established condition or controls are of a narrow spectrum, and in which the reference standard, if not objective, is applied by someone other than the person who performed (interpreted) the test. Level III studies are judged to have a moderate to high risk of bias.
IV	Any study design where the test is not applied in an independent evaluation or evidence is provided by expert opinion alone or in descriptive case series without controls. There is no blinding or there may be inadequate blinding. The spectrum of persons tested may be broad or narrow. Level IV studies are judged to have a very high risk of bias.

copyright Francesco Rundo and Karen Spruyt) was developed for the literature review form to standardize this part of the process. Of note, the quality assessment levels were comparable to the grading levels applied previously.[8,9] The quality assessment applied involved 4 tiers of evidence, with level I studies being judged to have a low risk of bias and level IV studies judged to have a very high level of bias. A weaker level of evidence indicates the need to integrate greater clinical judgment when applying results to clinical decision-making. The committee's quality assessment of data took into account not only the levels of evidence in relevant articles but also the number of articles identified, the magnitude and direction of various findings, and whether articles demonstrated convergent or divergent conclusions.

The evidence-based approach to guideline development requires that the evidence in support of each key action statement be identified, appraised, and summarized and that an explicit link between evidence and recommendations be defined. Evidence-based recommendations reflect the quality of evidence and the balance of benefit and harm that is anticipated when the recommendation is followed. The AAP policy statement "Classifying Recommendations for Clinical Practice Guidelines"[10] was followed in designating levels of recommendations (Fig 1, Table 2).

RESULTS OF LITERATURE SEARCH

The automated Medical Subject Heading search resulted in 3166 hits. After duplicates and erroneous hits were excluded, 2395 hits fulfilled the criteria. After title review, 1091 articles were accepted, with a 0.70 interrater agreement between the 2 reviewers. These remaining articles were reviewed on the basis of the abstract, which resulted in 757 articles remaining, with a 0.60 agreement rate between reviewers. A final decision on those without agreement was made by the chairperson of the committee. Text-mining, although not helpful in reducing the number of articles for further evaluation, illustrated the spectrum of topics covered by the articles (Table 3). A manual screening of the questionable articles after text-mining resulted in a pool of 605 articles. The final round of title selection resulted in 397 articles for detailed review. An additional 47 articles were found to not meet criteria during the detailed review. Thus, a total of 350 articles were included.

On the basis of the final 350 articles, one-third were epidemiologic studies, 26% were diagnostic studies, and 23% were treatment studies. Table 4 lists the type of study design; 34% of studies were descriptive and 32% were nonrandomized concurrent cohort series. PSG was the diagnostic method used for 57% of the articles, whereas 45% used questionnaires. The sample size varied from 9 to 6742 subjects. Figure 2 shows the level of evidence of the articles; 76% of studies were level III or IV. The majority of studies did not include a control group, which degraded the studies to level III or IV. Few studies applied any form of blinding.

Conclusion

There has been a large increase in the number of published studies since the initial guideline was published. However, there are few randomized, blinded, controlled studies. Most articles evaluated were level III or IV, and many studies were hampered by the lack of a control group. In most studies, blinding was not present or not reported. From a methodologic standpoint, a clear need for randomized clinical trials with blinding is evident.

TERMINOLOGY

OSAS in children is defined as a "disorder of breathing during sleep characterized by prolonged partial upper airway obstruction and/or intermittent complete obstruction (obstructive apnea) that disrupts normal ventilation during sleep and normal sleep patterns,"[2] accompanied by symptoms or signs as listed in Table 2 of the accompanying guideline. In this document, the term SDB is used to encompass

both snoring and OSAS when studies did not distinguish between these entities.

PREVALENCE OF OSAS

The original clinical practice guideline found a prevalence of OSAS of 2% (3 studies) and a prevalence of habitual snoring (HS) of 3% to 12% (7 studies). Since publication of the original guideline, 10 studies (in 12 separate articles) used the gold standard of conventional overnight laboratory PSG to diagnose OSAS (Table 5). These studies were all levels I through IV, depending on the size and characteristics of the sample population, and represented many countries and age groups. They used various criteria, not all of which are standard, to diagnose OSAS. Many of the studies had a small sample size and/or studied only a selected high-risk sample of the population. Despite these limitations, the 10 studies found a prevalence of OSAS in the general pediatric population of 0% to 5.7%. Three studies to note were those of Bixler et al[11] from the United States, Li et al[12] from China, and O'Brien et al[13] from the United States. These 3 studies (levels I–II) had large sample sizes from the general pediatric population and reported OSAS prevalence rates of 1.2% to 5.7%. Six studies investigated the prevalence of OSAS by using various ambulatory studies rather than full, laboratory-based PSG (Table 6). Although the sample sizes were generally larger, home studies are not considered the gold standard of diagnosis and were thus level III. These studies found an OSAS prevalence of 0.8% to 24%. The 2 outliers (at 12% and 24%)[14,15] used more liberal criteria to diagnose OSAS. Excluding those studies, the OSAS prevalence was 0.8% to 2.8%.

Several studies attempted to discern variables associated with the presence of OSAS. Three studies found an equal prevalence between males and females,[16–18] and 2 studies found an increased prevalence in males.[12,15] Two studies reported an increased risk in children of ethnic minorities,[11,19] supporting older data.[20] Four studies found an increased risk in obese patients,[12,17,21,22] but 3 studies did

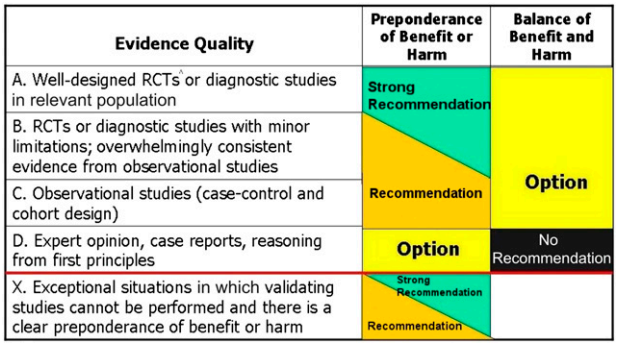

FIGURE 1
Evidence quality. Integrating evidence quality appraisal with an assessment of the anticipated balance between benefits and harms if a policy is carried out leads to designation of a policy as a strong recommendation, recommendation, option, or no recommendation. RCT, randomized controlled trial.

TABLE 2 Definitions and Recommendation Implications

Statement	Definition	Implication
Strong recommendation	A strong recommendation in favor of a particular action is made when the anticipated benefits of the recommended intervention clearly exceed the harms (as a strong recommendation against an action is made when the anticipated harms clearly exceed the benefits) and the quality of the supporting evidence is excellent. In some clearly identified circumstances, strong recommendations may be made when high-quality evidence is impossible to obtain and the anticipated benefits strongly outweigh the harms.	Clinicians should follow a strong recommendation unless a clear and compelling rationale for an alternative approach is present.
Recommendation	A recommendation in favor of a particular action is made when the anticipated benefits exceed the harms but the quality of evidence is not as strong. Again, in some clearly identified circumstances, recommendations may be made when high-quality evidence is impossible to obtain but the anticipated benefits outweigh the harms.	Clinicians would be prudent to follow a recommendation but should remain alert to new information and sensitive to patient preferences.
Option	Options define courses that may be taken when either the quality of evidence is suspect or carefully performed studies have shown little clear advantage to 1 approach over another.	Clinicians should consider the option in their decision-making, and patient preference may have a substantial role.
No recommendation	No recommendation indicates that there is a lack of pertinent published evidence and that the anticipated balance of benefits and harms is presently unclear.	Clinicians should be alert to new published evidence that clarifies the balance of benefit versus harm.

TABLE 3 Results of Text-Mining of the Methods Section of 757 Papers

Term Used for Text-Mining	Percentage of Papers
Snore/snoring	58.3
Polysomnography	53.6
Diagnosis	53.4
Medical management	51.6
Survey/questionnaire	38.8
Psychological	37.0
Surgery/surgical	35.9
Treatment	32.1
Design	27.8
Obese/obesity	25.0
BMI	24.6
Randomize	20.2
Blinding	16.4
Sampling	11.7
Control group	8.8
Actigraphy	2.6
Mortality	0.5

TABLE 4 Types of Studies in the Literature Based on 350 Articles

Type of Study	Percentage
Descriptive study	33.7
Nonrandomized concurrent cohort series	32.0
Descriptive study + other	10.8
Nonrandomized historical cohort series	7.8
Randomized clinical trial	4.6
Retrospective	3.6
Case-control study	1.3
Prospective consecutive cohort series	1.3
Cross-sectional population-based survey	1.0
Nonrandomized historical cohort series + other	1.0
Randomized + other	1.0
Undetermined	1.0
Nonrandomized concurrent cohort series + other	0.7
Experimental study	0.3

not.[15,16,23] Another study reported an increased risk of OSAS with increased waist circumference, a marker for obesity.[11] One study found an increased risk with nasal abnormalities,[11] 1 study found an increased risk with prematurity,[19] and 2 studies found increased risk with adenotonsillar hypertrophy.[12,22]

Multiple studies (levels II–IV) investigated the prevalence of HS, which is one of the most prominent manifestations of OSAS (Table 7). The presence of snoring was based on parental or personal questionnaires. Not all of the questionnaires used have been validated, and the data relied on subjective responses rather than objective clinical evaluations. The reported prevalence of HS varied widely, depending on the study and definition used, from 1.5% to 27.6%.

In summary, studies of OSAS and HS show varied prevalence rates, depending on the population studied, the methods used to measure breathing during sleep, and the definitions used for diagnosis. Nevertheless, the preponderance of evidence suggests a prevalence of OSAS in the range of 1% to 5%, making this a relatively common disease that would be encountered by most clinicians in primary practice.

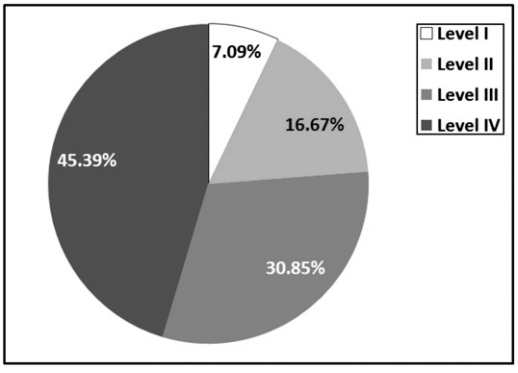

FIGURE 2
Levels of evidence of articles used for this report.

Areas for Future Research

- Population-based studies on the gender and race distribution of OSAS among different age groups.

SEQUELAE OF OSAS

Neuropsychological and Cognitive Problems Associated With OSAS

Of the 350 articles related to this search over the last 10 years, 61 articles directly explored the relationship between SDB and cognitive or neuropsychological deficits. In total, 29 658 subjects were studied, including 2 level I studies[24,25] with a total of 174 subjects and 5 level II studies.[26–30] The diagnosis of SDB was based on clinical symptoms in 29 articles and on PSG in 32 articles.

Cognitive Deficits

All but 1 study (level IV)[31] demonstrated deficits in cognition or neuropsychological function in association with symptoms, signs, or diagnosis of SDB. The 1 exception examined children who had mild OSAS over a wide age range and did not include behavioral assessments. In this study, the mean IQ in the OSAS population was significantly above the standard mean. Some[32–34] but not all studies showed a correlation between the severity of obstructive apnea as measured on PSG and increasing neuropsychological morbidity. There are several reasons why correlations were not found for all studies. Standard PSG was developed to detect cardiorespiratory variations and may not be an adequate tool for detection of sleep changes that affect neuropsychological function. Another possibility is that any degree of SDB is associated with abnormal neuropsychological outcomes and might be affected variably by social, medical, environmental, or socioeconomic factors not measured by using PSG. This

TABLE 5 Prevalence of OSAS on the Basis of Laboratory PSG

Source	Year	No.	No. Undergoing PSG	Country	Age, y	OSASPrevalence	HSPrevalence	OSAS Criteria/Comments
Anuntaseree et al[201]	2001	1005	8	Thailand	6–13	0.69%	8.5%	AHI ≥1
Anuntaseree et al[202]	2005	755	Unclear, possibly 10			1.3%	6.9%	Note: 2 studies used same cohort
							"most nights"	
Beebe et al[21]	2007	60 obese	All	United States	10–16.9	0% normal		AHI >5
		22 control				13% obese		↑ in obese
Bixler et al[11]	2009	5740	700	United States	5–12	1.2%		AHI ≥5
								↑ in ↑ waist circumference
								↑ with nasal abnormalities
								↑ in minority race
Brunetti et al[203]	2001	895	34 home monitoring	Italy	3–11	1%–1.8%	4.9%	AHI >3
Brunetti et al[23]	2010		12 PSG				5.4%	Not ↑ in obese; Note: 2 studies used same cohort
							"always"	
Li et al[172]	2010	6447	619	China	5–13	4.8%	7.2%	Using ICSD-II criteria 4.8%
Li et al[12]	2010						"frequently"	↑ in boys
								↑ in obese
								↑ in ↑ tonsil size
Ng et al[204]	2002	200	16	Hong Kong	6.4 ± 4	1%	14.5%	AHI >1
O'Brien et al[13]	2003	5728	110	United States	5–7	5.7%	11.7%	AHI >5
							"frequent and loud"	
Sogut et al[16]	2005	1198 total	28	Turkey	3–11	0.9%–1.3%	3.3%	Used AHI >3
							>3 times/week	Boys = girls
								Not ↑ in obese
Wing et al[17]	2003	46 obese,	All	China	7–15	2.3%–4.5% control;		OAI ≥1 or RDI ≥5
		44 control				26% to 32.6% obese		Boys = girls
								↑ in obese
Xu et al[22]	2008	99 obese,	All	China	Elementary school	0 if not obese and		AHI >5 or OAI >1
		99 control				no ATH		↑ obese
								↑ in ATH

ATH, adenotonsillar hypertrophy; ICSD, International Classification of Sleep Disorders; OAI, obstructive apnea index.

possibility is confirmed by a recent level I study showing that obesity, OSAS, and neurocognitive outcomes are all interdependent.[35] Furthermore, most studies were not controlled for socioeconomic status (SES), which is important because SES strongly affects the results of neurocognitive testing and because OSAS is associated with low SES.[36] Although some studies have shown abnormalities in snorers compared with nonsnoring controls, in many of these studies, data in snorers still fell within the normal range.[24] In addition, cutoffs for OSAS used in some studies resulted in a blurring of boundaries between the OSAS and snoring groups. For example, Chervin et al used an obstructive apnea index cutoff of only ≥0.5/hour to define OSAS, and the mean apnea index for the OSAS group was 2.9 events/hour, indicating that the study group had mild OSAS, which was not that different from the snorers.[37,38] A study with a wider spectrum of severity may have attained different results. Finally, most studies have not controlled for obesity, which has been associated with neurobehavioral and cognitive abnormalities.

Although most studies simply compared groups, others have looked at the correlation between polysomnographic indices and neurocognitive/behavioral outcomes and have shown a correlation between different polysomnographic factors and cognitive outcomes, behavioral outcomes, and sleepiness.[32–34,39]

Cognitive deficits associated with pediatric SDB include general intelligence level as well as processes measured by using IQ subtests (Table 8). Specific functions objectively measured by using neuropsychological assessments and included in the research studies include:

- Learning, memory, and visuospatial skills

TABLE 6 Prevalence of OSAS on the Basis of Ambulatory Monitoring

Source	Year	No.	No. Undergoing Ambulatory Monitoring	Country	Age, y	OSAS Prevalence, %	HS Prevalence	OSAS Criteria and Comments
Castronovo et al[14]	2003	595	265	Italy	3–6	12	34.5% "Often" or "Always"	OAI ≥5
Goodwin et al[15]	2005	480	All	United States	6–11	24	10.5% "Frequently"	RDI ≥1 ↑ in male Not ↑ in obese
Hultcrantz and Löfstrand Tideström[205]	2009	393	26	Sweden	12	0.8	6.9% "Regularly"	AHI ≥1 and/ or OAI ≥1
Rosen et al[19]	2003	850	All	United States	8–11	2.2		AHI ≥5 or OAI ≥1 ↑ in AA ↑ in premature infants
Sánchez-Armengol et al[18]	2001	101	All	Spain	12–16	1.9	14.8% "Often"	Based on RDI ≥10 and snoring, witnessed apneas, and/or excessive daytime sleepiness. Girls = boys
Urschitz et al[206]	2010	1144	183	Germany	7.3–12.4	2.8		AHI ≥1

OAI, obstructive apnea index; AA, African American.

TABLE 7 Prevalence of HS

Source	Year	No.	Country	Age, y	HS Prevalence, %	HS Criteria
Akcay et al[207]	2006	1784	Turkey	4–17	4.1	"Often"
Alexopoulos et al[208]	2006	1821	Greece	5–14	7.4	>3 times/wk
Archbold et al[209]	2002	1038	United States	2–13.9	17.1	"More than half of the time"
Bidad et al[167]	2006	2900	Iran	11–17	7.9	≥3 times/wk
Chng et al[210]	2004	11 114	Singapore	4–7	6.0	>3 times/wk
Corbo et al[166]	2001	2209	Italy	10–15	5.6	"Often"
Ersu et al[211]	2004	2147	Turkey	5–13	7.0	"Often"
Goodwin et al[212]	2003	1494	United States	4–11	10.5	"Snoring frequently or almost always"
Gottlieb et al[213]	2003	3019	United States	5	12	≥3 times/week
Johnson and Roth[45]	2006	1014	United States	13–16	6	"Every or nearly every night"
Kuehni et al[214]	2008	6811	United Kingdom	1–4	7.9	"Almost always"
Liu et al[215]	2005	517 in China 494 in USA	China United States	Grade school	1.5 (China) 9.9 (United States)	Snoring loudly 5–7 times/wk
Liu et al[215]	2005	5979	China	2–12	5.6	"Frequent"
Löfstrand-Tideström and Hultcrantz[216]	2007	509	Sweden	4–6	5.3–6.9	"Snoring every night"
Lu et al[217]	2003	974	Australia	2–5	10.5	≥4 times/week
Montgomery–Downs et al[44]	2003	1010	United States	Preschool	HS and risk of SDB, 22	≥3 times/week
Nelson and Kulnis[218]	2001	405	United States	6–17	17	"Often"
Ng et al[219]	2005	3047	China	6–12	10.9	6–7 times/wk
Perez-Chada et al[220]	2007	2210	Argentina	9–17	9	"Frequent"
Petry et al[221]	2008	998	Brazil	9–14	27.6	"Frequently" or "always"
Sahin et al[222]	2009	1164	Turkey	7–13	3.5	"Frequently" or "almost every day"
Sogut et al[16]	2005	1030	Turkey	12–17	4.0	"Often" or "always"
Tafur et al[223]	2009	806	Ecuador	6–12	15.1	"Often" or "always"
Urschitz et al[164]	2004	1144	Germany	Primary school	9.6	"Always" or "frequently"
Zhang et al[224]	2004	996	Australia	4–12	15.2	>4 times/wk

- Language, verbal fluency, and phonological skills
- Concept formation, analytic thinking, and verbal and nonverbal comprehension
- School performance and mathematical abilities
- Executive functions

Executive functions were measured by using both objective testing and parent questionnaires. Executive functions are a network of skills and higher order functions that control and regulate other cognitive processes. These skills require mental flexibility, impulse control,

TABLE 8 Cognitive Deficits Associated With Pediatric SDB

Type of Deficit	Source	Level	No.	Findings/Comments
Cognition, general intelligence	Beebe et al[225] Blunden et al[226] Kaemingk et al[33] Kennedy et al[34] Kurnatowski et al[227]	IV	895	Deficits of general intelligence, sensorimotor integration by objective measurement; behavioral abnormalities included as well
	Carvalho et al[228] Montgomery-Downs et al[50] Suratt et al[43]	III	1332	Objective measures of general intelligence, verbal skills affected by SDB
	Friedman et al[26] Halbower et al[28] O'Brien et al[29] Kohler et al[30]	II	473	General intelligence, executive function, language all affected by SDB and measured objectively
	O'Brien et al[24] Suratt et al[25]	I	174	General conceptual ability, verbal and nonverbal reasoning, vocabulary affected by SDB (and time in bed[25])
Poor school performance	Chervin et al[42] Johnson and Roth[45] Kaemingk et al[33] Ng et al[219] Perez-Chada et al[220] Shin et al[47] Urschitz et al[229]	IV	11 110	Academic achievement measured either by parent or school grades Additive factors were SES and ethnicity[42,45] or BMI,[42,45,47] which contributed to findings of poor school performance in SDB
	Montgomery-Downs et al[44]	III	1010	Snoring associates with ethnicity, school performance in SES-challenged preschool-aged children
Executive function	Beebe et al[225] LeBourgeois et al[230] Karpinski et al[231]	IV	179	Mental flexibility, impulse control Objective testing performed
	Halbower et al[28] Kohler et al[30]	II	123	Response preparation, working memory, fluid and quantitative reasoning; objective testing performed by blinded tester
Learning, information processing, memory, visuospatial skills	Goodwin et al[212] Hamasaki Uema et al[232] Kaemingk et al[33] Kennedy et al[34] Kurnatowski et al[227] O'Brien et al[233] Spruyt et al[234] Giordani et al[38]	IV	1838	Objective testing performed in all but Goodwin et al[212] (questionnaire)
	Halbower et al[28] Tauman et al[46]	II	112	Race[28] and BMI may play an additive role in inflammation[46] and cognitive dysfunction in SDB
	O'Brien et al[24]	I	118	Primary snoring without gas exchange abnormalities associates with significantly lower learning and memory
Language/verbal skills	Kurnatowski et al[227] O'Brien et al[233] Perez-Chada et al[220] Honaker et al[235] Lundeborg et al[51]	IV	3304	Deficits of language or verbal skills in SDB Objective testing performed in all studies
	Suratt et al[43] Montgomery-Downs et al[50]	III	114	Race and time in bed may contribute to abnormal language associated with SDB
	O'Brien et al[24] Suratt et al[25]	I	118	Primary snoring without gas exchange abnormalities associated with significantly lower verbal skills; deficits of language or verbal skills in SDB
Attention	Beebe et al[225] Chervin et al[236] Galland et al[237] Gottlieb et al[213] Hamasaki Uema et al[232] Kaemingk et al[33] Li et al[238] Mulvaney et al[32] Urschitz et al[229]	IV	6411	Objective testing performed for attention except in refs 32,33,213,229, and 236 in which parent or teacher questionnaires were used
	Chervin et al[37]	I	105	
	O'Brien et al[24]	I	118	Visual and auditory attention

and working memory. Executive functions are required for optimal school performance and are acquired through adolescence in developing children.

Behavioral Abnormalities

The investigations on the cognitive effects of SDB in the 61 studies often included measures of neurobehavioral outcomes (Table 9). Hyperactivity was the most commonly studied and/or reported behavioral abnormality associated with SDB. It was reported as a frequent symptom of SDB in younger children, and in fact, in 1 study, snoring was found to be strongly predictive of a future diagnosis of hyperactivity over the long-term (level IV).[40] Attention-deficit/hyperactivity disorder (ADHD) or ADHD symptoms, hypersomnolence, somatization, depression, atypicality, aggression, and abnormal social behaviors were the other most frequently reported behavioral abnormalities associated with SDB in children. Most behavioral difficulties were defined by using parent or teacher questionnaires in unblinded level IV studies.

Sleepiness

Two studies (levels I–II) have shown a relationship between polysomnographic measures and objective measurement of daytime sleepiness on multiple sleep latency testing.[27,39]

Exacerbation of Neuropsychological Deficits by Other Factors Underlying Childhood SDB

Abnormal behavioral alterations associated with SDB might be modified or directly caused by other sleep disorders, such as coexistent periodic limb movement disorder.[41] In children with SDB displaying deficits of cognition, school performance, or behavioral functioning, there may be additive roles played by race,[28,42–44] decreased time in bed,[25,43] and low SES,[28,42,44,45] at least in part because of the association between obesity and low SES.[42] Markers of inflammation and increased cardiovascular risk may point to 1 mechanism related to decreased cognitive function associated with OSAS,[46] seen also in children who are obese. BMI correlated with abnormal cognitive function in pediatric SDB,[42,45,47] although OSAS was found to be an independent risk factor for cognitive deficits. Finally, in 2 studies examining brain function, neuronal injury of the brain[28] and altered cerebral blood flow[48] were found in children who had SDB compared with normal controls and were associated with behavior and cognitive problems. These findings indicate the possibility of preexisting medical problems causing the development of OSAS or, alternatively, OSAS causing brain injury. Therefore, studies showing improved cognition and behavior after treatment of SDB are 1 key in the determination of causality (see the following discussion).

Neuropsychological and Cognitive Deficits in Children Who Have SDB Improve After Treatment

In the previous guideline, there were few before-and-after treatment studies of pediatric SDB focusing on objectively measured cognitive problems. In the last 10 years, 19 studies have examined changes in behavior and/or cognition after surgical treatment of OSAS. The majority of investigations demonstrated agreement about post-treatment improvement of behavior, quality of life (QoL), hyperactivity, ADHD, and impulsivity (Table 10). The exception was 1 study of exercise treatment (level IV),[49] in which snoring improved in obese children but behavior and sleepiness did not. Most studies used subjective questionnaire reports. Excessive daytime sleepiness improved in 1 study that measured this factor, as did depression, sleep quality, and aggressive behavior. Since publication of the last guideline, 3 additional studies have demonstrated improved cognitive function (by using objective measurement) after treatment of OSAS, including measures of general intelligence, attention, memory, and analytic thinking, including level II,[26] level III,[50] and level IV[37] studies (Table 10). Of concern, however, is that some recent articles suggest that certain deficits of cognition measured by using objective testing may not improve to a large extent after treatment of childhood OSAS. Language, IQ, and executive function did not improve significantly in a well-designed, controlled study of 92 children (level II).[30] General intelligence in at-risk populations improved in 1 study (level III),[50] but phonologic processes and verbal fluency did not improve to normal (level III[50] and level IV[51]). QoL increases after treatment.[37,52–58] Three studies demonstrated long-term (≥1 year) behavioral or QoL improvements.[37,52,53] The majority of these studies suggest that in developing children who are dependent on executive function, cognition, and behavioral skills for daily function and school performance, treatment of childhood SDB has benefits.

Conclusion

In summary, these studies suggest that, in developing children, early diagnosis and treatment of pediatric OSAS may improve a child's long-term cognitive and social potential and school performance. These findings imply that the earlier a child is treated for OSAS, the higher the trajectory for academic and, therefore, economic success, but research is needed to support that implication. There is demonstrated benefit in terms of behavior, attention, and social interactions, as well as likely improvement in cognitive abilities with

TABLE 9 Behavioral Abnormalities Associated With Pediatric SDB

Type of Deficit	Source	Level	No.	Test Conditions
Hyperactivity and/or ADHD	Chervin et al[236]	IV	8101	Hyperactivity generally measured by using parent questionnaire
	Chervin et al[40]			
	Galland et al[237]			
	Golan et al[239]			
	Gottlieb et al[213]			
	Johnson and Roth[45]			
	LeBourgeois et al[230]			
	Mitchell and Kelly[240]			
	Owens et al[189]			
	Roemmich et al[191]			
	Urschitz et al[229]			
	Montgomery-Downs et al[44]	III	1010	Survey data
	Chervin et al[37]	I	105	ADHD assessed by using psychiatric interview and validated instrument
Somatization, depression	Galland et al[237]	IV	205	
	Mitchell and Kelly[240]			
	Mitchell and Kelly[241]			
	Rudnick and Mitchell[242]			
	Suratt et al[43]	III	114	
	O'Brien et al[24]	I	118	
Behavior problems, general	Goldstein et al[55]	IV	1946	Behavior generally measured by using parent questionnaire
	Goldstein et al[243]			
	Hogan et al[48]			
	Li et al[238]			
	Mitchell and Kelly[241]			
	Mulvaney et al[32]			
	Owens et al[189]			
	Roemmich et al[191]			
	Rosen et al[244]			
	Rudnick and Mitchell[242]			
	Tran et al[58]			
	Wei et al[245]			
Aggression, oppositional and social problems	Chervin et al[246]	IV	4407	
	Gottlieb et al[213]			
	Galland et al[237]			
	Mitchell and Kelly[240]			
	Mulvaney et al[32]			
	O'Brien et al[24]	I	118	
Excessive daytime sleepiness	Goodwin et al[212]	IV	9729	Sleepiness measured by using questionnaire
	Perez-Chada et al[220]			
	Shin et al[47]			
	Urschitz et al[229]			
	Johnson and Roth[45]			
	Gozal et al[27]	II	92	Sleepiness measured objectively by multiple sleep latency testing on PSG
	Chervin et al[37]	I	105	Sleepiness measured objectively by multiple sleep latency testing on PSG
Anxiety	O'Brien et al[24]	I	118	

the treatment of pediatric OSAS. However, more long-term studies are needed. The risks of treatment depend on the type of treatment but include risk of surgery, risk of medication, nonadherence to therapy, and cost. The risks of not treating children who have OSAS include potentially affecting the child's trajectory of developmental gains dependent on intelligence, executive function, and proper social interactions, ultimately lowering lifetime academic and social achievements. Therefore, the benefit of treating childhood OSAS outweighs the risk where treatment is feasible.

Areas for Future Research

- Further research is required to determine which domains of cognitive function will improve with treatment of OSAS. Reversibility of cognitive deficits associated with OSAS must be adjusted for the confounding effects of age, length of symptoms, SES, BMI, sleep duration, environment, and race and ethnicity.

Cardiovascular Effects of OSAS

A total of 24 studies related to cardiovascular effects of OSAS in childhood were identified since the last review. The levels of evidence were III and IV.

In a retrospective, level IV study of 271 clinical cases, only 1 child, who had congenital heart disease, had signs of cardiac failure preoperatively, and other cases had no evidence of left or right ventricular hypertrophy.[59] However, studies using more sophisticated, prospective techniques have found subclinical evidence of cardiac dysfunction. These studies are described in Table 11. Although postoperative adenotonsillectomy (AT) cardiac complications are rare (level IV),[59] left and right ventricular hypertrophy is significantly associated with postoperative respiratory complications (level III),[60] supporting the recommendation in the current and the previous guidelines that children who have cardiac abnormalities be monitored as inpatients postoperatively.

Blood pressure (BP) has also been shown to be affected by OSAS in children. There were 9 recent level III or IV studies, most of which showed a correlation between the presence/

TABLE 10 Cognitive, Behavioral, and QoL Abnormalities Improved After Treatment of Pediatric SBD

Deficit Measured	Source	Level	No.	Abnormalities Improved After SDB Treatment
Cognition/IQ	Chervin et al[37]	I	105	Attention measured on continuous performance test improved significantly after treatment
	Montgomery-Downs et al[50]	III	38	General conceptual ability improved (verbal fluency did not improve)
	Friedman et al[26]	II	59	Auditory-visual integration, auditory-motor memory, short-term memory, retention, analytic thinking, IQ/mental processing, attention all improved
Hyperactivity and/or ADHD	Galland et al[237] Li et al[238] Mitchell and Kelly[240] Mitchell and Kelly[241] Roemmich et al[191]	IV	247	Hyperactivity and/or diagnosis of ADHD improved
	Chervin et al[37]	I	105	Long-term improvement in hyperactivity
Somatization, depression	Galland et al[237] Mitchell and Kelly[240] Mitchell and Kelly[241]	IV	153	All showed improvement in depression and/or somatization
Behavior problems, general	Goldstein et al[55]	IV	450	All showed behavior improvement except Davis et al[49]
	Goldstein et al[243] Hogan et al[48] Li et al[238] Roemmich et al[191] Tran et al[58] Wei et al[245] Mitchell et al[53] Davis et al[49]			Long-term behavior improvement in Mitchell et al[53]
Aggression, oppositional, and social problems	Galland et al[237] Mitchell and Kelly[240]	IV	113	Improvement in abnormal social behavior and aggression
Excessive daytime sleepiness	Chervin et al[37]	I	105	Sleepiness improved by 1 min, as measured by using multiple sleep latency testing on PSG
QoL	Colen et al[52]	IV	787	Includes disease-specific and emotional QoL[58]
	Constantin et al[54] Goldstein et al[55] Sohn et al[56] Silva and Leite[57] Tran et al[58]			Long-term improvements ≥1 y[52,53]
	Chervin et al[37]	I	105	Long-term improvements at 1 y
Sleep quality	Constantin et al[54] Wei et al[245]	IV	590	Improved in both studies

severity of OSAS and indices of elevated BP (Table 12).

In a study by Kaditis et al,[61] overnight changes in brain natriuretic peptide levels were large in children who had an apnea hypopnea index (AHI) ≥5/hour when compared with those with milder OSAS and with controls (level III). This finding suggests the presence of nocturnal cardiac strain in children who have moderate to severe OSAS.

Two studies evaluated brain oxygenation and cerebral artery blood flow. Khadra et al[62] reported that male gender, arousal index, and amount of non–rapid eye movement sleep were associated with diminished cerebral oxygenation, whereas increasing mean arterial pressure, age, oxygen saturation (Spo_2), and amount of rapid eye movement sleep were associated with augmented cerebral oxygenation (level III). Hogan et al[48] found a decrease in middle cerebral artery velocity postoperatively in patients treated for OSAS, whereas control subjects showed a slight increase over time (level IV).

Three studies evaluated autonomic variability in children who have OSAS. Constantin et al[63] reported resolution of tachycardia and diminished pulse rate variability after AT in children who had OSAS (diagnosis of OSAS based on oximetry plus questionnaire data) (level IV). Deng et al[64] studied heart rate variability and determined that heart rate chaos was modulated by OSAS as well as by sleep state (level IV). In a study of 28 children who had OSAS, O'Brien and Gozal[65] found evidence of altered autonomic nervous system regulation, as evidenced by increased sympathetic vascular reactivity, during wakefulness in these children (level III). These studies all suggest that OSAS places stress on the autonomic system.

In summary, a large number of studies, albeit primarily level III, found that cardiac changes occur in the presence of OSAS, with an effect on both the right and left ventricles. OSAS in childhood also has an effect on both systolic and diastolic BP. In addition, several studies suggest that childhood OSAS can affect autonomic regulation, brain oxygenation, and cerebral blood flow. These studies suggest that childhood OSAS may jeopardize long-term cardiovascular health.[66]

The association between left ventricular remodeling and 24-hour BP highlighted the role of SDB in increasing cardiovascular morbidity.

Areas for Future Research

- How reversible, after treatment, are cardiovascular changes in children who have OSAS?
- What are the long-term effects of OSAS on the cardiovascular system?

TABLE 11 Structural and Functional Cardiac Abnormalities in Children Who Have OSAS

Source	Level	No.	Findings
Left-sided cardiac dysfunction			
Amin et al[247]	III	28 OSAS 19 PS	Abnormalities of LV geometry in 39% of OSAS vs 15% of PS; OSAS associated with increased LV mass
Amin et al[248]	III	48 OSAS 15 PS	Dose-dependent decrease in LV diastolic function with increased severity of SDB
Right-sided cardiac dysfunction			
Duman et al[249]	III	21 children, ATH; 21 controls	Higher RV myocardial performance index in patient with adenotonsillar hypertrophy than in controls; this decreased significantly after AT, along with symptoms of OSAS
Ugur et al[250]	III	29 OSAS 26 PS	Improved RV diastolic function after AT, with postoperative values similar to controls
Biventricular cardiac dysfunction			
James et al[59]	IV	271	Case review of ECG and chest radiography results found only 1 case of cardiac failure, which occurred in a child who had congenital heart disease; most other cases showed no abnormalities
Weber et al[251]	III	30 OSAS 10 controls	Increased RV diameter and area during both systole and diastole; reduced LV diastolic diameter and ejection fraction

ATH, adenotonsillar hypertrophy; LV, left ventricle; PS, primary snoring; RV, right ventricle.

TABLE 12 BP in Children Who Have OSAS

Source	Level	No.	Findings
Kohyama et al[175]	IV	23 suspected OSAS	REM diastolic BP index correlated with AHI Age, BMI, and AHI were significant predictors of systolic BP index during REM
Kwok et al[66]	III	30 PS	Children with PS had increased daytime BP and reduced arterial distensibility
Leung et al[252]	III	96 suspected OSAS	Children with a higher AHI had higher wake systolic BP and sleep systolic and diastolic BP BMI, age, and desaturation index contributed to elevation of the diastolic BP during sleep, but only BMI contributed to the wake and sleeping systolic BP
Guilleminault et al[253]	III	Retrospective component: 301 suspected OSAS Prospective component: 78 OSAS	Some children who have OSAS have orthostatic hypotension
Li et al[176]	III	306 community sample	OSAS was associated with elevated daytime and nocturnal BP
Amin et al[177]	III	140 suspected OSAS	OSAS associated with an increase in morning BP surge, BP load, and 24-h BP. BP parameters predicted changes in left ventricular wall thickness
Amin et al[254]	III	39 OSAS 21 PS	OSAS was associated with 24-h BP dysregulation AHI, Spo_2, and arousal contribute to abnormal BP control independent of obesity
Enright et al[255]	III	239 community sample	Obesity, sleep efficiency, and RDI were independently associated with elevated systolic BP
Kaditis et al[174]	IV	760 community sample	No difference in morning BP between habitual snorers and nonhabitual snorers

PS, primary snoring; REM, rapid eye movement.

Growth

The section on obesity contains a detailed review of obesity and OSAS, including the relationship between OSAS and the metabolic syndrome. The previous guideline documented many studies showing a relationship between OSAS and growth, and an increase in growth parameters after treatment of SDB by AT; this outcome has been confirmed by a number of more recent studies (as discussed in the recent meta-analysis by Bonuck et al[67]). In a confirmation of previous reports,[68,69] Selimoğlu et al[70] found a decreased level of serum insulin-like growth factor-I in children who have OSAS, which increased significantly 6 months after AT (level III).

Inflammation

Since the publication of the 2002 AAP guideline, there has been growing research on the role of OSAS in systemic inflammation. It has been postulated that OSAS results in intermittent hypoxemia, leading to production of reactive oxygen species. In addition, the hypoxemia and arousals from sleep lead to sympathetic activation. These factors may trigger inflammation or exacerbate obesity-related inflammation. However, the data on OSAS and markers of systemic inflammation in children are scarce and contradictory.

Eight studies (level II–III) measured levels of C-reactive protein (CRP) in children who had OSAS. Four studies (including 2 from the same center) showed no relationship between CRP and OSAS,[71–74] whereas 4 studies (2 from the same center) did show a relationship.[46,75–77] Part of the discrepancy between studies may be attributable to the varying proportions of obese subjects (because obesity is associated with high CRP levels) and varied age of subjects and definitions of OSAS in the different studies. Some studies controlled for obesity and degree of OSAS, whereas others did not. The studies showing a positive relationship indicated that OSAS was associated with elevated

CRP levels only above a certain threshold of severity. Thus, the relationship between OSAS and CRP seems to be complex and is affected by obesity and severity of OSAS.

A few level II and III studies have evaluated other circulating markers of inflammation in children who have OSAS. Two studies showed no difference in circulating intercellular adhesion molecule-1 between patients with OSAS and controls.[71,73] A single study found elevated p-selectin (a measure of platelet activation) in children who had OSAS compared with controls.[73] A single study showed elevated levels of interferon-γ in children who had OSAS.[74] One study showed increased interleukin (IL)-6 and lower IL-10 in those with OSAS,[78] whereas another study did not.[74] Another study reported no difference in cytokines IL-1β, IL-2, IL-4, IL-8, IL-12, and granulocyte macrophage colony-stimulating factor levels between children who had OSAS and controls.[74] Data on tumor necrosis factor-α are conflicting,[74,79] and differences in levels may be related to tumor necrosis factor-α gene polymorphisms.[80]

A pathology-based study found increased glucocorticoid receptors in adenotonsillar tissue from children who had OSAS compared with tissue from children who experienced chronic throat infections (level III)[81]; another study from the same group found elevated leukotriene receptors (level IV).[82] These findings provide a theoretical construct for the potential utility of antiinflammatory drugs as treatment of children who have OSAS, although possibly not for those who have already undergone AT.

In summary, the data on CRP are conflicting, but it may be that CRP levels increase above a certain threshold of severity of OSAS. Further research involving large samples of subjects who have varying degrees of OSAS severity, with results controlled for BMI and age, are needed. There are too few data on other circulating markers of systemic inflammation to enable any recommendations.

Areas for Future Research

- Larger studies, stratified for the severity of OSAS and controlled for obesity, are required to determine whether OSAS is associated with systemic inflammation. If so, what are the long-term sequelae of this inflammation? Are inflammatory biomarkers potential good outcome measurements for OSAS treatment studies? Do they correlate with clinical outcomes or long-term prognosis?

METHODS OF DIAGNOSIS

The previous guideline discussed the diagnosis of OSAS in great detail. On the basis of published evidence at the time, it was concluded that the positive and predictive value of history and physical examination for the diagnosis of OSAS was 65% and 46%, respectively; that is, no better than chance. It was therefore recommended that objective testing be used for the diagnosis of OSAS. An evaluation of the literature regarding nocturnal pulse oximetry, video recording, nap PSG, and ambulatory PSG suggested that these methods tended to be helpful if results were positive but had a poor predictive value if results were negative. Thus, children who had negative study results should be referred for more comprehensive testing. These recommendations were based on only a few studies, most of which had a low level of evidence. Furthermore, it was recognized that these techniques were of limited use in evaluating the severity of OSAS (which is important in determining management, such as whether outpatient surgery can be performed safely). In addition, the cost efficacy of these screening techniques had not been evaluated and would depend, in part, on how many patients eventually required full PSG. Since the publication of the initial guideline, there have been a number of new studies, but few are level I or II. Because few of the studies cited here included data that would enable calculation of overall sensitivity and specificity or positive and negative predictive values, an overall table could not be provided. For this section, PSG was considered the gold standard for diagnosis of OSAS.

Utility of History Alone for the Diagnosis of OSAS

Several level IV studies evaluated the use of history alone for the diagnosis of OSAS. Preutthipan et al[83] found overall poor sensitivity and specificity when evaluating various historical factors. The Pediatric Sleep Questionnaire published by Chervin et al[84] performed slightly better than other published questionnaires, with a sensitivity of 0.85 and a specificity of 0.87 by using a set cutoff. A follow-up study by the same group showed a sensitivity of 78% and a specificity of 72% for PSG-defined OSAS.[85] However, this is still a relatively low sensitivity and specificity for clinical purposes. By using this instrument, the same group also found that negative answers to only 2 questions on the Pediatric Sleep Questionnaire were helpful in identifying patients who had normal PSG results.[86] Taken together, the overall performance of questionnaire tools seems to support their use more as a screening tool than as a diagnostic tool, such that a negative score would be unlikely to mislabel a child with OSAS as being healthy, but a positive score would be unlikely to accurately diagnose a particular child with certainty.

Utility of Clinical Evaluation for the Diagnosis of OSAS

Similar to the data presented in the previous guideline, most studies found that clinical evaluation was not predictive of OSAS on PSG. Godwin et al[15] performed a large (N = 480), population-based study of 6- to 11-year-old children. The study included use of a standardized history, some clinical parameters, and ambulatory, full PSG (level II). They concluded that the sensitivity of any individual or combined clinical symptoms was poor. Certain parameters, such as snoring, excessive daytime sleepiness, and learning problems, had a high specificity.

In a level III study, van Someren et al[87] compared history and clinical examination by a pediatrician or otolaryngologist with abbreviated PSG (video recording, oximetry, and measurement of snoring). Both the sensitivity and specificity of the clinician's impression of moderate/severe OSAS were low (59% and 73%, respectively). In a similar number of cases, the clinicians underestimated (17%) and overestimated (16%) study results.

In a level III study, it was shown that waist circumference z score had a statistically significant but clinically poor correlation with symptoms of OSAS (R = 0.32, P = .006); BMI z score did not correlate with symptoms.[88]

Radiologic Studies

Several studies, all level III or IV, evaluated the utility of radiologic examinations in addition to clinical factors in establishing the diagnosis of OSAS (Table 13). Overall, these studies showed that the presence of airway narrowing on a lateral neck radiograph increased the probability of predicting OSAS on PSG. Cephalometric studies tended to show a small mandible in patients who had OSAS compared with controls, although a study using an MRI did not confirm this.[89] None of the cephalometric studies provided sensitivity and specificity or positive and negative predictive values. Table 13 simplifies the cephalometric findings for the purpose of presentation. A level I study indicated that acoustic pharyngometry may be a useful screening technique for OSAS in older children, but approximately one-half of the children could not cooperate well with the testing.[90] One uncontrolled study (level IV) showed that nasal resistance, as measured by using rhinometry, had a high sensitivity and specificity for predicting polysomnographic OSAS.[91] This technique warrants further study and validation.

Snoring Evaluation

Two level IV studies found a weak association between objective snoring characteristics and the presence/severity of OSAS that was insufficient to assist in clinical diagnosis.[92,93]

Cardiovascular Parameters

Studies have evaluated the utility of screening tests based on heart rate or other vascular factors in predicting OSAS (Table 14). These studies ranged from studies of pulse rate alone to more sophisticated (and, hence, more expensive or time-consuming) studies, such as analyses of heart rate variability, pulse transit time, and peripheral arterial tonometry. Studies were level II through IV. Overall, the studies found changes in cardiovascular variables in children who had OSAS but with varying sensitivities and specificities. Thus, some of these measures may potentially be useful screening tests in the future if combined with other modalities that would increase the sensitivity and specificity but cannot be recommended for clinical use at this point.

Nocturnal Oximetry

The previous AAP guideline, on the basis of a single study by Brouillette et al,[94] indicated that nocturnal pulse oximetry could provide an accurate screen for OSAS if the result was positive but that full PSG was needed if the oximetry result was negative. A need for further research in this area was indicated. Four additional studies were identified for the current report. Two of these did not compare oximetry versus PSG and therefore will not be discussed further.[95,96]

A follow-up study (level II) from the same group as the previous report by Brouillette et al[94] used overnight oximetry, primarily obtained in the home, to develop a scoring algorithm.[97] The subjects' median age was 4 years. The oximetry score correlated with the AHI obtained from PSG as well as with the presence of postoperative complications. However, the positive predictive value of oximetry for major postoperative respiratory compromise was only 13%. Of note, 80% of the 223 children had normal, inconclusive, or technically unsatisfactory oximetry results and were therefore referred for either repeat oximetry or PSG. In contrast, Kirk et al[98] compared overnight home oximetry (by using a system with an automated oximetry analysis algorithm that provided a desaturation index) with laboratory PSG in 58 children aged ≥4 years who had suspected OSAS (level III). They found poor agreement between the desaturation index on the basis of oximetry and the PSG-determined AHI. The sensitivity of oximetry for the identification of moderate OSAS (AHI >5/hour) was 67%, and specificity was 60%. The oximetry algorithm tended to overestimate the AHI at low levels and underestimate at high

TABLE 13 Relationship Between Airway Measurements and OSAS

Clinical Evaluation	Sleep Evaluation	Airway Evaluation	Source	Level	No.	Findings
Standardized history, clinical examination	PSG	Lateral neck radiography	Xu et al[256]	IV	50	Combinations of different predictor variables resulted in positive and negative predictor values ranging from 70% to 80%
Clinical examination	PSG	Lateral neck radiography	Jain and Sahni[257]	IV	40	Degree of OSAS correlated with adenoid size on radiography but not with tonsillar size on clinical examination
Clinical examination	PSG	Lateral neck radiography	Li et al[258]	IV	35	Tonsillar–pharyngeal ratio on radiography correlated with AHI but not clinical tonsil size. Clinical tonsil size did not correlate with AHI For a ratio of 0.479, the sensitivity and specificity in predicting moderately severe OSAS (AHI >10/h) was 96% and 82%, respectively
NA	PSG	Cephalometry	Kawashima et al[259]	III	15 OSAS 30 controls	Evidence of retrognathia in OSAS group
Clinical examination	Ambulatory abbreviated recordings	Cephalometry	Kawashima et al[260]	III	38 OSAS 31 controls	OSAS: retrognathia, long facies in those OSAS subjects who had large tonsils
NA	None	Cephalometry	Kikuchi et al[261]	IV	29 suspected OSAS 41 controls	OSAS: long facies
Questionnaire	None	Cephalometry	Kulnis et al[262]	IV	28 snorers 28 controls	Snorers: retrognathia, shorter maxilla and cranial base
Standardized history	Nap PSG	Cephalometry	Zucconi et al[263]	III	26 snorers 26 controls	Snorers: retrognathia, decreased nasopharyngeal space
NA	PSG	MRI	Schiffman et al[89]	III	24 OSAS 24 controls	No difference in mandibular size between OSAS and controls
Clinical assessment of tonsillar size	Ambulatory cardiorespiratory recordings	Acoustic pharyngometry Cephalometry	Monahan et al[90]	I	203	Degree of OSAS correlated with airway size on pharyngometry but not with tonsillar size Pharyngometric measures also correlated with mandibular length on cephalometry, only 78% of 8- to 11-y-old children could produce minimally acceptable data, and only 54% could produce high-quality data
Questionnaire, clinical examination	PSG	Rhinometry	Rizzi et al[91]	IV	73	Nasal resistance of 0.59 Pa/cm^3/s had a positive predictive value of 97% and a negative predictive value of 86%

levels. The authors concluded that oximetry alone was not adequate for the diagnosis of OSAS. On the basis of these limited studies, it seems as if oximetry alone is insufficient for the diagnosis of OSAS because of the high rate of inconclusive test results and the poor sensitivity and specificity compared with PSG, probably, in part, because children may have OSAS that results in arousals and sleep fragmentation but little desaturation. In addition, children tend to move a lot during sleep, which can result in movement artifact.

Ambulatory PSG

The term "ambulatory PSG" is used for unattended sleep studies conducted in the home. Frequently, ambulatory PSG consists of cardiorespiratory recordings alone. Although the use of ambulatory PSG is considered appropriate under certain circumstances in adults,[99] there is a paucity of studies evaluating ambulatory PSG in children. Zucconi et al[100] evaluated a home portable system comprising measurements of airflow (by using thermistry), snoring, chest and abdominal wall movements, electrocardiography (ECG), position, and oximetry (level II). However, the portable system was used in the sleep laboratory for the purpose of the study. A small sample of 12 children, 3 to 6 years of age, underwent routine PSG and in-laboratory portable testing on a consecutive night with the portable system. The portable system had good sensitivity for detecting a respiratory distress index (RDI) >5/hour (78% with automated scoring; 89% with human scoring) but a specificity of zero. Rosen et al[19] reported on a study of 664 children aged 8 to 11 years who underwent abbreviated ambulatory study (by using inductance plethysmography, oximetry, heart rate, and position) (level III). Of these home studies, 94% were considered technically adequate. A subsample of 55 children also underwent full laboratory PSG. Few details were given regarding this subsample. However, it was reported that the ambulatory studies had a sensitivity

TABLE 14 Utility of Cardiovascular Parameters in Predicting OSAS

Measure	Sleep Evaluation	Source	Level	No.	Findings
Pulse rate	Oximetry	Constantin et al[63]	IV	25 OSAS	Pulse rate decreases in children who have OSAS after AT
Pulse rate	Home cardiorespiratory studies	Noehren et al[264]	III	5 OSAS 20 controls	Pulse rate changes poor at detecting differences between respiratory events and movements, and between central and obstructive apneas
Heart rate variability	PSG	Deng et al[64]	IV	34 OSAS 18 controls	Heart rate chaos intensity had sensitivity of 72% and specificity of 81% for OSAS
Pulse transit time	PSG	Katz et al[265]	III	24 SDB 10 controls	Depending on the severity of the event, 80%–91% of obstructive respiratory events were associated with pulse transit time changes. However, pulse transit time changes also occurred with spontaneous arousals from sleep
Heart rate, pulse transit time	PSG	Foo et al[266] (similar data published in Foo and Lim[267])	III	15 suspected OSAS	Pulse rate had 70% sensitivity and 89% specificity, and pulse transit time had 75% sensitivity and 92% specificity in identifying obstructive events
Peripheral arterial tonometry	PSG	Tauman et al[268]	II	40 OSAS 20 controls	Peripheral arterial tonometry had sensitivity of 95% and specificity of 35% in identifying EEG arousals

of 88% and specificity of 98% in diagnosing a laboratory PSG–based AHI >5/hour. It is not clear why the results of this study were so different from that of Zucconi et al but may possibly be related to the older age of the subjects. Goodwin et al[101] used a full PSG system, including EEG measurements, in the unattended home environment in 157 children aged 5 to 12 years (level IV). Adequate data were obtained from 91% of subjects on the first attempt and 97% when the test was repeated if needed. Data were reported as excellent in 61% of cases and good in 36%. In a small subsample of 5 subjects, data were similar to those with laboratory PSG. This study shows the feasibility of performing unattended full ambulatory PSG in older children, but results may not be the same for young children. In summary, ambulatory PSG seems to be technically feasible in school-aged children, although data are not available for younger children. Studies of differing levels, and studying different age groups, found widely discrepant specificities for diagnosing moderate OSAS. Clearly, additional studies are needed.

Nocturnal PSG

Nocturnal, attended, laboratory PSG is considered the gold standard for diagnosis of OSAS because it provides an objective, quantitative evaluation of disturbances in respiratory and sleep patterns. A recent review describes some of the relationships between PSG and sequelae of OSAS (see "Pediatric Issues" section in Redline et al[102]). PSG allows patients to be stratified in terms of severity, which helps determine which children are at risk for sequelae (thus alerting pediatricians to screen for complications of OSAS); which children are at risk for postoperative complications and would, therefore, benefit from inpatient observation postoperatively; and which children are at high risk of persistence of OSAS postoperatively, who may then need postoperative PSG to assess the need for further treatment (eg, CPAP).

Adult patients may sleep poorly the first time they are in a sleep laboratory because of anxiety, the unfamiliar environment, and the attached sensors. This "first night effect" can lead to altered sleep architecture and possible underestimation of the severity of OSAS. Five studies (levels I–IV) evaluated the night-to-night variability of PSG in children[101,103–106]; in one of these articles,[101] only a small subsample had night-to-night variability evaluated (Table 15). The time difference between PSGs varied from 24 hours to 4 weeks. Although some of the studies showed minor differences in respiratory parameters from night to night, the studies suggest that few children would have been clinically misclassified on the basis of a single night's PSG. Thus, 1 night of PSG seems to be adequate to establish the diagnosis of OSAS. All studies showed significant differences in sleep architecture from night to night. Therefore, research studies evaluating sleep architecture would require >1 night of PSG. For consistency, it is recommended that PSG be performed and scored by using the pediatric criteria from the American Academy of Sleep Medicine scoring manual.[107]

Other Tests

The shape of the maximal flow-volume loop on pulmonary function testing has been used to attempt to screen for OSAS in adults. Young children cannot perform standard maximal flow-volume loops. One small study of 10 subjects evaluated the relationship between tidal breathing flow-volume loops and PSG (level III).[108] The sensitivity was 37.5% and specificity was 100%, indicating that this method is of limited utility in screening for OSAS.

Two studies by the same group evaluated whether urinary/serum

TABLE 15 Night-to-Night Variability in Polysomnographic Respiratory Parameters

Time Between Evaluations	Source	Level	No.	Findings
1–4 wk	Katz et al[103]	I	30 suspected OSAS	No significant group difference in the AHI between nights. Those with the highest AHI had the most variability. However, no patient was reclassified as primary snoring versus OSAS on the basis of the second study
7–50 d	Goodwin et al[101]	IV	12	Used unattended home PSG. Studies were successful in 10. No difference in AHI between nights in this small sample
Consecutive nights	Scholle et al[105]	III	131 OSAS	No difference in AHI between nights
Consecutive nights	Li et al[104]	III	46 obese 44 controls	AHI was greater on night 2 The first night would have correctly identified 11 (85%) of the 13 cases of OSAS if the worst obstructive apnea index over any single night was used as the criterion. However, the 2 cases that would have been missed by the single PSG had only borderline OSAS
Consecutive nights	Verhulst et al [106]	I	70 suspected OSAS	First night classified OSAS correctly in 91% of subjects, if the worst AHI over any night was used as the diagnostic criterion. All but 1 of those who were missed had an AHI <5/h

proteinomic analysis could be used to screen for the presence of OSAS. In a level I study of urinary proteinomics, the investigators found that a combination of urinary proteins could predict OSAS with a sensitivity of 95% and a specificity of 100%.[109] Similarly, in a level III study from the same group, the investigators found that a different set of proteins could be used to identify 15 of 20 children who had OSAS and 18 of 20 children who were snorers.[110] The authors note that they studied a highly selected population matched for age, gender, ethnicity, BMI, and inflammatory respiratory disorders, such as allergic rhinitis or asthma. Thus, this technique, although promising, requires further validation in typical clinical cohorts and duplication in another laboratory.

Summary

In summary, few of the screening techniques mentioned here have a sensitivity and specificity high enough to be relied on for clinical diagnosis. In addition, it should be noted that many of the studies used an AHI >5/hour when determining sensitivity and specificity, although an AHI >1.5/hour is considered statistically abnormal in children.[111–113] Few studies used large study samples, and few were blinded. As a result, some of the studies of screening techniques resulted in contradictory evidence. On a pragmatic level, however, it is realized that current infrastructure is inadequate to provide PSG for all children with suspected OSAS. Therefore, the use of screening tests may be better than no objective testing at all. However, clinicians using these tests should familiarize themselves with the sensitivity and specificity of the test used and consider proceeding to full PSG if the test result is inconclusive.

Areas for Future Research

- Well-designed, large, controlled, blinded, multicenter, prospective studies are required to provide more definitive answers regarding the utility of screening tests for the diagnosis of OSAS. In particular, additional studies of ambulatory PSG in children of varying ages are needed.

TREATMENT OF OSAS

AT

Adenotonsillar hypertrophy is the most common cause of OSAS, and AT continues to be the primary treatment for this issue. Adenoidectomy alone may not be sufficient for children who have OSAS because it does not address oropharyngeal obstruction secondary to tonsillar hyperplasia. The previous guideline stated the importance of AT as the primary treatment for OSAS in children. No new literature is available to suggest a change to these recommendations. Table 3 in the guideline lists relative contraindications to AT. Note that whereas a submucus cleft palate is a relative contraindication to adenoidectomy, a partial adenoidectomy may be performed in such patients. However, postoperative PSG should be performed to ensure that OSAS has resolved.

AT in most children is associated with a low complication rate. Minor complications include pain and poor oral intake. More severe complications may include bleeding, infection, anesthetic complications, respiratory decompensation, velopharyngeal incompetence, subglottic stenosis, and, rarely, death.

Tarasiuk et al found that health care utilization costs were 226% higher in children with OSAS before diagnosis compared with control children[114] and that health care costs decreased by one-third in children who underwent AT, whereas there was no change in health care costs in control children or children who had untreated OSAS[115] (both studies were level IV).

Partial Tonsillectomy

Several newer techniques for tonsillectomy have gained increasing use since publication of the last guideline. The primary goal of these techniques

is to decrease the morbidity associated with traditional tonsillectomy methods. One such technique is partial tonsillectomy (PT), in which a portion of tonsil tissue is left to cover the musculature of the tonsillar fossa. Multiple studies, ranging in level from II to IV, have evaluated recovery times and adverse effects from PT. However, only a few small, lower-level studies have specifically looked at the effect of PT on OSAS. In a level IV study, Tunkel et al[116] evaluated 14 children who underwent PSG before and after PT and found a cure rate (AHI $\leq$1/hour) of 93% postoperatively. In a retrospective study (level IV), Mangiardi et al[117] compared 15 children who underwent PT (of 45 eligible) with 15 children who underwent total tonsillectomy. This study had a number of technical limitations. A variety of techniques (overnight laboratory PSG, nap sleep studies, and limited-channel home sleep studies) were performed in subjects preoperatively, and limited-channel home sleep studies were performed in all patients postoperatively. These different monitoring techniques would be expected to provide varying results.[118,119] In both surgical groups, the authors found a higher rate of postoperative OSAS than typically reported in the literature, with a median (range) AHI of 7.5 $\pm$ 4.3/hour in the PT group and 8.8 $\pm$ 4.7/hour in the total tonsillectomy group (not significant).

PT carries an increased risk of regrowth of the tonsils, which occurred in 0.5% to 16% of patients in studies of varied duration. Celenk et al[120] performed a retrospective review of 42 children 1 to 10 years of age who underwent PT via radiofrequency ablation for symptoms of OSAS (level IV). Follow-up ranged from 6 to 32 months, with a mean follow-up of 14 months. They found tonsillar regrowth on physical examination in 7 (16.6%) patients; 5 of these were symptomatic and underwent completion tonsillectomy. The time frame for occurrence of regrowth ranged from 1 to 18 months. The authors noted that some episodes of regrowth occurred after episodes of tonsillitis. Zagólski[121] evaluated 374 children who underwent PT on the basis of clinical symptoms of OSAS (level IV). Patients underwent otolaryngology examinations annually for 4 years. Twenty-seven (7.2%) children had tonsillar regrowth; of those, 20 had clinical symptoms and, therefore, underwent completion tonsillectomy. Regrowth of the palatine tonsils was observed at a mean period of 3.8 years, suggesting the need for long-term follow-up. In a multicenter, retrospective case series of 870 children with a mean follow-up of 1.2 years, Solares et al[122] found an incidence of tonsillar regrowth of 0.5% (level III). The methods and criteria for assessing regrowth were not detailed in this article but may have been a clinical follow-up at 1 and 6 months postoperatively. The lower rate of regrowth in this study compared with the other studies may have been related to the shorter follow-up period. Eviatar et al[123] performed a long-term (10–14 years), retrospective, telephone survey comparing 33 children who had undergone PT for symptoms of OSAS versus 16 children who underwent tonsillectomy; children undergoing concomitant adenoidectomy were excluded (level III). They found similar rates of parent-reported snoring in the 2 groups (6.1% for PT, 12.5% for total tonsillectomy; not significant) but no cases of OSAS on the basis of symptoms.

PT for the treatment of adenotonsillar hypertrophy has shown some success in decreasing immediate postoperative pain. Derkay et al[124] prospectively evaluated 300 children undergoing either PT or total tonsillectomy for adenotonsillar hypertrophy (level II). They found that children in the PT group had an earlier return to normal activity and were 3 times more likely not to need pain medication at 3 days compared with the total tonsillectomy group. There was no difference between groups in median return to a normal diet (3.0 vs 3.5 days). In a level III, retrospective study of 243 children undergoing PT versus 107 undergoing total tonsillectomy, Koltai et al[125] found less pain and quicker return to a normal diet in children undergoing PT. In a level II study, Sobol et al[126] prospectively evaluated 74 children who had adenotonsillar hypertrophy scheduled for AT. Their results showed a resumption to normal diet 1.7 days earlier in the PT group compared with children undergoing total tonsillectomy. There was no significant difference in the resolution of pain or return to normal activities between the 2 groups, but there was increased intraoperative blood loss in the PT group.

In summary, there are no level I studies comparing PT with total tonsillectomy in the pediatric population. Additional data are needed regarding the efficacy of PT for OSAS, by using objective outcome measurements. There is possibility of tonsillar regrowth after PT, with studies showing varied rates of regrowth. These studies are all limited by lack of blinding, lack of objective measures to quantitate tonsillar regrowth, and lack of polysomnographic data relating tonsillar regrowth to OSAS. Some studies found that patients who undergo PT have less pain and quicker recovery during the first few days compared with children undergoing total tonsillectomy. However, PT may be associated with greater intraoperative blood loss, and there is a risk of recurrent infections in the tonsillar remnants.[120,121,123] At

this point, data are insufficient to recommend any particular surgical technique for tonsillectomy over another in terms of OSAS. However, children undergoing PT should be monitored carefully long-term to ensure that symptoms of OSAS related to tonsillar regrowth do not occur, and families should be warned about the possibility of recurrence of OSAS.

Postoperative Management After AT

Tonsillectomy and adenoidectomy can be safely performed in the vast majority of children on an outpatient basis. Risk factors that increase the risk of postoperative complications include age <3 years, severe OSAS, presence of cardiac complications, failure to thrive, obesity, and presence of upper respiratory tract infection (URI). Although there have been numerous publications regarding postoperative complications since publication of the last guideline, there have been no data to suggest a change in the previous recommendations. Children with medical comorbidities such as craniofacial anomalies, genetic syndromes, and neuromuscular disease are also high risk; these special populations are not covered by this guideline.

An important advantage of the objective documentation of the severity of OSAS by using PSG should be the ability to predict the need for overnight hospital stay after AT on the basis of a higher risk of postoperative complications. Severe OSAS has been proposed as a criterion for inpatient observation; the current evidence to define severe OSAS is derived primarily from level III retrospective studies. Although considerable physiologic information regarding the respiratory pattern and gas exchange during sleep is available from an overnight PSG, the available studies have focused primarily on the AHI and, to a lesser degree, the nadir of the Sp_{O_2}. Relevant studies are listed in Table 16. Studies varied with regard to the type of patients included (proportion of obese patients; patients who had craniofacial and genetic syndromes) and severity of OSAS. Although the definition of postoperative respiratory compromise varied, most studies required that an intervention (eg, supplemental oxygen, nasopharyngeal tube, CPAP, intubation) be performed. Most studies found a high rate of postoperative respiratory complications. Different studies showed different PSG predictive factors for postoperative complications, and few studies developed receiver operating characteristic curves.[127] Nevertheless, studies were fairly consistent in indicating that an Sp_{O_2} <80% and an AHI >24/hour were predictive of postoperative respiratory compromise. These criteria are more conservative than the recently published clinical practice guidelines from the American Academy of Otolaryngology–Head and Neck Surgery, which recommend that children who have an AHI ≥10/hour and/or an Sp_{O_2} nadir <80% be admitted for overnight observation after AT.[3]

It is difficult to provide exact PSG criteria for OSAS severity because these criteria will vary depending on the age of the child; additional comorbidities, such as obesity, asthma, or cardiac complications of OSAS; and other PSG criteria that have not been evaluated in the literature, such as the level of hypercapnia and the frequency of desaturation (compared with Sp_{O_2} nadir). Therefore, on the basis of published studies (Table 16), it is recommended that patients who have an Sp_{O_2} nadir <80% (either on preoperative PSG or during observation in the recovery room postoperatively) or an AHI ≥24/hour be observed as inpatients postoperatively because they are at increased risk of postoperative respiratory compromise. In addition, on the basis of expert consensus, it is recommended that patients with significant hypercapnia on PSG (peak P_{CO_2} ≥60 mm Hg) be admitted postoperatively. Clinicians may decide to admit patients who have less severe PSG abnormalities on the basis of a constellation of risk factors (age, comorbidities, and additional PSG factors) on an individual basis.

Data regarding URIs were based on studies of children undergoing general anesthesia for a variety of procedures. The committee could not identify any studies related specifically to URIs and AT. In a large, level III study, Tait et al[128] evaluated 1078 children 1 month to 18 years of age who were undergoing an elective surgical procedure. The presence of a URI was diagnosed by using a parental questionnaire. Data regarding perioperative respiratory events were recorded. There were no differences between children who had active URIs, recent URIs (within 4 weeks), and asymptomatic children with respect to the incidences of laryngospasm and bronchospasm. However, children who had active and recent URIs had significantly more episodes of breath-holding, desaturation <90%, and overall adverse respiratory events than children who had no URIs. Independent risk factors for the development of adverse respiratory events in children who had active URIs included use of an endotracheal tube (in those <5 years of age), preterm birth, history of reactive airway disease, paternal smoking, surgery involving the airway, the presence of copious secretions, and nasal congestion. In a large level III study of 831 children undergoing surgery with a laryngeal mask airway, von Ungern-Sternberg et al[129]

TABLE 16 Relationship Between PSG Parameters and Postoperative Respiratory Complications

Source	Level	Type of Study	No.	Study Group	Age, y	Special Populations Included[a]	Findings
Hill et al[269]	III	Retrospective	83	AHI >10	≤18	Yes	Major respiratory complication in 5%; minor in 20% Only age <2 y ($P < .01$) and AHI >24 ($P < .05$) significantly predicted postoperative airway complications Complication rate only 4% if special populations were excluded AHI >24 predicted 63% of complications
Jaryszak et al[270]	III	Retrospective	151	Any child who had a PSG	Not stated	Yes	Respiratory complication rate was 15% Children with complications had higher AHI (32 vs 14) and lower Spo_2 nadir (72% vs 84%) compared with those without complications
Koomson et al[271]	III	Retrospective	85	AHI >5	Not stated	Yes	Postoperative desaturation in 28% More likely to desaturate postoperatively if PSG Spo_2 nadir <80%
Ma et al[272]	III	Retrospective	86	Any child who had a PSG	1–16	Yes	Postoperative desaturation in 7% No difference in AHI between those with and without postoperative desaturation (11.6 ± 4.5 vs 14.7 ± 16.6)
Sanders et al[273]	I	Prospective	61	61 children who had OSAS vs 21 who had tonsillitis	2–16	No	Respiratory complication rate was 28% Subjects with RDI ≥30 were more likely to have laryngospasm and desaturation At an RDI ≥20, OSAS was more likely to have breath-holding on induction
Schroeder et al[274]	III	Retrospective	53	Severe OSAS (AHI >25)	Not stated	Yes	43% required oxygen or PAP Note: an additional 17 children were electively kept intubated postoperatively
Shine et al[196]	III	Retrospective	26	Obese OSAS	2–17	Obese; other comorbidities not stated	46% had respiratory complications Those requiring intervention for respiratory problems had a lower Spo_2 (68 ± 20% vs 87 ± 18%) but no difference in RDI (27 ± 44 vs 15 ± 28) than those who did not require intervention By using univariate analysis, a preoperative Spo_2 <70% was associated with postoperative respiratory compromise, but no threshold was found for RDI
Ye et al [127]	III	Retrospective	327	AHI ≥5	4–14	No	11% had respiratory complications An AHI of 26 had 74% sensitivity and 92% specificity for predicting postoperative respiratory complications

[a] Special populations include children with genetic syndromes and craniofacial abnormalities.

compared children who had a URI within 2 weeks of surgery versus those without a URI; 27% of children had a recent URI. They found a doubling of the incidence of laryngospasm, bronchospasm, and oxygen desaturation intraoperatively and in the recovery room in the children who had recent URIs, although the overall incidence of these events was low. The risk was highest in young children; those undergoing ear, nose, and throat surgery; and those in whom multiple attempts were made to insert the laryngeal mask airway. On the basis of data available regarding risk with general anesthesia, the committee concluded that children who have an acute respiratory infection on the day of surgery, as documented by fever, cough, and/or wheezing, are at increased risk for postoperative complications and, therefore, should be rescheduled or monitored closely postoperatively. Clinicians should decide on an individual basis whether these patients should be rescheduled, taking into consideration the severity of OSAS in the particular patient and keeping in mind that many children who have adenotonsillar hypertrophy exhibit chronic rhinorrhea and nasal congestion even in the absence of viral infections.

Postoperative Persistence of OSAS After AT

Although the majority of children have a marked improvement in OSAS after AT, OSAS may persist postoperatively. OSAS is especially likely to persist in children who have underlying illnesses such as craniofacial anomalies, Down syndrome, and neuromuscular disease; these special populations are not included in this review.

Over the years since the committee's first consensus report, a number of studies have been published discussing the impact of surgery on childhood OSAS. Most of these studies were omitted from consideration for

this review because of their lack of preoperative and postoperative PSGs. Many other studies reported changes in group averages for polysomnographic and other measures postoperatively. All published articles found that AT leads to significant improvement in polysomnographic parameters in the majority of patients (although not in all). Studies providing data that could be interpreted to provide an estimate of the proportion of patients who were cured of their OSAS are shown in Table 17. Twenty original articles on the topic have been published since 2002, including 2 meta-analyses[130,131] of other articles included in the review. The lack of uniform agreement regarding the polysomnographic criteria for diagnosis of OSAS complicates this analysis of postoperative persistence of OSAS, as it does other aspects of this review, in part because the preoperative PSG criteria for surgery are not uniform across the different articles, but more importantly, because the postoperative prevalence of OSAS is highly dependent on the stringency of diagnostic criteria. In some cases, articles helpfully provided data on residual prevalence of OSAS by using different polysomnographic criteria (eg, AHI >1/hour and AHI >5/hour). At this point, it is generally accepted that AT has a higher success rate than isolated adenoidectomy or tonsillectomy, so although a few of the articles included some patients undergoing only adenoidectomy, only tonsillectomy, or ancillary procedures such as nasal turbinectomy, most focused exclusively on the impact of AT.

As shown in Table 17, a total of 11 articles were published, describing 10 general population cohorts referred either to a pediatric sleep specialist or otolaryngologist for OSAS, and 1 meta-analysis of articles dating back to 1980. Most of these were case series of patients, with significant methodologic flaws, including nonblinding and incomplete follow-up for a high proportion of patients, and these issues were present even in the methodologically strongest articles.[132–134] The polysomnographic criteria for OSAS in each article may or may not have been the same as those used as an indication for AT, and these varied from an AHI ≥1/hour to AHI ≥5/hour and RDI >2 to 5/hour. Surprisingly, the overall estimate of postoperative persistence of OSAS did not seem to vary greatly by polysomnographic criteria for surgery. Conversely, the estimates of residual OSAS were clearly related to which polysomnographic criteria for OSAS were applied to the postoperative PSGs. When using an AHI ≥1/hour as the criterion for residual OSAS, estimates of persistence ranged from 19%[135] to 73%,[133] whereas when using an AHI ≥5/hour as the criterion, the estimate of persistence of OSAS ranged from 13%[134] to 29%.[132] It is important to recognize that there are clearly recognizable risk factors for postoperative persistence of OSAS and that the prevalence of these risk factors in the populations studied had an important impact on their estimates of postoperative persistence of OSAS. For example, >50% of patients in the multicenter study of Bhattacharjee et al[133] were obese, whereas 21% of the patients in the series by Ye et al[134] were obese, defined as 95th percentile for the Chinese population. It should be emphasized that although many of these studies showed a high proportion of patients with residual OSAS after AT, most patients exhibited a marked decrease in AHI postoperatively.

Risk Factors for Postoperative OSAS

1. Obesity

Five studies focused attention on obese patients (defined as 95th percentile for weight or BMI for age), and 1 meta-analysis[131] combined 4 of these studies. The meta-analysis reported that 88% of obese patients still had a postoperative AHI ≥1/hour, 75% had a postoperative AHI ≥2/hour, and 51% had a postoperative AHI ≥5/hour. Preoperative obesity was found to be a significant risk factor for postoperative residual OSAS in several other studies[133–135] as well, even when multivariable modeling was used to control for other factors such as age and preoperative AHI. The odds ratios of persistent OSAS in obese patients ranged in these models from 3.2[134] to 4.7.[136] One study found that the relationship of BMI to risk of persistent OSAS was no longer significant when adjusted for preoperative AHI.[137] In contrast to all of the studies that looked at this factor, a study of obese Greek children found no difference in the prevalence of residual OSAS in obese versus nonobese children; part of the reason for this finding might be that this study used a slightly less stringent criterion for obesity (1.645 SDs weight for age, which is the 90th percentile).[138]

2. Baseline Severity of OSAS

All studies that evaluated baseline AHI as a potential risk factor for persistent postoperative OSAS found it to be a significant risk factor, even when adjusted for other comorbidities such as obesity.[132–134,136,139]

3. Age

A series limited to children aged <3 years reported a high incidence (65%) of treatment failures in these younger children, but this cohort included a large proportion of children who have other risk factors, such as severe OSAS and chromosomal and craniofacial abnormalities.[140] In contrast, 2 studies reported that increasing age (especially 7 years and older) is a risk factor for persistent

TABLE 17 Studies Providing an Estimate of the Proportion of Patients Who Were Cured of OSAS With Surgery

Source	Year	Level	No.	Age, y	Population	Polysomnographic Criterion for Surgery	Operation	Follow-up Period, mo	Subjects Who Had OSAS at Follow-up	Miscellaneous
General population studies										
Chervin et al[37] Dillon et al[275]	2006	I	39	5.0–12.9		AHI ≥1	AT	13 ± 1.4	21%	2 articles documented findings in the same population
Guilleminault et al[135]	2004	III	56	1.25–12.5		AHI ≥1 or RDI >2	AT: 36 (some of whom also had nasal turbinectomy and/or tonsillar wound suturing); A: 8; T: 11	3	AT: 19.4%; A: 100%; T: 100%	Half of AT failures were in obese patients
Guilleminault et al[141]	2007	III	199	1.5–14		AHI ≥1	AT in 183; A or T in 19; nasal turbinectomy in 17.4%	3–5	46.2%	Increased nasal turbinate score, presence of deviated nasal septum and increased Mallampati score of relationship of tongue to uvula and retro position of the mandible were all predictive of higher failure rate
Guilleminault et al[276]	2004	IV	284	2–12.1		AHI >1.5	AT in 228; A or T inferior turbinectomy in 73	3–4	8.8% of those with preoperative AHI <10 and AT; 64.7% of those with preoperative AHI ≥10. No breakdown provided regarding results of AT versus other surgery	An additional 99 children had RDI >1.5 and AHI <1.5. Of this group, 100% had normal RDI after AT and 9.2% had residual abnormal RDI after A or T. Difficult to interpret findings because of inconsistent reporting of data
Mitchell[132]	2007	III	79	3–14		AHI ≥5	AT	1–9.3	16% (AHI ≥5); 29% (AHI >1.5)	Severity of preoperative AHI predicted response: preoperative 5–10, 0% ≥5; preoperative 10–20, postoperative 12% ≥5; preoperative >20, postoperative 36% ≥5; 13/22 with postoperative snoring had AHI ≥5; 0/57 without postoperative snoring had AHI ≥5
Tal et al[277]	2003	IV	36	1.8–12.6		RDI >1	AT	4.6 (1–16)	11.11% had RDI >5	
Tauman et al[137]	2006	III	110	6.4 ± 3.9		AHI ≥1	AT	1–15	46% AHI 1–5, 29% with AHI >5	In logistic regression, AHI before surgery and family history of OSAS were significant predictors of AHI >5 postoperative
Walker et al[278]	2008	IV	34	0.93–5		RDI >5 in REM sleep	AT	9.8	35% with RDI >5	Treatment failures limited to those with preoperative RDI in REM >30
Bhattacharjee et al[133]	2010	III	578	6.9 ± 3.8		AHI ≥1	AT	1–24	72.8% with AHI ≥1; 21.6% >5	Large multicenter study. Age >7 y, increased BMI, presence of asthma, and high preoperative AHI were independent predictors of persistent postoperative OSAS

TABLE 17 Continued

Source	Year	Level	No.	Age, y	Population	Polysomnographic Criterion for Surgery	Operation	Follow-up Period, mo	Subjects Who Had OSAS at Follow-up	Miscellaneous
Brietzke and Gallagher[130]	2006	III	325	4.9	Various	AHI ≥1	AT	3.3	17.1% (depended on OSAS criteria for each study)	Meta-analysis of 11 case series published between 1980 and 2004
Ye et al[134]	2010	IV	84	7.1 ± 3.2	Chinese	AHI ≥5	AT	18–23	31% with AHI ≥1; 13.1% with AHI ≥5	Obesity and high preoperative AHI were significant independent predictors of treatment failure
Focus on obese populations										
Mitchell and Kelly[279]	2004	III	30	3.0–17.2	Obese (BMI > 95th percentile)	AHI >5	AT	5.6	54%	
Mitchell and Kelly[139]	2007	III	72	3–18	Comparison of obese (BMI >95th percentile) with nonobese	AHI ≥2: AHI 2–5 mild, AHI 5–15 moderate AHI ≥15 severe	AT	5–6	Obese: 76%: (46% mild; 15% moderate; 15% severe). Nonobese: 28%: (18% mild; 10% moderate).	Preoperative AHI and obesity were independent risk factors for postoperative OSAS. OR for persistent OSAS in obese, adjusted for preoperative AHI, was 3.7 (95% CI: 1.3–10.8)
O'Brien et al[136]	2006	III	69	7.1 ± 4.2	Obese (weight >2 SDs from mean for age)	RDI ≥5	AT	20.4 ± 16.8	Nonobese: 22.5%; Obese: 55%	Preoperative AHI and obesity were independent risk factors for postoperative OSAS. OR for persistent OSAS in obese, adjusted for preoperative AHI, was 4.7 (95% CI: 1.7–11.2)
Shine et al[194]	2006	IV	19	6.5 ± 4.4	Obese (BMI >95th percentile)	RDI>5	18 AT (1 with UPPP), 1 T	2–6	63%	Missing data
Costa and Mitchell[131]	2009	III	110	7.3–9.3	Obese	Various	AT	3–5.7	88% had postoperative AHI ≥1; 75% had postoperative AHI ≥2; 51% had postoperative AHI ≥5	Meta-analysis of 4 obesity studies included here
Apostolidou et al[138]	2008	IV	70	6.5 ± 2.2	Greek; obese defined as >1.645 SDs from mean weight for age	OAHI ≥1	AT	2-14	Overall: 75.7% with AHI ≥1 (77.3% obese, 75% nonobese). Among children with a preoperative OAHI ≥5: 9% with AHI ≥5 (8% obese, 10% nonobese)	
Focus on other special populations										
Mitchell and Kelly[140]	2005	III	20	1.1–3.0	Children <3 y	RDI >5	AT	4.1–20.4	65%: 25% RDI 5–10; 25% RDI 10–20; 15% RDI >20	Included comorbidities (Down syndrome, cardiac disease, cerebral palsy) excluded from this guideline. 60% of patients were severe, with RDI >20 at baseline
Mitchell and Kelly[280]	2004	III	29	1.4–17	Severe OSAS	RDI >5; severe: RDI ≥30	AT	6	69% with postoperative RDI >5	48% were obese

A, adenoidectomy; CI, confidence interval; OAHI, obstructive AHI; OR, odds ratio; T, tonsillectomy; REM, rapid eye movement; UPPP, uvulopharyngopalatoplasty.

OSAS, even when controlling for obesity.[132,133]

4. Other Potential Risk Factors

Individual studies have noted that nasal abnormalities or craniofacial disproportion,[141] family history of OSAS,[137] and presence of asthma[133] were all predictive of higher failure rate, but these findings were not substantiated by other studies. Of note, Mitchell[132] found that 13 of 22 patients in the cohort who had postoperative snoring had an AHI ≥5/hour, whereas none of the 57 patients who did not exhibit postoperative snoring had an AHI ≥5/hour. This supports the findings of older studies reviewed in the previous technical report that found absence of snoring to have a 100% negative predictive value for postoperative OSAS.[6] However, in the Chinese cohort, 2 of 11 patients who have persistent AHI ≥5/hour reportedly did not snore; it is unclear whether cultural considerations might have affected parental report of snoring.[134]

Summary

AT is the most effective surgical therapy for pediatric patients, leading to an improvement in polysomnographic parameters in the vast majority of patients. Despite this improvement, a significant proportion of patients are left with persistent OSAS after AT. The estimate of this proportion in a relatively low-risk population ranges from a low of 13% to 29% when using an AHI ≥5/hour as the criterion to a high of 73% when including obese children and adolescents and a conservative AHI ≥1/hour. Children at highest risk of persistent OSAS are those who are obese and those with a high preoperative AHI, especially those with an AHI ≥20/hour, as well as children >7 years of age. Absence of snoring postoperatively is reassuring but may not be 100% specific; it may therefore be advisable to obtain a postoperative PSG in very-high-risk children even in the absence of reported persistent snoring.

Areas for Future Research

- What are the risks of persistence of OSAS and long-term recurrence of OSAS after PT versus total tonsillectomy? Large, prospective, randomized trials with objective outcome measures including PSG are needed.
- Better delineation of which patients would benefit from postoperative PSG.
- How well does resolution of OSAS correlate with resolution of complications of OSAS?
- Are some of the newer surgical techniques for AT equally effective in resolving OSAS?
- What are the risks of performing AT in a patient with a URI?
- What are the PSG parameters that predict postoperative respiratory compromise? Future research should focus on refining the AHI and Spo_2 nadir cutoffs for severe OSAS. In addition, it may be possible to glean other predictive information from the PSG, such as the extent of hypoventilation, the percent sleep time spent with Spo_2 <90%, the frequency of desaturation events, the length of apneas and hypopneas, and the presence of central apneas, to create formulae for risk scores.

CPAP

At the time of the previous report, there were few prospective studies on CPAP use in children, although several retrospective studies indicated that CPAP was efficacious in the treatment of pediatric OSAS. Since that time, there have been at least 7 recent studies evaluating the use of positive airway pressure (PAP) in children and adolescents who have OSAS. One of these was a randomized trial with low power (level II),[142] and others were case series without controls (level IV). A descriptive study examined the use of behavioral intervention in improving CPAP adherence.[143] In addition, a level III study described use of a high-flow nasal cannula as an alternative to CPAP.[144] In contrast to the previous guidelines, several of the current studies obtained objective evaluation of CPAP adherence by downloading usage data from the CPAP device. In most studies, CPAP therapy was instituted for persistent OSAS after AT; in many cases, the patients had additional risk factors for OSAS, such as obesity or craniofacial anomalies.

A multicenter study (level II) evaluated PAP in 29 children who were randomly assigned either CPAP or bilevel positive airway pressure (BPAP).[142] Patients demonstrated significant improvement in sleepiness, snoring, AHI, and oxyhemoglobin saturation while using PAP during the 6-month follow-up period. However, approximately one-third of patients dropped out, and of those who used PAP, objective adherence was 5.3 ± 2.5 hours/night. Parents overestimated the hours of PAP use compared with the devices' actual objective recordings of use. There was no significant difference in adherence between the CPAP and BPAP groups. A retrospective chart review of 46 children started on PAP for OSAS that persisted after AT also showed significant improvement in symptoms of OSAS as well as in polysomnographic parameters (level IV).[145] Seventy percent of patients were considered adherent. Parental report of adherence was most divergent from the machines' recording in the least adherent patients. More

than one-half of the children had complicating factors, such as Down syndrome and Prader-Willi syndrome.[145] Another study of a heterogeneous group of patients displayed varying CPAP adherence, with 31 of 79 children showing continued CPAP use (level IV).[146] A small, nonblinded retrospective study (level IV) suggested that adherence to CPAP could be improved with behavioral techniques if the family accepted the interventions.[143]

A retrospective review described 9 children who successfully used BPAP in the intensive care setting because of respiratory compromise after AT.[147] Another retrospective review described the successful use of CPAP in 9 patients of a heterogeneous group of 18 children aged <2 years.[148] A nonrandomized, prospective level III study of 12 children who had OSAS treated in the sleep laboratory with a high-flow open nasal cannula system as an alternative to formal CPAP demonstrated an improvement in oxyhemoglobin saturation and arousals, but not AHI, compared with baseline.[144] There was a decrease in sleep efficiency with the cannula compared with baseline. Long-term use and use in the home situation were not assessed.

In summary, several studies (levels II–IV) have confirmed earlier data demonstrating that nasal CPAP is effective in the treatment of both symptoms and polysomnographic evidence of OSAS, even in young children. However, adherence can be a major barrier to effective CPAP use. For this reason, CPAP is not recommended as first-line therapy for OSAS when AT is an option. However, it is useful in children who do not respond adequately to surgery or in whom surgery is contraindicated. Patient and family preference may also be a consideration (eg, in families with religious beliefs against surgery or blood transfusions). Objective assessment of CPAP adherence is important because parental estimates of use are often inaccurate. If the patient is nonadherent, then attempts should be made to improve adherence (eg, by addressing adverse effects, by using behavior modification techniques), or the patient should be treated with alternative methods. A study described in the previous report noted that CPAP pressures change over time in children, presumably because of growth and development.[149] Therefore, it is recommended that CPAP pressures be periodically reassessed in children.

At this time, data are insufficient to make a recommendation on the use of high-flow, open nasal cannula systems.

Areas for Future Research

- Efficacy of CPAP use as a first-line treatment of obese children.
- Determinants of CPAP adherence and ways to improve adherence.
- Long-term effects of CPAP, particularly on the development of the face, jaw, and teeth.
- Changes in CPAP pressure over time, and the frequency with which this needs to be monitored.
- Development of pediatric-specific devices and interfaces.

Medications

There have been several studies evaluating the use of corticosteroids and leukotriene antagonists in the treatment of OSAS. An older study showed no therapeutic effect of systemic steroids on OSAS.[150] Since then, 3 studies (1 level I, 1 level II, and 1 level III) have evaluated topical nasal steroids as treatment of OSAS, 1 level II study has evaluated montelukast, and 1 level IV study has evaluated a combination thereof. An additional level I study evaluated the effect of intranasal steroids on adenoidal size and symptoms related to adenoidal hypertrophy but did not include PSG in the evaluation.[151]

A small, level II, randomized, double-blind trial,[152] a level I, randomized, double-blind trial of 62 children,[153] and a nonrandomized, open-label level III study of intranasal steroids[154] all showed a moderate improvement in patients who had mild OSAS. However, significant residual OSAS remained in 2 of the studies. Berlucchi et al[151] reported an improvement in symptoms of adenoidal hypertrophy, including snoring and observed apnea, but did not obtain objective evidence of improvement in OSAS. Two studies showed shrinkage of adenoidal tissue.[151,153] All studies were short term (2–6 weeks), although 1 study showed persistent improvement 8 weeks after discontinuation of the steroids (Table 18).[153]

An open-label, nonrandomized, 16-week level IV study of montelukast in children who had mild OSAS found a statistically significant but small change in the AHI (AHI decreased from 3.0 ± 0.2 to 2.0 ± 0.3; $P = .017$).[82] Another small, open-label, nonrandomized, 12-week level IV study of combined montelukast and nasal steroids found a mild but statistically significant improvement in AHI in children who had mild OSAS (AHI decreased from 3.9 ± 1.2/hour to 0.3 ± 0.3/hour; $P < .001$).[155]

In summary, several small level I through IV studies suggest that topical steroids may ameliorate mild OSAS. However, the clinical effects are small. On the basis of these studies, intranasal steroids may be considered for treatment of mild OSAS (defined, for this indication, as an AHI <5/hour, on the basis of studies described in Table 18). Steroids should not be used as the primary treatment of moderate

TABLE 18 Studies of Antiinflammatory Medications for the Treatment of OSAS

Medication	Source	Level	No.	Duration, wk	Randomized	Placebo-Controlled	Baseline AHI (per h)	AHI on Treatment (per h)	*P*
Intranasal steroids	Brouillette et al[152]	II	13 OSAS 12 controls	6	Yes	Yes	10.7 ± 9.4	5.8 ± 7.9	.04
Intranasal steroids	Alexopoulos et al[154]	III	27 OSAS	4	No	No	5.2 ± 2.2	3.2 ± 1.5	<.001
Intranasal steroids	Kheirandish-Gozal and Gozal[153]	I	62 OSAS	6; crossover	Yes	Yes	3.7 ± 0.3	1.3 ± 0.2	<.001
Montelukast	Goldbart et al[82]	IV	24 OSAS 16 controls	16	No	No	3.0 ± 0.2	2.0 ± 0.3	.017
Intranasal steroids + montelukast	Kheirandish et al[155]	IV	22 OSAS 14 controls	12	No	No	3.9 ± 1.2	0.3 ± 0.3	<.001

or severe OSAS. Because the long-term effects of intranasal steroids are not known, follow-up evaluation is needed to ensure that the OSAS does not recur and to monitor for adverse effects. Of note, no studies specifically evaluated children who had atopy or chronic rhinitis, although 1 study mentioned that similar improvements were seen in children who had a history of allergic symptoms compared with those without.[153] Further study to determine whether children who have atopy are more likely to respond to this therapy is needed. Data are insufficient at this time to recommend treatment of OSAS with montelukast.

Areas for Future Research

- What is the optimal duration of intranasal steroid use? All trials have been short-term with a short-term follow-up. Does the OSAS recur on discontinuation of therapy? How often should objective assessment of treatment effects be performed?
- What is the efficacy of intranasal steroids in children who have chronic or atopic rhinitis?
- How do the benefits and adverse effects of long-term nasal steroids compare with surgery?
- Larger studies, stratified for severity of OSAS and controlled for obesity, to determine whether OSAS is associated with systemic inflammation
- Will these biomarkers be good outcome measurements for treatment studies? Do they correlate with clinical outcomes or long-term prognosis?

Rapid Maxillary Expansion

Rapid maxillary expansion has recently been used to treat OSAS in select pediatric populations. It is an orthodontic procedure designed to increase the transverse diameter of the hard palate by reopening the midpalatal suture. It does this by means of a fixed appliance with an expansion screw anchored on selected teeth. After 3 to 4 months of expansion, a normal mineralized suture is built up again. The procedure is typically used only in children with maxillary constriction and dental malocclusion. Two case series without controls (level IV) have evaluated this procedure as a treatment of OSAS in children. One study described 31 patients selected from an orthodontic clinic; 4 months after surgery, all patients had normalized AHI.[156] Another screened 260 patients in a sleep center to find 35 that were eligible; only 14 were studied.[157] There was a significant improvement in signs and symptoms of OSAS as well as polysomnographic parameters. In summary, rapid maxillary expansion is an orthodontic technique that holds promise as an alternative treatment of OSAS in children. However, data are insufficient to recommend its use at this time.

Areas for Future Research

- A randomized controlled trial to assess the efficacy of rapid maxillary expansion in the treatment of OSAS in children.

Positional Therapy

Several level IV, retrospective studies evaluated the effect of body position during sleep on OSAS. The studies had conflicting results. One study found that young children had an increased AHI in the supine position,[158] and another study found that young children did not have a positional change in AHI but older children did.[159] Another study found an increased obstructive apnea index but not AHI (except in the obese subgroup) in the supine position,[160] whereas a study of obese and nonobese children, which controlled for sleep stage in each position, found that AHI was lowest when children were prone.[161] No study evaluated the effect of changing body positions or the feasibility of maintaining a child in a certain position overnight. Therefore, at this point, no recommendations can be made with regard to positional therapy for OSAS in children.

Other Treatment Options

Specific craniofacial procedures, such as mandibular distraction osteogenesis, are appropriate for select children with craniofacial anomalies. However, a discussion of these children is beyond the scope of this

guideline. Minimal experience is available regarding intraoral appliances in children.[162] A tracheotomy is extremely effective at treating OSAS but is associated with much morbidity and is typically a last resort if CPAP and other treatments fail to offer improvement for a child who has severe OSAS.

OBESITY AND OSAS

This section reviews the evidence regarding the relationships between obesity and SDB (this term is used to encompass both snoring and OSAS, especially in studies that did not distinguish between these entities) in the pediatric population. The prevalence of childhood obesity is increasing,[163] and many studies on obesity and OSAS have been published since the last guideline. Because childhood obesity has a major impact on OSAS, it is described in detail in this report. Obesity is defined as BMI >95th percentile for age and gender.

Epidemiology: Obesity as a Risk Factor for Snoring and OSAS

A number of large, cross-sectional, community-based studies including more than 21 500 children have examined the risk of SDB conferred by overweight and obesity (Table 19). The majority of these studies obtained information regarding potential SDB from questionnaires, but some included objective measurements such as oximetry or overnight PSG. Similarly, many studies based the determination of BMI on data from questionnaires. The ages ranged from 6 to 17 years, consistent with recruitment strategies using local schools. Countries from around the world are represented, including North America, Asia, Europe, and the Middle East. Taken together, these studies indicate that the risk of snoring in children is increased twofold to fourfold with obesity (defined as BMI ≥90th or 95th percentile). When analyzed, BMI was found to be an independent risk factor for snoring.

Several studies based on surveys of thousands of children, in some cases supplemented by use of physical examinations, showed that overweight/obesity was associated with an increased prevalence of snoring (Table 19).[47,164–167] Fewer studies that included objective measurements to identify SDB were available. Two population-based studies using PSG demonstrated a relationship between overweight/obesity and OSAS.[11,12] In contrast to the findings of the majority of studies, Brunetti et al[23] found that although HS was more prevalent in obese children in a sample of schoolchildren, there was no difference in the incidence of OSAS on PSG among the subset of normal-weight, overweight, and obese children who have HS who had abnormal overnight oximetry results. Similar to the population-based studies, studies using case series or subjects recruited from sleep disorders programs (some of which use PSG and some of which use surveys) also showed a relationship between weight and SDB.[168,169]

From these studies, it can be concluded that obesity is an independent risk factor for snoring and OSAS. The range of evidence from individual studies was II to III (Table 19) and on the aggregate rise to level I. The studies reported on large numbers of children recruited from community-based samples, some of whom had face-to-face examinations and measurements. Data obtained in different settings yielded similar results. The impact of race, if any, is not yet clear. Population-based studies of Hispanic children, a group at high risk of obesity and related comorbidities, are not yet available.[163] For the clinician, it is recommended that particular attention is needed for screening obese and overweight children for signs and symptoms of OSAS, with a low threshold for ordering diagnostic tests. Future research should focus on population-based studies, with objective measurements of both measures of adiposity and PSG, and should include larger numbers of African American and Hispanic youth.

Predictors of Obesity-Related SDB

A number of program-based studies provide information regarding the predictors for SDB in obese children. Carotenuto et al[88] reported via data gathered from parental questionnaires that in obese subjects referred for obesity evaluation and nonobese controls randomly selected from schools, the waist circumference *z* score correlated with symptoms of SDB ($R = 0.37$, $P < .006$) but BMI and subcutaneous fat did not (level III). Verhulst et al[170] examined 91 consecutive overweight or obese children referred for PSG and found that OSAS was not related to indices of obesity, including bioelectric impedance analysis fat mass (level III). Central apnea was significantly predicted by using BMI score, waist circumference, waist-to-hip circumference ratio, and percent fat mass. Tonsillar size was the only significant correlate in their model for moderate to severe OSAS. In a retrospective review of 482 Chinese children referred for PSG and evaluated by using BMI and a tonsillar grading scale, the group of 111 obese children had a significantly higher median AHI and percentage with AHI >1.5/hour than did the nonobese group (level III).[171] In a regression analysis of log AHI as dependent variable, BMI and tonsil grade were predictors, but age and gender were not. In a large study of schoolchildren in

TABLE 19 Risk of SDB Conferred by Overweight and Obesity

Source	Level	Type of Study	No.	Duration	Diagnostic Technique	Other Features	Findings	*P* for Obesity as a Risk Factor
Urschitz et al[164]	II	Community-based sample of third graders	1144	1 y	Parental report of snoring, BMI, SES, risk factors for rhinitis, asthma	Habitual snorers reassessed at 1 y, with 49% continuing to snore	BMI ≥90% conferred a 4 times higher risk of HS versus a BMI <75%; 25% of obese subjects had HS	
Corbo et al[166]	II	Community-based sample of 10- to 15-y-old children from 10 schools	2439	2 y	Parental questionnaire and nasal examination and BMI by physician		Snoring increased significantly with BMI >90% and was >2 times for BMI >95% vs <75%	.000
Shin et al[47]	IV	Cross-sectional community-based sample of high school students	3871	NA	Questionnaire (tested for reliability) completed by subject, caretakers, and sleep partner	Korean children; 81% response rate to survey	Snoring frequency was significantly associated with increasing BMI	<.001
Bidad et al[167]	II	Cross-sectional study of 11- to 17-y-old children	3300	NA	Scripted face-to-face interview and measurements of BMI and tonsil size by physician	7.9% of sample with HS (≥3 nights per week when well)	>Twofold risk of snoring in overweight or obesity	
Stepanski et al[168]	III	Case series; mean age: 5.9 ± 3.7 y	190	NA	Clinical interview, PSG	68% with SDB (≥5 AHI, <90% Spo_2, sleep fragmentation, ECG changes)	BMI was higher in the SDB group	<.01
Rudnick et al[169]	III	Compared children scheduled for AT with control group from same urban setting	170 SDB 129 controls	NA	BMI, ethnicity		African American children who had SDB were more likely to be obese than African American children who did not have SDB	<.02
Li et al[12]	II	Cross-sectional study of 13 primary schools	6447 by questionnaire 410 high risk and 209 low risk with exam and PSG	NA	Questionnaire in all with PSG and examination in high-risk group and low-risk subset for comparison	Hong Kong 9172 sampled with 70% response rate	Male gender, BMI, and AT size were independently associated with OSA	
Li et al[172]	II	Cross-sectional study of 13 primary schools; same population as previous study	6349	NA	Questionnaire	Designed to determine prevalence of HS and associated symptoms.	Prevalence of HS was 7.2%; male gender, BMI, parental HS, nasal allergies, asthma were associated with snoring	<.0001
Brunetti et al[23]	II	Cross-sectional; mean age 7.3 y	1207 screened, 809 eligible	NA	Questionnaire in all followed by oximetry in the 44 who had HS; PSG in subset who had abnormal oximetry results	Southern Italy	HS more common in the obese group; no difference in OSA by PSG across weight groups	.02
Bixler et al[11]	II	Cross-sectional study of grades K–5	5740 had questionnaire 700 randomly selected for PSG, 490 completed	NA	Questionnaire followed by PSG in subset	Prevalence of AHI >5 1.2%. Strong linear relationship between waist circumference and BMI with SDB	Waist circumference associated with all levels of SDB, also nasal complaints and minority race	
Urschitz et al[165]	III	Cross-sectional community-based of primary schoolchildren	995	NA	Overnight oximetry		Overweight, smoke exposure, respiratory allergies were independent risk factors for sleep hypoxemia	

AT, adenotonsillar; K, kindergarten; NA, not available; OSA, obstructive sleep apnea.

Hong Kong, Li et al reported that male gender, BMI score, and tonsillar size were independently associated with OSAS (level II).[12,172] In 490 US schoolchildren studied by using overnight PSG, Bixler et al[11] found waist circumference to be an independent risk factor for all levels of severity of OSAS (level II). Urschitz et al[165] studied 995 children in a cross-sectional, program-based study in Germany and divided those with SDB into mild ($Sp0_2$ nadir 91%–93%), moderate (<90%), and recurrent hypoxemia (>3.9 episodes of desaturation per hour of sleep) groups (level III). Overweight (BMI >75th percentile) was found to be an independent risk factor for mild, moderate, and recurrent hypoxemia during sleep.

From these studies, it is observed that the distribution of body fat may be more important in predicting SDB than BMI alone. In addition, tonsillar size is important in predicting SDB, even in obese children. The authors of these articles comment that SDB is likely more complicated in obese children, with obesity contributing to gas exchange and respiratory pattern abnormalities. Obesity can result in decreased lung volumes, abnormal central nervous system ventilatory responses, decreased upper airway caliber, a potential impact of leptin on ventilation, and other factors. Taken together, the strength of the evidence for these study findings is level II. Findings are limited by the fact that controls were drawn from different populations than subjects and that the studies did not all reach the same conclusions regarding the importance of body fat distribution. The latter may have been affected by the use of different measurement techniques. Anthropomorphic measurement thresholds that indicate increased risk for SDB in children would be of use to clinicians. It is recommended that clinicians consider fat distribution (eg, waist circumference) and not just BMI in their assessment of the risk of SDB.

Comorbidities: Interactions Between Obesity and SDB

Cardiovascular

Adults who have SDB and are obese are at increased risk of cardiovascular disease, including systemic hypertension and blunting of the normal decrease in BP during sleep (nocturnal dipping). This section deals with the evidence that children and adolescents who are obese and have SDB may be similarly at risk. Six studies evaluating SDB, obesity, and cardiovascular complications in children are available. Reade et al[173] retrospectively evaluated 130 patients referred for PSG and described 56 obese subjects (BMI >95th percentile), of whom 70% had hypertension and 54% had OSAS (level IV). Among the 34 nonobese subjects, only 8% ($P < .0005$) had hypertension and 29% had OSAS ($P < .05$). The authors concluded that BMI was a significant determinant of both SDB and diastolic BP, with the number of hypopneas predictive of diastolic BP in both weight categories. In a community-based sample of 760 Greek children evaluated by using morning BP measurements, BMI, and a questionnaire regarding sleep habits, Kaditis et al[174] identified 50 children who had HS (level IV). They found that 28% of the children in the HS group were obese versus 15% of nonsnoring children (significance not reported). They reported that HS had no impact on BP, but that age, gender, and BMI were significant covariates in predicting systolic BP; inclusion of HS in this analysis did not affect these relationships. Similar findings were identified for diastolic BP, with the exception that age had no effect. This study compared absolute BP measurements rather than the variance from normal values on the basis of race, age, gender, and body size. Because children from 4 to 14 years of age were included, this may have affected the results and conclusions. Kohyama et al[175] examined 32 Asian subjects referred for PSG and measured overnight BP every 15 minutes. In this study, obstructive apneas and hypopneas were identified indirectly and, thus, could have been underestimated or overestimated compared with studies with more direct measurements of airflow (level IV). Subjects were divided into low (<10 obstructive events per hour; 16 subjects) and high AHI (>10 obstructive events per hour; 7 subjects). Of the total, 23 subjects tolerated the BP measurements. Three subjects were obese. BMI predicted the systolic BP during rapid eye movement sleep ($P < .001$) but did not predict any of the diastolic BP indices. Li et al[176] performed a population-based study of 306 Asian children 6 to 13 years of age who had overnight PSG and ambulatory day and night BP measurements (level III). Children who had primary snoring were excluded, and those who had OSAS were divided into normal, mild, and moderate (AHI >5) groups. Multiple linear regression analysis revealed significant associations for the severity of hypoxemia and AHI with day and night BP, respectively, independent of obesity. Although BP levels both awake and asleep increased with the severity of OSAS, obesity and waist circumference partially accounted for elevations in sleep systolic BP and sleep mean arterial pressure but not for diastolic BP measurements. Amin et al[177] studied 88 children who had OSAS ranging in severity from mild to severe and 52 controls matched for age and gender. They used PSG, ambulatory BP measurements, and actigraphy (level III). The obese SDB group, compared with the nonobese SDB group, had higher

waking systolic BP ($P < .001$) and sleeping systolic BP ($P = .02$) after adjusting for severity of SDB. They concluded that there was no difference between the effects of SDB and obesity on waking systolic or diastolic BP or sleeping systolic BP but did find that SDB had a greater contribution to sleeping diastolic BP than did obesity.

In summary, this group of articles demonstrates that both obesity and SDB are associated with increased day and night BP in children, although hypertension per se is rare (aggregate evidence level III). It seems that after controlling for obesity, significant independent effects of SDB remain and that hypoxemia and the frequency of obstructive events, perhaps via sleep disruption or intrathoracic fluid shifts, are important. Practitioners should be aware that children and adolescents who have OSAS are at increased risk of elevated BP. Future studies would benefit from a treatment arm to determine whether BP improves with resolution of sleep apnea, as well as longitudinal studies to determine the impact of pediatric obesity related–SDB on adult hypertension.

Metabolic

Obesity is a risk factor for impaired glucose tolerance, liver disease, abnormal lipid profiles, and other metabolic derangements. OSAS has been explored as a possible contributor to these metabolic abnormalities. Ten articles were reviewed. Verhulst et al[178] studied 104 overweight/obese children and adolescents with Tanner staging, overnight PSG, oral glucose tolerance testing, lipid profile, and BP measurements (level IV). The subjects were divided into normal, mild, and moderate/severe SDB groups. Findings consistent with the metabolic syndrome were present in 37%. Those who had a moderate degree of SDB had a higher BMI *z* score than the normal group, and the waist-to-hip circumference ratio increased across the 3 SDB groups. The severity of SDB was independently correlated with impaired glucose homeostasis and worse lipid profile. Mean $Sp0_2$ and $Sp0_2$ nadir during sleep were significant predictors of the metabolic syndrome ($P = .04$ for both). A community-based cohort of 270 adolescents was studied by Redline et al[179] using PSG, oral glucose tolerance testing, homeostatic model assessment (HOMA [a measure of insulin sensitivity]), BMI, waist circumference, BP measurements, Tanner stage, sleep diary, SES, and birth history (level II). Metabolic syndrome was defined as having at least 3 of the following 5 features: (1) waist circumference >75% of normal; (2) mean BP or diastolic BP >90% of normal or receiving current therapy for hypertension; (3) elevated triglycerides; (4) low high-density lipoprotein; or (5) abnormal oral glucose tolerance or fasting glucose test results. Twenty-five percent of the sample was overweight, and 19% were deemed to have metabolic syndrome. The authors found that children who had metabolic syndrome had more severe hypoxemia and decreased sleep efficiency and that as AHI severity increased, there was a progressive increase in the number of children who had metabolic syndrome ($P < .001$). Both overweight children and those who had metabolic syndrome were more prevalent in the SDB group ($P < .001$) and more were male. Age, race, birth history, and SES did not vary with SDB. With adjustment for BMI, the SDB group had higher BP, fasting insulin, and more abnormal HOMA and lipid profile. They concluded that adolescents who experience SDB are at a sevenfold increased risk of metabolic syndrome and that the relationship is not explained by gender, race, or SES and, furthermore, persists with adjustment for BMI percentile.

A study by Kaditis et al[180] of 110 children (2–13 years of age) referred for snoring did not find an impact of SDB on glucose homeostasis in nonobese children. The subjects were divided into AHI ≥5/h and <5/h; the authors found no difference in HOMA, insulin, glucose, or lipid concentrations between the 2 groups (level III). There was no relationship identified between PSG indices and HOMA or fasting insulin. BMI, age, and gender were significant predictors for fasting insulin and HOMA in multiple linear regression analysis. They speculated that OSAS may have more detrimental effects in obese than in nonobese young subjects. Similarly, Tauman et al[181] studied 116 subjects referred for PSG, one-half of whom were obese, and 19 nonsnoring controls. The authors found no impact of SDB indices on metabolic parameters (level III). Only BMI and age were important, and there was no relationship between SDB and surrogate measures of insulin resistance. They concluded that obesity was the major determinant of insulin resistance and dyslipidemia. In obese children, data from de la Eva et al[182] demonstrated that the severity of OSAS correlated with fasting insulin levels, independent of BMI (level III). Of note, the study by Redline et al[179] included children older than those in the studies by Kaditis et al[180] and Tauman et al[181]; thus, the variation in the findings may be a function of the length of time SDB had been present or perhaps attributable to the strong influence puberty has on glucose homeostasis. Kelly et al[183] compared 37 prepubertal and 98 pubertal children in a study by using PSG, HOMA, adiponectin (an insulin-sensitizing hormone secreted by adipose tissue) measurements, as well as urinary catecholamine metabolites (level III).

Tanner stage was determined by self-attestation. In the prepubertal children, they found no association between polysomnographic parameters and metabolic measurements after correcting for BMI. Elevated fasting insulin (≥20 μU/mL) was significantly more common in the OSAS group (P = .03), even when corrected for BMI. When pubertal obese subjects were considered separately, the risk of elevated fasting insulin (P = .04) and impaired HOMA was greater in the OSAS group (P = .05). Pubertal children who had OSAS also had lower adiponectin and higher urinary catecholamine levels, even when controlled for BMI. Kelly et al concluded that OSAS further predisposes obese children to metabolic syndrome, likely through multiple mechanisms involving adipose tissue and the sympathetic nervous system.

In a study that included pretreatment and posttreatment measurements in 62 prepubertal children who had moderate to severe OSAS, Gozal et al[184] found that although nonobese children had no change in measures of glucose homeostasis after treatment of OSAS, obese children had a significant improvement even while BMI remained stable (P < .001) (level II). Similar effects were not seen in nonobese children. Treatment (AT) improved the lipid profile and inflammatory markers in both obese and nonobese children.

Other studies have examined different aspects of altered metabolism in obesity-related OSAS. Kheirandish-Gozal et al[185] found elevated alanine transaminase (a marker for fatty liver) in a large sample of obese children who had OSAS (level IV). Verhulst et al[186] found elevated serum uric acid (a marker of oxidative stress) in 62 overweight children who had OSAS, with a significant relationship between the severity of OSAS and serum uric acid independent of abdominal adiposity (P = .01) (level IV). Verhulst et al[187] demonstrated that, in a group of 95 obese and overweight children, total white blood cell and neutrophil counts increased with hypoxemia, and they speculated that inflammation may contribute to cardiovascular morbidity in obesity-related SDB (level IV).

In summary, as expected, this group of studies confirms that obesity increases the risk of insulin resistance, dyslipidemia, and other metabolic abnormalities in children. The role that OSAS plays in altering glucose metabolism is still not entirely clear but is likely less important in younger children and in lean children. Conflicting studies exist regarding the independent effect of OSAS on metabolic measures when it coexists with obesity in children. Puberty has an important role in this relationship. Screening of obese children who have OSAS for markers of metabolic syndrome should be considered, especially in the adolescent age group. Individual studies were level II through IV, with an aggregate level of III.

Neurobehavioral

The neurobehavioral complications of OSAS are discussed in detail elsewhere in this technical report. However, 6 studies have explored the potential contribution of obesity to behavior and cognition in children with OSAS and will be discussed in this section. A subanalysis of the Tucson Children's Assessment of Sleep Apnea Study evaluating parent-rated behavioral problems in overweight children before and after controlling for OSAS was performed by Mulvaney et al (level II).[188] They analyzed data from 402 subjects, 15% of whom were overweight; data were derived from home overnight PSG, the Conners scale, and the Child Behavior Checklist (CBCL). They found that, after controlling for OSAS, behaviors such as withdrawal and social problems were higher in obese children compared with nonobese children. This finding emphasizes the need to control for obesity when designing studies evaluating neurobehavioral issues in children with OSAS. Chervin et al[42] evaluated students in the second and fifth grades in 6 elementary schools (level IV). Only 146 of 806 surveys were returned. Parental survey of health, race, BMI, Pediatric Sleep Questionnaire, teacher-rated performance, and SES were collected. SDB was associated with African American race, SES, and poor teacher ratings (P < .01), but only SES was independently associated with school performance. Low SES was not associated with SDB when controlled for BMI. The authors concluded that future studies evaluating the relationship between school performance and SDB should incorporate direct measurements of SES and obesity. Owens et al[189] examined all children evaluated at a tertiary center for sleep problems between 1999 and 2005; they used PSG, BMI, the Children's Sleep Health Questionnaire, and a mental health history, including the CBCL (level IV). In this study of 235 participants, 56% had a BMI >85th percentile and were thus considered overweight. They found modest correlations between measures of SDB and both somatic complaints and social problems but not with other behavioral complaints. Increased BMI was associated with total CBCL score, internalizing, social, thought, withdrawn, anxious, somatic, and aggressive behavior domains in a dose-response fashion (P = .03), thus emphasizing the need to control for obesity in future studies. Short sleep also correlated with a number of subscales on the CBCL (P < .001). Additional sleep disorders added to the risk of behavior

problems ($P < .001$). BMI predicted both total and internalizing CBCL scores, and sleep duration predicted externalizing scores. The presence of an additional sleep diagnosis was the strongest predictor of all 3 CBCL scores. They concluded that overweight, insufficient sleep, and other sleep disorders should be considered when evaluating and treating behavioral problems associated with SDB. Beebe et al[21] studied 60 obese subjects recruited from a weight-management program compared with 22 controls; tools used included BMI; parent- and self-reported validated sleep, behavior, and mood questionnaires; actigraphy; and PSG (level IV). They reported that the obese group had later bedtimes ($P < .05$), shorter ($P < .01$) and more disrupted sleep ($P < .05$), more symptoms of OSAS ($P < .001$), sleepiness ($P = .009$), parasomnias ($P = .007$), higher AHI ($P < .01$), and poorer school performance. Another study by Beebe et al[190] of 263 overweight subjects enrolled in a hospital-based weight-management program found a negative relationship between the severity of OSAS and school performance and parent- and teacher-reported behaviors that persisted with adjustment for gender, race, SES, sleep duration, and BMI (level IV). Interestingly, Roemmich et al[191] found a relationship between a decrease in motor activity and increasing weight in overweight children after surgical treatment of OSAS by using AT ($P = .03$) (level IV). They hypothesized that a decrease in physical activity and "fidgeting" energy expenditure were responsible for the weight gain. However, because obese controls without surgery were not studied, it is unclear whether the degree of weight gain was greater than typically seen in obese children.

In summary, these studies point to obesity as a potential important factor in childhood performance, mood, and behavior (aggregate level III). Clinicians should be aware that children who are obese and have OSAS might continue to have difficulties in these domains after treatment of OSAS. It is recommended that sleep habits and nonrespiratory sleep complaints be included in the evaluation and treatment of obesity-related OSAS. The relationship between SES, obesity, and OSAS is complex and adds further emphasis to the premise that studies of behavior and cognition must be carefully designed and controlled.

QoL

Both obesity and OSAS can affect health-related QoL. Two studies have examined measures of QoL in children who are obese and have OSAS. In a study of 151 overweight children by Carno et al[192] that used surveys of QoL and SDB and PSG, overweight youth who have OSAS were found to have lower self- and parent-related QoL (level IV). Neither objective measures of OSAS by PSG nor BMI correlated with QoL, whereas reported symptoms of OSAS did ($P < .05$). Similarly, Crabtree et al[193] compared 85 children 8 to 12 years of age who had been referred for OSAS and who underwent PSG, BMI, QoL ascertainment, and the Children's Depression Inventory with a control group with previously documented normal PSG (level IV). They found that OSAS did not differ between obese and nonobese children and that there was no difference in QoL between children who snore and have OSAS. The referred SDB group had lower QoL scores than the control group ($P < .001$), but the authors found no difference between obese and nonobese SDB subjects or in those with OSAS versus snoring. They concluded that children who snore have a lower QoL than nonsnoring controls, and that this finding was not related to obesity of the severity of SDB.

In summary, QoL is an important outcome measure that may be more related to perceived symptoms of OSAS than measured physiologic disturbances of sleep and breathing, even in the obese patient (aggregate level IV). The impact of obesity on QoL in children with SDB is yet to be determined by using population-based studies and is an important outcome measure to be included in longitudinal and treatment studies.

Surgical Treatment of OSAS in the Obese Child

Surgical treatment of OSAS in general is discussed in detail in the technical report, but 5 studies have examined this area in obesity-related OSAS and are discussed here. Shine et al[194] evaluated 19 obese patients treated with AT (level IV). Although OSAS improved significantly ($P < .01$), only 37% of patients were deemed cured (defined as a postoperative AHI <5/hour), and 10 (53%) subjects needed CPAP postoperatively. A level IV retrospective review by Spector et al[195] included 14 patients who were morbidly obese who were electively sent to the ICU after AT (per policy). One patient needed intubation, and 2 patients required BPAP. Another retrospective review of 26 morbidly obese patients, all of whom were sent to the ICU after AT as per routine, found that 14 patients (54%) had an uncomplicated postoperative course, and 12 (45%) required respiratory intervention, including 1 requiring intubation and 2 requiring BPAP.[196] Costa and Mitchell[131] evaluated the response to AT in a meta-analysis of 4 studies that included 110 obese children who had OSAS (level III). They found that OSAS improved but did not resolve after AT, with 88% of children having an AHI >1/hour and 51% of

children having an AHI >5/hour postoperatively. Apostolidou et al[138] reported on 70 snoring children with a mean age of 5.8 ± 1.8 years who underwent AT; 22 (31%) were obese (level IV). PSG was performed both preoperatively and postoperatively. They found no difference in cure rates between obese and nonobese subjects who had OSAS, by using an AHI <1/hour as the definition of cure. However, there was an improvement in AHI in both groups, and approximately 90% of all subjects had an AHI <5/hour postoperatively.

In summary, few studies have evaluated the effects of AT in the obese child who has OSAS, and studies have been of a low level of evidence (aggregate level IV). Studies suggest that the AHI may improve significantly after AT, even in obese children, supporting the idea that surgery may be a reasonable first-line treatment, even in obese patients. However, better-level studies are needed to assess the effects of AT in obese children and adolescents, including evaluation of subgroups such as adolescents and the morbidly obese. A significant number of children required intubation or CPAP postoperatively, which reinforces the need for inpatient observation in obese children postoperatively. Studies have not been performed to determine whether children at high risk who are obese and have OSAS, such as those with pulmonary or systemic hypertension, waking hypoventilation, or pathologic daytime sleepiness, may benefit from stabilization with BPAP therapy before undergoing AT to decrease the risk of postoperative complications.

Weight Loss and Other Nonsurgical Treatments

There is a paucity of data regarding the effects of weight loss on OSAS in children and adolescents. Verhulst et al[197] found that weight loss was a successful treatment of OSAS in a group of 61 adolescents being cared for in a residential weight loss treatment program (level IV). Davis et al[49] studied the effects of exercise in 100 overweight children by administering the Pediatric Sleep Questionnaire before and after enrollment in a no-exercise group, a low-dose aerobic exercise program, or a high-dose aerobic exercise program for 3 months (level IV). They found no change in BMI, but 50% of children who screened positive for SDB improved to a negative screening result after intervention. They found their results to be consistent with a dose-response effect of exercise on improvement in SDB ($P < .001$). Academic achievement did not improve in concert with changes in the Pediatric Sleep Questionnaire. Kalra et al[198] showed a significant improvement in OSAS after bariatric surgery, in association with a mean weight loss of 58 kg (level IV). In summary, along with many other health-related benefits, achieving weight loss and increasing exercise seem to be beneficial for OSAS and should be recommended along with other interventions for OSAS in obese children and adolescents (aggregate level IV). However, it should be noted that the 2 weight loss studies involved treatment regimens that are not commonly available to the majority of obese children. The effects of more modest weight loss regimens require further evaluation.

Pulmonary Disease and Obesity-Related SDB

Two studies addressed the relationship between obesity-related SDB and pulmonary disease. This has been described in adults as the "overlap syndrome," when chronic obstructive pulmonary disease and OSAS are present in the same individual. As part of the Cleveland Children's Sleep and Health Study, Sulit et al[199] evaluated parent-reported wheeze and asthma, history of snoring, and PSG in 788 participants (level III). They found that children who experienced wheeze and asthma were more likely to be obese ($P = .0097$) and concluded that SDB may partially explain this finding. They speculated that obesity changes airway mechanics and that SDB may increase gastroesophageal reflux, leptin levels, and cytokines and, thus, increase lower airways inflammation. Dubern et al[200] studied 54 children who had BMI z scores >3, 74% of whom were pubertal, by using history, physical examination, assessment of body fat mass, Tanner stage, HOMA, lipid profile, leptin, pulmonary function tests, and PSG (level IV). They confirmed the presence of OSAS, lower functional residual capacity, increased airways resistance, lower airways obstruction, and insulin resistance in this group of morbidly obese children. Snoring and AHI correlated with BMI ($P = .01$) and neck/height ratio ($P = .03$) (adjusted for age, gender, Tanner stage, and ethnicity). Airways resistance correlated with snoring index and AHI after adjustment. These studies remind us that the upper airway is part of the respiratory system and that its function is affected by lung mechanics. Abnormalities of pulmonary mechanics related to obesity affect OSAS and may add to abnormalities of gas exchange during sleep. It is suggested that evaluation of the child who is obese and has OSAS should include a history and physical examination directed at the entire respiratory system, and pulmonary function testing may be indicated.

Areas for Future Research

- What threshold of easily obtained anthropomorphic measurements predicts a significant risk of OSAS?

Overweight as well as obese children should be included in future studies.

- Are there additive or multiplicative effects of OSAS and obesity on BP? How do these relationships evolve over time, and what is the impact of genetic and racial background? Does treatment of OSAS improve hypertension in obese children and adolescents?
- The effect of OSAS on metabolic syndrome in children and adolescents remains controversial. Future research should include treatment arms with careful measurements before and after interventions. Longitudinal studies that track changes during puberty and into adulthood would be of interest.
- Further research is needed to clarify the effects of AT on OSAS, including evaluation of subgroups such as adolescents and morbidly obese patients. There should also be studies evaluating the use of CPAP or BPAP before surgery in the obese population, as a way of stabilizing the cardiopulmonary system and reducing operative risk.
- What is the effect of modest weight loss on OSAS in children and adolescents? Research should be directed at identifying strategies to effectively implement weight loss and exercise programs in this population.

SUBCOMMITTEE ON OBSTRUCTIVE SLEEP APNEA SYNDROME*

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OVERSIGHT FROM THE STEERING COMMITTEE ON QUALITY IMPROVEMENT AND MANAGEMENT, 2009--2011

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ACKNOWLEDGMENT

The Committee thanks Christopher Hickey for administrative assistance.

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(Continued from first page)

www.pediatrics.org/cgi/doi/10.1542/peds.2012-1672

doi:10.1542/peds.2012-1672

PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275).

Diagnosis and Management of Childhood Obstructive Sleep Apnea Syndrome

Carole L. Marcus, Lee J. Brooks, Sally Davidson Ward, Kari A. Draper, David Gozal, Ann C. Halbower, Jacqueline Jones, Christopher Lehmann, Michael S. Schechter, Stephen Sheldon, Richard N. Shiffman and Karen Spruyt

Pediatrics; originally published online August 27, 2012;
DOI: 10.1542/peds.2012-1672