Practice Parameters for the Respiratory Indications for Polysomnography in Children

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Background: There has been marked expansion in the literature and practice of pediatric sleep medicine; however, no recent evidence-based practice parameters have been reported. These practice parameters are the first of 2 papers that assess indications for polysomnography in children. This paper addresses indications for polysomnography in children with suspected sleep related breathing disorders. These recommendations were reviewed and approved by the Board of Directors of the American Academy of Sleep Medicine.

Methods: A systematic review of the literature was performed, and the American Academy of Neurology grading system was used to assess the quality of evidence.

Recommendations for PSG Use:

1. Polysomnography in children should be performed and interpreted in accordance with the recommendations of the AASM Manual for the Scoring of Sleep and Associated Events. (Standard)
2. Polysomnography is indicated when the clinical assessment suggests the diagnosis of obstructive sleep apnea syndrome (OSAS) in children. (Standard)
3. Children with mild OSAS preoperatively should have clinical evaluation following adenotonsillectomy to assess for residual symptoms. If there are residual symptoms of OSAS, polysomnography should be performed. (Standard)
4. Polysomnography is indicated following adenotonsillectomy to assess for residual OSAS in children with preoperative evidence for moderate to severe OSAS, obesity, craniofacial anomalies that obstruct the upper airway, and neurologic disorders (e.g., Down syndrome, Prader-Willi syndrome, and myelomeningocele). (Standard)
5. Polysomnography is indicated for positive airway pressure (PAP) titration in children with obstructive sleep apnea syndrome. (Standard)
6. Polysomnography is indicated when the clinical assessment suggests the diagnosis of congenital central alveolar hypoventilation syndrome or sleep related hypoventilation due to neuromuscular disorders or chest wall deformities. It is indicated in selected cases of primary sleep apnea of infancy. (Guideline)
7. Polysomnography is indicated when there is clinical evidence of a sleep related breathing disorder in infants who have experienced an apparent life-threatening event (ALTE). (Guideline)
8. Polysomnography is indicated in children being considered for adenotonsillectomy to treat obstructive sleep apnea syndrome. (Guideline)
9. Follow-up PSG in children on chronic PAP support is indicated to determine whether pressure requirements have changed as a result of the child's growth and development, if symptoms recur while on PAP, or if additional or alternate treatment is instituted. (Guideline)
10. Polysomnography is indicated after treatment of children for OSAS with rapid maxillary expansion to assess for the level of residual disease and to determine whether additional treatment is necessary. (Option)
11. Children with OSAS treated with an oral appliance should have clinical follow-up and polysomnography to assess response to treatment. (Option)
12. Polysomnography is indicated for noninvasive positive pressure ventilation (NIPPV) titration in children with other sleep related breathing disorders. (Option)
13. Children treated with mechanical ventilation may benefit from periodic evaluation with polysomnography to adjust ventilator settings. (Option)
14. Children treated with tracheostomy for sleep related breathing disorders benefit from polysomnography as part of the evaluation prior to decannulation. These children should be followed clinically after decannulation to assess for recurrence of symptoms of sleep related breathing disorders. (Option)
15. Polysomnography is indicated in the following respiratory disorders only if there is a clinical suspicion for an accompanying sleep related breathing disorder: chronic asthma, cystic fibrosis, pulmonary hypertension, bronchopulmonary dysplasia, or chest wall abnormality such as kyphoscoliosis. (Option)

Recommendations against PSG Use:

16. Nap (abbreviated) polysomnography is not recommended for the evaluation of obstructive sleep apnea syndrome in children. (Option)
17. Children considered for treatment with supplemental oxygen do not routinely require polysomnography for management of oxygen therapy. (Option)

Conclusions: Current evidence in the field of pediatric sleep medicine indicates that PSG has clinical utility in the diagnosis and management of sleep related breathing disorders. The accurate diagnosis of SRBD in the pediatric population is best accomplished by integration of polysomnographic findings with clinical evaluation.

Keywords: Polysomnography, pediatric, indications, clinical utility, sleep related breathing disorders, obstructive sleep apnea syndrome

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1.0 INTRODUCTION

Assessment for sleep disorders in children has long relied on a comprehensive history and physical exam. In certain conditions, most commonly sleep related breathing disorders (SRBD), polysomnography (PSG) was performed as an adjunctive tool to aid in the diagnosis. PSG also has been used to evaluate abnormal sleep related movements and behaviors as well as part of the diagnostic assessment for narcolepsy. Because PSG is relatively expensive, time consuming, and not consistently applied by pediatric physicians, it is important to understand its strengths, limitations, and clinical utility in children.

Over the past 30 years, pediatric sleep medicine has exponentially advanced in terms of improved awareness of pediatric sleep disorders and the development of many important technical tools. The early data supporting the clinical utility of PSG were limited because of inconsistency in polysomnographic criteria for SRBDs. Clinical guidelines regarding indications for pediatric PSG based on these early data were published by professional organizations,[1,2] but these older publications were largely consensus-based due to a paucity of evidence-based data.

In 2007 the AASM commissioned a task force to review the literature published on the indications for PSG in children. Because the task force identified such a large number of publications, it divided the project into 3 separate review papers: (1) the respiratory indications for PSG; (2) the non-respiratory indications for PSG; and (3) the indications for PSG in children with attention deficit hyperactivity disorder (ADHD) or autistic spectrum disorder (ASD). It should be noted that in 2009 the SPC changed its grading system to the GRADE methodology, redefining criteria for Standards, Guidelines, and Options to be consistent with GRADE specifications.[3] Because this project began in 2007, the older grading process in use at that time was used to grade the evidence.

This parameter paper focuses on the respiratory indications for PSG. It is based on a comprehensive review of the literature to evaluate the validity and reliability of PSG and to determine its clinical utility for assessment and management of various respiratory disorders. It highlights pediatric respiratory disorders with a high prevalence of polysomnographic abnormalities and addresses various treatment modalities such as surgery and positive airway pressure.

2.0 METHODS

The Standards of Practice Committee of the AASM, in conjunction with specialists and other interested parties, developed these practice parameters based on the accompanying review paper.[4] A task force of content experts was appointed by the AASM in 2007 to review and grade evidence in the scientific literature regarding the validity, reliability, and clinical utility of PSG in pediatric sleep disorders. In most cases recommendations are based on evidence from studies published in the peer reviewed literature or on generally accepted patient care strategies. When scientific data were absent, insufficient or inconclusive, the Rand/UCLA Appropriateness Method was used to develop consensus recommendations by identifying the collective opinion of the SPC and task force. The Rand/UCLA Appropriateness Method combines the best available scientific evidence with the collective judgment of experts to yield statements regarding the appropriateness of performing procedures. In particular, it involves development of a list of specific indications derived from the scientific evidence. Completion of rating sheets that evaluated the appropriateness of these indications was conducted in 2 rounds by members from both the SPC and task force. Based on these ratings, indications were classified as appropriate, uncertain, or inappropriate. Indications that were classified as appropriate were used to develop these recommendations; indications that were uncertain or inappropriate were rejected.

The Board of Directors of the AASM approved these recommendations. All members of the AASM Standards of Practice Committee and Board of Directors completed detailed conflict-of-interest statements and were found to have no conflicts of interest with regard to this subject.

These practice parameters define principles of practice that should meet the needs of most patients in most situations. These guidelines should not, however, be considered inclusive of all proper methods of care or exclusive of other methods of care reasonably directed to obtaining the same results. The ultimate judgment regarding propriety of any specific care must be made by the physician, in light of the individual circumstances presented by the patient, available diagnostic tools, accessible treatment options, and resources.

The AASM expects these guidelines to have an impact on professional behavior, patient outcomes, and, possibly, health care costs. These practice parameters reflect the state of knowledge at the time of publication and will be reviewed, updated, and revised as new information becomes available. This parameter paper is referenced, where appropriate, using square-bracketed numbers to the relevant sections and tables in the accompanying review paper, or with additional references at the end of this paper. For this paper, the Standards of Practice Committee decided to use an evidence grading system developed by the American Academy of Neurology (AAN) for assessment of diagnostic tests. The system involves 4 tiers of evidence, with Level 1 studies judged to have a low risk of bias and Level 4 studies judged to have a very high risk of bias. Table 1 describes the essential features of the evidence grading system used by the task force. Definitions of levels of recommendations used by the AASM appear in Table 2.

3.0 RECOMMENDATIONS

3.1 Methodology

3.1.1 Polysomnography in children should be performed and interpreted in accordance with the recommendations of the AASM Manual for the Scoring of Sleep and Associated Events. (Standard)

A detailed evidence-based and consensus-based review of PSG was conducted during development of the AASM Manual for the Scoring of Sleep and Associated Events under the aegis of the AASM Scoring Manual Steering Committee.[7] This document specifies the optimal equipment for PSG and standardizes the scoring of events. The SPC considers this the standard of practice, and therefore did not conduct a separate, independent review for this practice parameter.

In the opinion of the task force, PSG is best tolerated by the child and parent under the following conditions: (a) when PSG is performed with the caretaker present; (b) when the

parent and child have been oriented to the sleep laboratory in advance, including, in some cases, application of a limited number of sensors to illustrate the procedure to the child; (c) when the PSG technologist is experienced and comfortable in dealing with children in the sleep laboratory environment that is child-friendly; and (d) when the sleep specialist provides specific guidance and recommendations in advance of the PSG with regard to the child's care during the night.

Unattended testing outside the sleep laboratory in children has been used predominantly in research settings. There is a paucity of research comparing it to traditional in-laboratory attended sleep studies or other objective clinical outcomes, and there are insufficient data upon which to base reliable clinical recommendations for children at this time.

Table 1—Levels of evidence[5]

Level	Description
1	Evidence provided by a **prospective** study in a **broad spectrum** of persons with the suspected condition, using a **reference (gold) standard** for case definition, where 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 1 studies are judged to have a low risk of bias.
2	Evidence provided by a **prospective** study of a **narrow spectrum** of persons with the suspected condition, or a **well designed retrospective** study of a **broad spectrum** of persons with an established condition (by **"gold standard"**) compared to a **broad spectrum of controls**, where test is applied in a **blinded** evaluation, and enabling the assessment of appropriate tests of diagnostic accuracy. Level 2 studies are judged to have a moderate risk of bias.
3	Evidence provided by a **retrospective** study where either person with the established condition or controls are of a **narrow spectrum**, and where **the reference standard, if not objective, is applied by someone other than the person that performed (interpreted) the test**. Level 3 studies are judged to have a moderate to high risk of bias.
4	Any study design where **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 4 studies are judged to have a very high risk of bias.

Table 2—AASM levels of recommendations

Term	Definition
Standard	This is a generally accepted patient-care strategy that reflects a high degree of clinical certainty and generally implies the use of Level 1 evidence or overwhelming Level 2 evidence.
Guideline	This is a patient-care strategy that reflects a moderate degree of clinical certainty and implies the use of Level 2 evidence or a consensus of Level 3 evidence.
Option	This is a patient-care strategy that reflects uncertain clinical use and implies inconclusive or conflicting evidence or conflicting expert opinion.

Adapted from Eddy[6]

3.2 Diagnostic Indications for Polysomnography in Sleep Related Breathing Disorders

3.2.1 Polysomnography is indicated when the clinical assessment suggests the diagnosis of obstructive sleep apnea syndrome in children. [Review Section 4.2.1] (Standard)

3.2.2 Polysomnography is indicated when the clinical assessment suggests the diagnosis of congenital central alveolar hypoventilation syndrome or sleep related hypoventilation due to neuromuscular disorders or chest wall deformities. It is indicated in selected cases of primary sleep apnea of infancy. (Guideline)

Obstructive sleep apnea syndrome should be diagnosed based upon clinical and polysomnographic criteria. This parameter is based on an extensive literature review conducted by the Indications for PSG in Children task force.

The task force found that clinical evaluation alone does not have sufficient sensitivity or specificity to establish a diagnosis of OSAS. Clinical parameters such as history [4.2.1.1.1, 4.2.1.1.3, 4.2.1.1.4.1], physical examination [4.2.1.1.5], audio or visual recordings [4.2.1.1.2], and standardized questionnaires [4.2.1.1.3] did not consistently identify the presence or absence of OSAS when compared with PSG. Snoring and other nocturnal symptoms in 2 Level 1,[8,9] 4 Level 2,[10-13] 11 Level 3,[14-24] and 18 Level 4[25-42] studies showed inconsistent correlations with respiratory parameters of PSG. Physical features of children evaluated in 11 papers (2 Level 2[11,13]; 3 Level 3[23,24,43]; 6 Level 4)[25,40,41,44-46] showed variable strengths of association with respiratory PSG parameters with obesity demonstrating the strongest association (see below). Polysomnographic parameters correlated best with a combination of multiple signs and symptoms rather than for any individual parameter.[13,24] Audio or video recordings correlated with PSG respiratory parameters, but 2 Level 2[11,47] and 2 Level 3[48,49] studies showed they were not sufficient to establish a diagnosis of OSAS. Pediatric sleep questionnaires were evaluated in 9 articles in children referred for snoring, including 2 with Level 2 evidence,[10,50] 3 with Level 3 evidence,[14,51] and 4 with Level 4 evidence.[27,34,37,41] The 2 Level 2 studies showed low sensitivities for questionnaires to predict polysomnographic evidence of OSAS with better specificities but were still insufficient to differentiate primary snoring from OSAS. Two of the 3 level 3 studies and all of the level 4 studies showed insufficient correlation between sleep questionnaires and PSGs.

The task force found that PSG in children is a reliable and valid measure of the presence of OSAS. Test-retest reliability, or consistency of results across multiple nights of polysomnographic testing, was demonstrated in 1 Level 2,[52] 2 Level 3,[18,53] and 1 Level 4[54] studies. There were no studies designed to test interrater reliability for scoring PSGs in children. Test-retest validity for PSG, or movement of various PSG parameters in the

expected direction after a therapeutic intervention for OSAS, was robust as demonstrated in all 45 studies [4.2.1.1.10].

Convergent validity, or whether PSG and an independent measure both change consistently in the presence of OSAS, was extensively evaluated, including objective [4.2.1.1.4.2] and subjective sleepiness [4.2.1.1.4.1], radiographic evaluation [4.2.1.1.6], neurocognitive and psychological evaluation [4.2.1.1.7], blood pressure [4.2.1.1.8], and quality of life measures [4.2.1.1.9]. Some, but not all, of these measures demonstrated convergent validity with PSG.

Additionally, there was clinical consensus by experts that the PSG is necessary to diagnose congenital central alveolar hypoventilation syndrome, sleep related hypoventilation due to chest wall disorders, and sleep related hypoventilations due to neuromuscular disorder, for which there was also low level evidence.[55-63] There was also clinical consensus by experts that PSG is necessary in selected cases to diagnose primary sleep apnea of infancy when other medical disorders have been ruled out. Patients with CCHS and primary sleep apnea of infancy often present as neonates in the Neonatal Intensive Care Unit, where PSG may not be available. These recommendations are consistent with the recommendations outlined in the ICSD-2 Manual.[64]

Lastly, the task force identified certain conditions associated with an elevated prevalence of SRBD including OSAS. The clinician should consider PSG in children with the following conditions if there is even the slightest suspicion of SRBD. A high prevalence of SRBD has been reported in children with obesity[13,65-71] (13%[67] to 78%[65]) [4.2.2.1], Down syndrome[37,72-74] (57%[37] to 100%[74]) ([4.2.2.8.1.1], Prader-Willi syndrome[75-80] (93%[75]) [4.2.2.8.1.2], neuromuscular disorders[55-63] (53% in one study of children with Duchenne Muscular Dystrophy[56]) [4.2.2.8.4], Chiari malformations and myelomeningocele[81-84] (60% in one series of children with CM[84]) [4.2.2.8.4], and craniofacial anomalies that obstruct the upper airway[32,38,85-87] (48% in children with achondroplasia,[85] 76% in a group of infants with Pierre Robin sequence,[86] and 50% to 91% in 2 small series of craniofacial dysostoses[32,87]) [4.2.2.8.2]. Obesity was identified as an independent risk factor for having SRBD. Furthermore, SRBD was identified as an independent risk factor for hypertension,[20,88-96] suggesting that the clinician should evaluate children who present with hypertension for the possibility of underlying SRBD.

Children in the following categories have an intermediate level of risk for OSAS: those with history of prematurity[25,97-99] (prevalence of OSAS of 7.3%)[97] [4.2.2.2], African American race[34,100-102] (conflicting data) [4.2.2.3], family history of SRBD[102,103] (sparse data) [4.2.2.4], and allergic rhinitis[101,102,104] (not well defined) [4.2.2.5]. Children in these intermediate risk groups ought to be queried for signs or symptoms of SRBDs, and should undergo PSG if present.

Overall, the above data on SRBD prevalence and disease associations included 1 Level 1,[65] 6 Level 2,[13,86,88-90,100] 17 Level 3,[20,57,60,61,66,67,70,76,84,85,91-93,95,97,98,102] and 32 Level 4 studies.[25,32,34,37,38,55,56,58,59,62,63,68,69,71-75,77-83,87,94,96,99,101,103,104]

In summary, high quality data demonstrate that PSG is indicated for the diagnosis of OSAS in children both because clinical parameters alone lack reliability to correctly classify disease and because PSG is a valid and reliable clinical tool. PSG is also indicated for the diagnosis of sleep related hypoventilation due to neuromuscular or chest wall deformities and congenital central alveolar hypoventilation syndrome and in selected cases of primary sleep apnea of infancy, with the realization that some of these patients will be in a NICU and not have access to polysomnography; these conclusions are based less on evidence than on a consensus of expert opinion. Children should be diagnosed with SRBDs by an integration of clinical evaluation and PSG. In addition, certain clinical conditions, as delineated above, are associated with a high prevalence of SRBD. These patients should be screened for symptoms and signs of SRBD and undergo PSG if present.

3.2.3 Nap (abbreviated) polysomnography is not recommended for the evaluation of obstructive sleep apnea syndrome in children. [Review Section 4.2.1.3] (Option)

Three Level 4 studies[74,105,106] in children demonstrated that nap PSG is not as reliable as overnight PSG for identifying SRBD [4.2.1.3] and underestimated the prevalence and severity of SRBD, with sensitivities ranging from 69% to 75% and specificities from 60% to 100%. This recommendation is based on these limited studies and collective expert opinion using the Rand/UCLA Appropriateness Method.

3.2.4 Polysomnography is indicated when there is clinical evidence of a sleep related breathing disorder in infants who have experienced an apparent life-threatening event (ALTE). [Review Sections 4.2.4.4, 4.2.4.6] (Guideline)

Four Level 2,[52,107-109] 8 Level 3,[22,110-116] and 3 Level 4 studies[35,117,118] [4.2.4.4, 4.2.4.6] showed subtle, nonspecific PSG abnormalities in some infants who had experienced an ALTE. The PSG findings were not predictive of recurrence of ALTE. Studies of infants who eventually succumbed to sudden infant death syndrome (SIDS) (4 Level 3)[22,111,113,114] demonstrated PSG abnormalities that were neither sufficiently distinctive nor predictive to support routine use of PSG for children at risk for SIDS. Finally, 2 low level studies[109,116]showed that infants who had experienced an ALTE may be at increased risk to develop SRBD. These infants, however, had other risk factors for SRBD, such as a family history of SRBD or facial dysmorphology. Clinical suspicion of SRBD in a patient with ALTE should prompt consideration of PSG.

3.3 Indications for Preoperative Polysomnography

3.3.1 Polysomnography is indicated in children being considered for adenotonsillectomy to treat obstructive sleep apnea syndrome. [Review Section 4.2.3] (Guideline)

Adenotonsillectomy (AT) is commonly performed as a first-line treatment of OSAS in children, yet the diagnosis of OSAS is often based on clinical parameters alone.[119] The task force reviewed the literature to determine the clinical utility of PSG to confirm the diagnosis of OSAS prior to AT. Whether or not a PSG prior to AT has clinical utility is important for some of the following issues (see review paper for a more complete discussion):

(1) AT is a surgical procedure with a small risk of hemorrhage, infection, upper airway compromise, and pain, and should only be performed if necessary.

(2) Clinical parameters may be unreliable for predicting OSAS.

(3) Children with certain medical disorders are at higher surgical risk (e.g., sickle cell anemia, HIV, coagulopathies, congenital heart disease).

(4) Children with severe OSAS have a higher risk for certain postoperative complications including respiratory compromise.

The task force identified 30 papers pertaining to the clinical utility of PSG prior to AT. Twenty-three of these papers were referenced previously as they demonstrated test-retest validity of PSG in the setting of AT.

First the task force reviewed the literature to determine whether PSG prior to AT correlated with symptoms or physical features of SRBD. As detailed above, history and physical examination were not always reliable predictors of SRBD in children [4.2.1.1.1, 4.2.1.1.3, 4.2.1.1.4.1, 4.2.1.1.3, 4.2.1.1.5]. Three Level 3 papers[18,23,43] showed the limitations of history and physical examination for diagnosing OSAS in children specifically scheduled for AT. In 1 Level 2[11] and 2 Level 4[26,41] studies, the authors concluded that a negative PSG did not rule out clinically significant SRBD that may respond to AT; however, all 3 demonstrate that as one changes the cut-off of a polysomnographic definition of OSAS to be more in-line with the recommendation in the ICSD-2, the percentage of PSG-positive subjects increases, improving the clinical utility of PSG.

To further establish clinical utility of PSG prior to AT, the task force reviewed the literature to determine whether PSG identified those children likely to develop perioperative complications. Ten papers addressed the clinical utility of PSG for assessment of perioperative risk of respiratory complications related to AT in children. One Level 2,[120] 1 Level 3,[121] and 4 Level 4 studies[122-125] showed a positive correlation between PSG measures of OSAS severity and postoperative respiratory complications. In 1 study, respiratory complications occurred as late as the first postoperative night[124] and in another, as long as 14 hours[125]after surgery. In 2 Level 4 studies,[126,127] children without significant comorbid diseases who had no or mild abnormalities on preoperative PSG were likely to have an uncomplicated postoperative course. Although in 2 Level 4 studies[30,128] preoperative PSG did not identify those who later developed perioperative complications, the majority of the evidence supports the clinical utility of preoperative PSG for predicting which children are at risk for postoperative respiratory compromise or prolonged stay and will need postoperative monitoring.

Lastly, to establish clinical utility of PSG prior to AT the task force examined whether the PSG could predict which children were more likely to have residual OSAS following surgery. Two Level 2 studies[129,130] demonstrated that children with higher preoperative AHIs had a greater likelihood of residual OSAS following AT.

In summary, as delineated in practice parameter 3.2.1, PSG is indicated for the diagnosis of SRBD in children. Logically it follows that PSG is indicated to diagnose OSAS prior to AT when OSAS is the primary indication for AT. There are data indicating that history and physical examination alone have limitations for predicting OSAS and that the preoperative AHI can guide the physician in perioperative and postoperative management. At this time, the task force was unable to identify prospective studies addressing whether clinical outcomes following AT are better when PSG is routinely performed prior to AT. The evidence taken as an aggregate indicates that preoperative PSG has strong clinical utility prior to AT for treatment of OSAS.

3.4 Indications for Polysomnography to Assess Response to Treatment

3.4.1 Children with mild obstructive sleep apnea syndrome preoperatively should have clinical evaluation following adenotonsillectomy to assess for residual symptoms. If there are residual symptoms of obstructive sleep apnea syndrome, polysomnography should be performed. [Review Section 4.2.3] (Standard)

This recommendation is a logical extension of parameter 3.2.1. There are Level 2 and 3 studies documenting a high prevalence of residual OSAS after AT. In 1 Level 2 study, residual OSAS was detected in as many as 75% of non-obese children,[131] and in 1 Level 3 study,[132] 75% of a combined obese and non-obese pediatric population had residual OSAS following AT for OSAS. Although many patients had some degree of residual obstruction, most had a considerable reduction in the severity of the OSAS (postoperative results in the 2 studies above showed 90%[131] and 71%[132] of the children had a postoperative OAHI < 5). In 1 Level 2 study,[130] which excluded obese children, the rate of residual polysomnographic evidence of OSAS was 10% to 28% (depending on the criteria used). Another Level 2 study found that OSAS can recur 1 year postoperatively, so patients should be reevaluated periodically.[129] Given the high prevalence of residual obstruction, clinicians should evaluate children postoperatively for symptoms of OSAS and if present, a PSG should be performed.

3.4.2 Polysomnography is indicated following adenotonsillectomy to assess for residual sleep related breathing disorder in children with preoperative evidence for moderate to severe OSAS, obesity, craniofacial anomalies that obstruct the upper airway, and neurologic disorders (e.g., Down syndrome, Prader-Willi syndrome, and myelomeningocele). [Review Section 4.2.3] (Standard)

Incomplete resolution of OSAS was more common in children with high preoperative AHI (exact cut-off not established as norms and statistical methods varied from study to study) (2 Level 2,[129,130] 1 Level 3[132]), obesity (2 Level 2,[129,130] 1 Level 3,[132] 3 Level 4[101,133,134]), craniofacial anomalies that obstruct the upper airway (1 Level 3[135] and 1 Level 4[38]), and neurologic disorders such as myelomeningocele (1 level 4[81]) and Down syndrome (1 Level 4[42]), while absence of obesity and craniofacial abnormalities (2 Level 3,[132,136] 1 Level 4[137]) and lower AHI (2 Level 2,[129,130] 1 Level 3[132]) favored a good outcome. Children with neuromuscular disorders can have a mix of both obstructive and central sleep apnea and require PSG following AT to determine the need for CPAP or NIPPV as demonstrated in 2 level 4[81,83] studies. This recommendation is a logical extension of 3.2.1. These populations are at increased risk for having residual SRBD and are often difficult to reliably evaluate clinically.

3.4.3 Polysomnography is indicated after treatment of children for obstructive sleep apnea syndrome with rapid maxillary expansion

to assess for the level of residual disease and to determine whether additional treatment is necessary. [Review Section 4.4.3] (Option)

This is a rarely performed treatment for OSAS in children with only limited data regarding efficacy. Two low level studies evaluated children following rapid maxillary expansion (RME). Significant residual disease remained after RME in 1 Level 3[138] and 1 Level 4 study[139] [4.4.3]. PSG was useful to establish residual disease and the efficacy of staged treatment. Given the paucity of evidence demonstrating the efficacy for this new procedure, it is recommended that children treated with rapid maxillary expansion have follow-up PSG. This recommendation is based on consensus expert opinion using the Rand/UCLA Appropriateness Method.

3.4.4 Children with OSAS treated with an oral appliance should have clinical follow-up and polysomnography to assess response to treatment. [Review Section 4.4.3] (Option)

This is a relatively new treatment for OSAS in children with limited studies available. One Level 2[86] and 1 Level 4 study[40] used PSG to demonstrate clinical efficacy of dental devices in children [4.4.3]. Given the paucity of evidence demonstrating the efficacy for this new procedure, it is recommended that children treated with a dental device have follow-up PSG. This recommendation is based on consensus expert opinion using the Rand/UCLA Appropriateness Method.

3.4.5 Polysomnography is indicated for positive airway pressure (PAP) titration in children with obstructive sleep apnea syndrome (Standard)

3.4.6 Polysomnography is indicated for noninvasive positive pressure ventilation (NIPPV) titration in children with other sleep related breathing disorders. [Review Section 4.4.1] (Option)

PSG was shown to be a valid measure to assess response to therapy for SRBD. There were insufficient data on autotitration PAP (APAP) in children to assess this mode of therapy. Four papers (1 Level 2,[140] 3 Level 4[141-143]) demonstrated that PSG was useful to determine optimal PAP settings in children and infants. It was particularly important for children at risk for multiple types of SRBD, such as those with neurologic disorders. Five studies (1 Level 3[57] and 4 Level 4[56,144-146]) evaluated or described the use of PSG for titration of nocturnal intermittent positive pressure ventilation (NIPPV) in children with SRBD and neuromuscular disorders. These studies demonstrated either clinical or physiologic improvement with NIPPV that was titrated using PSG.

3.4.7 Follow-up PSG in children on chronic PAP support is indicated to determine whether pressure requirements have changed as a result of the child's growth and development, if symptoms recur while on PAP, or if additional or alternate treatment is instituted. [Review Section 4.4.2] (Guideline)

Growth and development should be considered in children on chronic PAP treatment as symptoms may resolve or worsen with significant changes in the child's morphology. One Level 4 study[146] [4.4.2] demonstrated that many children required pressure changes (66%) in PAP level, even in the absence of interventions, suggesting that PAP requirements might change with growth and development. Clinical judgment should be used to determine the timing and frequency of follow-up PSG. This recommendation is a generally accepted patient care strategy.

3.4.8 Children treated with mechanical ventilation may benefit from periodic evaluation with polysomnography to adjust ventilator settings. [Review Section 4.4.5] (Option)

PSG is useful for assessing optimal ventilator settings since respiration worsens during sleep and wake ventilator settings may not be adequate for sleep. There were no studies addressing the use of PSG in the management of ventilator settings that met inclusion criteria. This recommendation is based on consensus expert opinion using the Rand/UCLA Appropriateness Method.

3.4.9 Children considered for treatment with supplemental oxygen do not routinely require polysomnography for management of oxygen therapy. [Review Section 4.4.6] (Option)

Nocturnal oximetry is generally sufficient to assess adequate oxygenation. However, PSG can be useful for titrating supplemental oxygen in patients who may hypoventilate and may be at risk for developing central apneas or hypercarbia with supplemental oxygen. There were no studies addressing this topic that met inclusion criteria. This recommendation is based on consensus expert opinion using the Rand/UCLA Appropriateness Method.

3.4.10 Children treated with tracheostomy for sleep related breathing disorders benefit from polysomnography as part of the evaluation prior to decannulation. These children should be followed clinically after decannulation to assess for recurrence of symptoms of sleep related breathing disorders. [Review Section 4.4.4] (Option)

One Level 3 study[147] demonstrated clinical usefulness of PSG as part of the evaluation to assess readiness for decannulation in children with long-term tracheostomy [4.4.4]. This recommendation is based on this study and on consensus expert opinion using the Rand/UCLA Appropriateness Method.

3.5 Indications for Polysomnography in Respiratory Diseases

3.5.1 Polysomnography is indicated in the following respiratory disorders only if there is a clinical suspicion for an accompanying sleep related breathing disorder: chronic asthma, cystic fibrosis, pulmonary hypertension, bronchopulmonary dysplasia, or chest wall abnormality such as kyphoscoliosis. [Review Sections 4.3.1.1 and 4.3.1.2] (Option)

In children with asthma, prevalence studies showed conflicting low level evidence for increased risk of SRBD ([4.3.1.1][102,148]). The data were too sparse to assess the clinical utility of PSG in children with cystic fibrosis, although 1 Level 4 study[149] demonstrated clinical utility of PSG to initiate and titrate noninvasive ventilation [4.3.1.2]. There were no studies addressing the clinical utility of PSG in children with unexplained pulmonary hypertension [4.2.2.7] or bronchopulmonary dysplasia [4.3.1.3]. Finally, a low level study[150] of children with kyphoscoliosis who could not undergo PFTs did not find statistically significant evidence that preoperative polysomnography could predict those at risk for prolonged postoperative ventilation after scoliosis repair [4.3.2.1]. As with all children, those with asthma, cystic fibrosis, unexplained pulmonary hy-

pertension, bronchopulmonary dysplasia, kyphoscoliosis, or chest wall deformity for whom there is a clinical suspicion of a SRBD should undergo PSG.

4.0 FUTURE RESEARCH

Improvement in diagnostic accuracy and clinical utility of PSG for evaluation of SRBD in children will require investigations that address a wide range of pediatric populations using adequate sample sizes and standardized methodology. Study designs should account for changes in respiratory physiology by age and maturation and should avoid bias through inclusion of a broad spectrum of subjects with a range of severity from normal to severe disease. Development of definitive and well-validated normative and pathological respiratory PSG values will help refine diagnosis and possibly treatment options in children with SRBD. Improved research design and methodology will also improve understanding of the earliest forms of SRBD and the natural history of SRBD in children.

There is an urgent need for research clarifying the clinical utility of polysomnography. For example, comparison of validated and relevant outcomes in patients managed with and without PSG would provide the most compelling evidence to evaluate the value of PSG in managing children with suspected SRBDs.

Findings indicate that PSG may be particularly useful in the evaluation of children with chronic health conditions such as obesity, metabolic syndrome, overt or evolving hypertension, and other conditions associated with cardiovascular risk. There is a need for further investigation of SRBD in children with neurodevelopmental and neuromuscular disorders, sickle cell anemia, craniofacial disorders, and certain infants who experience ALTEs.

The clinical utility and cost effectiveness of testing outside the sleep laboratory for certain groups of children with suspected SRBD needs further investigation. Preliminary evidence suggests that efforts to make the sleep laboratory experience more child-friendly will improve the quality of data collected as well as patient and family satisfaction.

Pediatric sleep medicine is a complex, dynamic, and multidisciplinary field, and the sleep specialist involved with evaluation and management of children faces an evolving landscape. The medical literature regarding pediatric PSG is expanding exponentially, and recording techniques and methods for digital analysis are changing at a rapid pace. Superimposed on these dynamic changes is the challenge of characterizing respiratory sleep disorders and the impact of SRBD on behavior and cognition in the developing child. Performance of PSG using standardized and developmentally appropriate methods has strong clinical utility in the diagnosis and management of SRBD in children. Accurate diagnosis and management of SRBD in children is best accomplished through careful integration of polysomnographic findings with the clinical evaluation.

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REFERENCES

1. American Thoracic Society. Standards and Indications for Cardiopulmonary Studies in Children. Am J Respir Crit Care Med 1996;153:866-78.
2. American Academy of Pediatrics. Clinical practice guideline: Diagnosis and management of childhood obstructive sleep apnea syndrome. Pediatrics 2002;109:704-12.
3. Aurora RN, Morgenthaler T. On the Goodness of Recommendations: The Changing Face of Practice Parameters. Sleep In press.
4. Wise MS, Nichols CD, Grigg-Damberger MM, et al. Respiratory Indications for Polysomnography in Children: An evidence-based review. Sleep 2010;TBD:TBD.
5. Edlund W, Gronseth G, So Y, Franklin G. Clinical Practice Guideline Process Manual. 4th ed. St. Paul, MN: American Academy of Neurology, 2005.
6. Eddy DM, American College of P. A manual for assessing health practices & designing practice policies : the explicit approach. Philadelphia, Pa.: American College of Physicians, 1992.
7. Iber C, Ancoli-Israel S, Chesson A, Quan SF, for the American Academy of Sleep Medicine. The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications, 1st ed. Westchester, IL: American Academy of Sleep Medicine, 2007.
8. Masters IB, Harvey JM, Wales PD, O'Callaghan MJ, Harris MA. Clinical versus polysomnographic profiles in children with obstructive sleep apnoea. J Paediatr Child Health 1999;35:49-54.
9. Chervin RD, Weatherly RA, Ruzicka DL, et al. Subjective sleepiness and polysomnographic correlates in children scheduled for adenotonsillectomy vs other surgical care. Sleep 2006;29:495-503.
10. Goodwin JL, Kaemingk KL, Fregosi RF, et al. Parasomnias and sleep disordered breathing in Caucasian and Hispanic children - the Tucson children's assessment of sleep apnea study. BMC Med 2004;2:14.
11. Goldstein NA, Pugazhendhi V, Rao SM, et al. Clinical assessment of pediatric obstructive sleep apnea. Pediatrics 2004;114:33-43.
12. Goodwin JL, Kaemingk KL, Fregosi RF, et al. Clinical outcomes associated with sleep-disordered breathing in Caucasian and Hispanic children--the Tucson Children's Assessment of Sleep Apnea study (TuCASA). Sleep 2003;26:587-91.
13. Wing YK, Hui SH, Pak WM, et al. A controlled study of sleep related disordered breathing in obese children. Arch Dis Child 2003;88:1043-7.
14. Brouillette R, Hanson D, David R, et al. A diagnostic approach to suspected obstructive sleep apnea in children. The Journal of Pediatrics 1984;105:10-4.
15. Carroll JL, McColley SA, Marcus CL, Curtis S, Loughlin GM. Inability of clinical history to distinguish primary snoring from obstructive sleep apnea syndrome in children. Chest 1995;108:610-8.
16. Goldbart AD, Krishna J, Li RC, Serpero LD, Gozal D. Inflammatory mediators in exhaled breath condensate of children with obstructive sleep apnea syndrome. Chest 2006;130:143-8.
17. Kaditis AG, Finder J, Alexopoulos EI, et al. Sleep-disordered breathing in 3,680 Greek children. Pediatr Pulmonol 2004;37:499-509.
18. Nieminen P, Tolonen U, Lopponen H. Snoring and obstructive sleep apnea in children: a 6-month follow-up study. Arch Otolaryngol Head Neck Surg 2000;126:481-6.
19. Pagel JF, Snyder S, Dawson D. Obstructive sleep apnea in sleepy pediatric psychiatry clinic patients: polysomnographic and clinical correlates. Sleep Breath 2004;8:125-31.
20. Reade EP, Whaley C, Lin JJ, McKenney DW, Lee D, Perkin R. Hypopnea in pediatric patients with obesity hypertension. Pediatr Nephrol 2004;19:1014-20.
21. Rimell FL, Rosen G, Garcia J. Full polysomnographic evaluation of the infant airway. Arch Otolaryngol Head Neck Surg 1998;124:773-6.
22. Sawaguchi T, Franco P, Kato I, et al. From epidemiology to physiology and pathology: apnea and arousal deficient theories in sudden infant death syndrome (SIDS)--with particular reference to hypoxic brainstem gliosis. Forensic Sci Int 2002;130 Suppl:S21-9.

23. Wang RC, Elkins TP, Keech D, Wauquier A, Hubbard D. Accuracy of clinical evaluation in pediatric obstructive sleep apnea. Otolaryngol Head Neck Surg 1998;118:69-73.
24. Xu Z, Cheuk DK, Lee SL. Clinical evaluation in predicting childhood obstructive sleep apnea. Chest 2006;130:1765-71.
25. Greenfeld M, Tauman R, DeRowe A, Sivan Y. Obstructive sleep apnea syndrome due to adenotonsillar hypertrophy in infants. Int J Pediatr Otorhinolaryngol 2003;67:1055-60.
26. Guilleminault C, Winkle R, Korobkin R, Simmons B. Children and nocturnal snoring: evaluation of the effects of sleep related respiratory resistive load and daytime functioning. Eur J Pediatr 1982;139:165-71.
27. Mallory GB, Jr., Fiser DH, Jackson R. Sleep-associated breathing disorders in morbidly obese children and adolescents. J Pediatr 1989;115:892-7.
28. Marcus CL, Hamer A, Loughlin GM. Natural history of primary snoring in children. Pediatr Pulmonol 1998;26:6-11.
29. Nanaware SK, Gothi D, Joshi JM. Sleep apnea. Indian J Pediatr 2006;73:597-601.
30. Pang KP, Balakrishnan A. Paediatric obstructive sleep apnoea: is a polysomnogram always necessary? J Laryngol Otol 2004;118:275-8.
31. Pavone M, Paglietti MG, Petrone A, Crino A, De Vincentiis GC, Cutrera R. Adenotonsillectomy for obstructive sleep apnea in children with Prader-Willi syndrome. Pediatr Pulmonol 2006;41:74-9.
32. Pijpers M, Poels PJ, Vaandrager JM, et al. Undiagnosed obstructive sleep apnea syndrome in children with syndromal craniofacial synostosis. J Craniofac Surg 2004;15:670-4.
33. Preutthipan A, Suwanjutha S, Chantarojanasiri T. Obstructive sleep apnea syndrome in Thai children diagnosed by polysomnography. Southeast Asian J Trop Med Public Health 1997;28:62-8.
34. Rosen CL. Clinical features of obstructive sleep apnea hypoventilation syndrome in otherwise healthy children. Pediatr Pulmonol 1999;27:403-9.
35. Rosen CL, Frost JD, Jr., Harrison GM. Infant apnea: polygraphic studies and follow-up monitoring. Pediatrics 1983;71:731-6.
36. Sanchez-Armengol A, Fuentes-Pradera MA, Capote-Gil F, et al. Sleep-related breathing disorders in adolescents aged 12 to 16 years : clinical and polygraphic findings. Chest 2001;119:1393-400.
37. Shott SR, Amin R, Chini B, Heubi C, Hotze S, Akers R. Obstructive sleep apnea: Should all children with Down syndrome be tested? Arch Otolaryngol Head Neck Surg 2006;132:432-6.
38. Sisk EA, Heatley DG, Borowski BJ, Leverson GE, Pauli RM. Obstructive sleep apnea in children with achondroplasia: surgical and anesthetic considerations. Otolaryngol Head Neck Surg 1999;120:248-54.
39. Southall DP, Talbert DG, Johnson P, et al. Prolonged expiratory apnoea: a disorder resulting in episodes of severe arterial hypoxaemia in infants and young children. Lancet 1985;2:571-7.
40. Villa MP, Bernkopf E, Pagani J, Broia V, Montesano M, Ronchetti R. Randomized controlled study of an oral jaw-positioning appliance for the treatment of obstructive sleep apnea in children with malocclusion. Am J Respir Crit Care Med 2002;165:123-7.
41. Weatherly RA, Ruzicka DL, Marriott DJ, Chervin RD. Polysomnography in children scheduled for adenotonsillectomy. Otolaryngol Head Neck Surg 2004;131:727-31.
42. Wiet GJ, Bower C, Seibert R, Griebel M. Surgical correction of obstructive sleep apnea in the complicated pediatric patient documented by polysomnography. Int J Pediatr Otorhinolaryngol 1997;41:133-43.
43. Shatz A. Indications and outcomes of adenoidectomy in infancy. Ann Otol Rhinol Laryngol 2004;113:835-8.
44. Lam YY, Chan EYT, Ng DK, et al. The correlation among obesity, apnea-hypopnea index, and tonsil size in children. Chest 2006;130:1751-6.
45. Tauman R, O'Brien LM, Ivanenko A, Gozal D. Obesity rather than severity of sleep-disordered breathing as the major determinant of insulin resistance and altered lipidemia in snoring children. Pediatrics 2005;116:e66-73.
46. Zhang XW, Li Y, Zhou F, Guo CK, Huang ZT. Comparison of polygraphic parameters in children with adenotonsillar hypertrophy with vs without obstructive sleep apnea. Arch Otolaryngol Head Neck Surg 2007;133:122-6.
47. Lamm C, Mandeli J, Kattan M. Evaluation of home audiotapes as an abbreviated test for obstructive sleep apnea syndrome (OSAS) in children. Pediatr Pulmonol 1999;27:267-72.
48. Sivan Y, Kornecki A, Schonfeld T. Screening obstructive sleep apnoea syndrome by home videotape recording in children. Eur Respir J 1996;9:2127-31.
49. Jacob SV, Morielli A, Mograss MA, Ducharme FM, Schloss MD, Brouillette RT. Home testing for pediatric obstructive sleep apnea syndrome secondary to adenotonsillar hypertrophy. Pediatr Pulmonol 1995;20:241-52.
50. Chau KW, Ng DK, Kwok KL, et al. Application of videotape in the screening of obstructive sleep apnea in children. Sleep Med 2008;9:442-5.
51. Chervin RD, Weatherly RA, Garetz SL, et al. Pediatric sleep questionnaire: prediction of sleep apnea and outcomes. Arch Otolaryngol Head Neck Surg 2007;133:216-22.
52. Rebuffat E, Groswasser J, Kelmanson I, Sottiaux M, Kahn A. Polygraphic evaluation of night-to-night variability in sleep characteristics and apneas in infants. Sleep 1994;17:329-32.
53. Katz ES, Greene MG, Carson KA, et al. Night-to-night variability of polysomnography in children with suspected obstructive sleep apnea. J Pediatr 2002;140:589-94.
54. Li AM, Wing YK, Cheung A, et al. Is a 2-night polysomnographic study necessary in childhood sleep-related disordered breathing? Chest 2004;126:1467-72.
55. Smith PE, Calverley PM, Edwards RH. Hypoxemia during sleep in Duchenne muscular dystrophy. Am Rev Resp Dis 1988;137:884-8.
56. Suresh S, Wales P, Dakin C, Harris MA, Cooper DG. Sleep-related breathing disorder in Duchenne muscular dystrophy: disease spectrum in the paediatric population. J Paediatr Child Health 2005;41:500-3.
57. Mellies U, Dohna-Schwake C, Stehling F, Voit T. Sleep disordered breathing in spinal muscular atrophy. Neuromuscul Disord 2004;14:797-803.
58. Quera Salva MA, Blumen M, Jacquette A, et al. Sleep disorders in childhood-onset myotonic dystrophy type 1. Neuromuscul Disord 2006;16:564-70.
59. Santamaria F, V. AM, Parenti G, et al. Upper airway obstructive disease in mucopolysaccharidoses: polysomnography, computed tomography and nasal endoscopy findings. J Inherit Metab Dis 2007;30:743-9.
60. McGrath-Morrow SA, Sterni LM, McGinley B, Lefton-Grief MA, Rosquist K, Lederman H. Polysomnographic values in adolescents with ataxia telangiectasia. Pediatr Pulmonology 2008;43:674-9.
61. Khan Y, Heckmatt J. Obstructive apnoeas in Duchenne muscular dystrophy. Thorax 1994;49:157-61.
62. Barbe F, Quera-Salva MA, McCann C, et al. Sleep-related respiratory disturbances in patients with Duchenne muscular dystrophy. Eur Respir J 1994;7:1403-8.
63. Kerr S, Kohrman M. Polysomnographic abnormalities in Duchenne Muscular Dystrophy. J Child Neurol 1994;9:332-4.
64. American Academy of Sleep Medicine. International classification of sleep disorders, 2nd ed.: Diagnositic and coding manual. Westchester, IL: American Academy of Sleep Medicine, 2005.
65. Xu Z, Jiaqing A, Yuchan L, Shen K. A case-control study of obstructive sleep apnea-hypopnea syndrome in obese and nonobese chinese children. Chest 2008;133:684-9.
66. Kohler M, Lushington K, Couper R, et al. Obesity and risk of sleep related upper airway obstruction in Caucasian children. J Clin Sleep Med 2008;4:129-36.
67. Beebe DW, Lewin D, Zeller M, et al. Sleep in overweight adolescents: Shorter sleep, poorer sleep quality, sleepiness, and sleep-disordered breathing. J Pediatr Psychol 2007;32:69-79.
68. Marcus CL, Curtis S, Koerner CB, Joffe A, Serwint JR, Loughlin GM. Evaluation of pulmonary function and polysomnography in obese children and adolescents. Pediatr Pulmonol 1996;21:176-83.
69. McKenzie SA, Bhattacharya A, Sureshkumar R, et al. Which obese children should have a sleep study? Respir Med 2008;102:1581-5.
70. Chay OM, Goh A, Abisheganaden J, et al. Obstructive sleep apnea syndrome in obese Singapore children. Pediatr Pulmonol 2000;29:284-90.
71. Kalra M, Inge T, Garcia V, et al. Obstructive sleep apnea in extremely overweight adolescents undergoing bariatric surgery. Obes Res 2005;13:1175-9.
72. Dyken ME, Lin-Dyken DC, Poulton S, Zimmerman MB, Sedars E. Prospective polysomnographic analysis of obstructive sleep apnea in down syndrome. Arch Pediatr Adolesc Med 2003;157:655-60.
73. Levanon A, Tarasiuk A, Tal A. Sleep characteristics in children with Down syndrome. J Pediatr 1999;134:755-60.
74. Marcus CL, Keens TG, Bautista DB, von Pechmann WS, Davidson Ward SL. Obstructive sleep apnea in children with Down syndrome. Pediatrics 1991;88:132-9.
75. Lin HY, Lin SP, Lin CC, et al. Polysomnographic characteristics in patients with Prader-Willi syndrome. Pediatr Pulmonology 2007;42:881-7.

76. Festen DA, de Weerd AW, van den Bossche RA, Joosten K, Hoeve H, Hokken-Koelega AC. Sleep-related breathing disorders in prepubertal children with Prader-Willi syndrome and effects of growth hormone treatment. J Clin Endocrinol Metab 2006;91:4911-5.
77. Festen DA, Wevers M, de Weerd AW, van den Bossche RA, Duivenvoorden HJ, Hokken-Koelega AC. Cognition and behavior in pre-pubertal children with Prader-Willi syndrome and associations with sleep-related breathing disorders. Am J Med Genet A 2008;146A:3018-25.
78. Festen DA, Wevers M, de Weerd AW, et al. Psychomotor Development in Infants with Prader-Willi Syndrome and Associations with Sleep-Related Breathing Disorders. Pediatric Research 2007;62:221-4.
79. Miller J, Silverstein J, Shuster J, Driscoll DJ, Wagner M. Short-term effects of growth hormone on sleep abnormalities in Prader-Willi syndrome. J Clin Endocrinol Metab 2006;91:413-7.
80. Williams K, Scheimann A, Sutton V, Hayslett E, Glaze DG. Sleepiness and sleep disordered breathing in Prader-Willi syndrome: relationship to genotype, growth hormone therapy, and body composition. J Clin Sleep Med 2008;4:111-8.
81. Kirk VG, Morielli A, Gozal D, et al. Treatment of sleep-disordered breathing in children with myelomeningocele. Pediatr Pulmonol 2000;30:445-52.
82. Murray C, Seton C, Prelog K, Fitzgerald DA. Arnold Chiari type 1 malformation presenting with sleep disordered breathing in well children. Arch Dis Child 2006;91:342-3.
83. Waters KA, Forbes P, Morielli A, et. al. Sleep-disordered breathing in children with myelomenigocele. J Pediatr 1998;132:672-81.
84. Dauvilliers Y, Stal V, Abril B, et al. Chiari malformation and sleep related breathing disorders. J Neurol Neurosurg Psychiatry 2007;78:1344-8.
85. Mogayzel PJ, Jr., Carroll JL, Loughlin GM, Hurko O, Francomano CA, Marcus CL. Sleep-disordered breathing in children with achondroplasia. J Pediatr 1998;132:667-71.
86. Buchenau W, Urschitz MS, Sautermeister J, et al. A randomized clinical trial of a new orthodontic appliance to improve upper airway obstruction in infants with Pierre Robin sequence. J Pediatr 2007;151:145-9.
87. Gonsalez S, Thompson D, Hayward R, Lane R. Breathing patterns in children with craniofacial dysostosis and hindbrain herniation. Eur Respir J 1998;11:866-72.
88. Bixler EO, Vgontzas AN, Lin HM, et al. Blood pressure associated with sleep-disordered breathing in a population sample of children. Hypertension 2008;52:841-6.
89. Enright PL, Goodwin JL, Sherrill DL, Quan JR, Quan SF. Blood pressure elevation associated with sleep-related breathing disorder in a community sample of white and hispanic children: The Tucson Children's Assessment of Sleep Apnea Study. Arch Pediatr Adolesc Med 2003;157:901-4.
90. Redline S, Storfer-Isser A, Rosen CL, et al. Association between metabolic syndrome and sleep-disordered breathing in adolescents. Am J Respir Crit Care Med 2007;176:401-8.
91. Leung LCK, Ng DK, Lau MW, et al. Twenty-four-hour ambulatory BP in snoring children with obstructive sleep apnea syndrome. Chest 2006;130:1009-17.
92. Li AM, Au CT, Sung RY, et al. Ambulatory blood pressure in children with obstructive sleep apnoea: a community based study. Thorax 2008;63:803-9.
93. Amin R, Somers V, McConnell K, et al. Activity-adjusted 24-hour ambulatory blood pressure and cardiac remodeling in children with sleep disordered breathing. Hypertension 2008;51:84-91.
94. Marcus CL, Greene MG, Carroll JL. Blood pressure in children with obstructive sleep apnea. Am J Respir Crit Care Med 1998;157:1098-103.
95. Amin RS, Carroll JL, Jeffries JL, et al. Twenty-four-hour ambulatory blood pressure in children with sleep-disordered breathing. Am J Respir Crit Care Med 2004;169:950-6.
96. Kohyama J, Ohinata JS, Hasegawa T. Blood pressue in sleep disordered breathing. Arch Dis Child 2003;88:139-42.
97. Hibbs AM, Johnson NL, Rosen CL, et al. Prenatal and neonatal risk factors for sleep disordered breathing in school-aged children born preterm. J Pediatr 2008;153:176-82.
98. Emancipator JL, Storfer-Isser A, Taylor HG, et al. Variation of cognition and achievement with sleep-disordered breathing in full-term and preterm children. Arch Pediatr Adolesc Med 2006;160:203-10.
99. Paul K, Melichar J, Meletin J, Dittrichova J. Differential diagnosis of apneas in preterm infants. Eur J Pediatr 2009;168:195-201.
100. Stepanski E, Zayyad A, Nigro C, Lopata M, Basner R. Sleep-disordered breathing in a predominantly African-American pediatric population. J Sleep Res 1999;8:65-70.
101. Morton S, Rosen C, Larkin E, Tishler P, Aylor J, Redline S. Predictors of sleep-disordered breathing in children with a history of tonsillectomy and/or adenoidectomy. Sleep 2001;24:823-9.
102. Redline S, Tishler P, Schluchter M, Aylor J, Clark K, Graham G. Risk Factors for Sleep-disordered Breathing in Children. Am J Respir Crit Care Med 1999;159:1527-32.
103. Ovchinsky A, Rao M, Lotwin I, Goldstein NA. The familial aggregation of pediatric obstructive sleep apnea syndrome. Arch Otolaryngol Head Neck Surg 2002;128:815-8.
104. McColley SA, Carroll J, Curtis S, Loughlin GM, Sampson HA. High Prevalence of Allergic Sensitization in Children with Habitual Snoring and Obstructive Sleep Apnea. Chest 1997;111:170-3.
105. Marcus CL, Keens TG, Davidson Ward SL. Comparison of nap and overnight polysomnography in children. Pediatr Pulmonol 1992;13:16-21.
106. Saeed MM, Keens TG, Stabile MW, Bolokowicz J, Davidson Ward SL. Should children with suspected obstructive sleep apnea syndrome and normal nap sleep studies have overnight sleep studies? Chest 2000;118:360-5.
107. Harrington C, Kirjavainen T, Teng A, Sullivan CE. Altered autonomic function and reduced arousability in apparent life-threatening event infants with obstructive sleep apnea. Am J Respir Crit Care Med 2002;165:1048-54.
108. Horemuzova E, Katz-Salamon M, Milerad J. Increased inspiratory effort in infants with a history of apparent life-threatening event. Acta Paediatr 2002;91:280-6.
109. Guilleminault C, Pelayo R, Leger D, Philip P. Apparent life-threatening events, facial dysmorphia and sleep-disordered breathing. Eur J Pediatr 2000;159:444-9.
110. Arad-Cohen N, Cohen A, Tirosh E. The relationship between gastroesophageal reflux and apnea in infants. J Pediatr 2000;137:321-6.
111. Franco P, Szliwowski H, Dramaix M, Kahn A. Decreased autonomic responses to obstructive sleep events in future victims of sudden infant death syndrome. Pediatr Res 1999;46:33-9.
112. Guilleminault C, Souquet M, Ariagno RL, Korobkin R, Simmons FB. Five cases of near-miss sudden infant death syndrome and development of obstructive sleep apnea syndrome. Pediatrics 1984;73:71-8.
113. Kato I, Franco P, Groswasser J, et al. Incomplete arousal processes in infants who were victims of sudden death. Am J Respir Crit Care Med 2003;168:1298-303.
114. Kato I, Groswasser J, Franco P, et al. Developmental characteristics of apnea in infants who succumb to sudden infant death syndrome. Am J Respir Crit Care Med 2001;164:1464-9.
115. Kahn A, Montauk L, Blum D. Diagnostic categories in infants referred for an acute event suggesting near-miss SIDS. Eur J Pediatr 1987;146:458-60.
116. Guilleminault C, Stoohs R. From apnea of infancy to obstructive sleep apnea syndrome in the young child. Chest 1992;102:1065-71.
117. Abreu e Silva F, MacFadyen U, Williams A, Simpson H. Sleep apnoea in infancy. Journal of the Royal Society of Medicine 1985;78:1005-8.
118. Tirosh E, Jaffe M. Apnea of infancy, seizures, and gastroesophageal reflux: an important but infrequent association. J Child Neurol 1996;11:98-100.
119. Weatherly RA, Mai EF, Ruzicka DL, Chervin RD. Identification and evaluation of obstructive sleep apnea prior to adenotonsillectomy in children: a survey of practice patterns. Sleep Med 2003;4:297-307.
120. Sanders JC, King MA, Mitchell RB, Kelly J. Perioperative complications of adenotonsillectomy in children with obstructive sleep apnea syndrome. Anesth Analg 2006;103:1115-21.
121. Wilson K, Lakheeram I, Morielli A, Brouillette R, Brown K. Can assessment for obstructive sleep apnea help predict postadenotonsillectomy respiratory complications? Anesthesiology 2002;96:313-22.
122. Brown KA, Morin I, Hickey C, Manoukian JJ, Nixon GM, Brouillette RT. Urgent adenotonsillectomy: an analysis of risk factors associated with postoperative respiratory morbidity. Anesthesiology 2003;99:586-95.
123. Rosen GM, Muckle RP, Mahowald M, Goding GS, Ullevig C. Postoperative Respiratory Compromise in Children with Obstructive Sleep Apnea Syndrome: Can It Be Anticipated? Pediatrics 1994;93:784-88.
124. Ye J, Liu H, Zhang G, Huang Z, Huang P, Li Y. Postoperative respiratory complications of adenotonsillectomy for obstructive sleep apnea syndrome in older children: Prevalence, risk factors, and impact on clinical outcome. J Otolaryngol Head and Neck Surgery 2008;38:49-58.

125. McColley SA, April MM, Carroll J, Naclerio RM, Loughlin GM. Respiratory Compromise After Adenotonsillectomy in Children with Obstructive Sleep Apnea. Arch Otolaryngol Head Neck Surg 1992;118:940-3.
126. Biavati MJ, Manning SC, Phillips DL. Predictive factors for respiratory complications after tonsillectomy and adenoidectomy in children. Arch Otolaryngol Head Neck Surg 1997;123:517-21.
127. Helfaer MA, McColley SA, Pyzik PL, et al. Polysomnography after adenotonsillectomy in mild pediatric obstructive sleep apnea. Crit Care Med 1996;24:1323-7.
128. Rieder AA, Flanary V. The effect of polysomnography on pediatric adenotonsillectomy postoperative management. Otolaryngol Head Neck Surg 2005;132:263-7.
129. Amin R, Anthony L, Somers V, et al. Growth velocity predicts recurrence of sleep-disordered breathing 1 year after adenotonsillectomy. Am J Respir Crit Care Med 2008;177:654-9.
130. Mitchell RB. Adenotonsillectomy for obstructive sleep apnea in children: outcome evaluated by pre- and postoperative polysomnography. Laryngoscope 2007;117:1844-54.
131. Apostolidou MT, Alexopoulos EI, Chaidas K, et al. Obesity and persisting sleep apnea after adenotonsillectomy in Greek children. Chest 2008;134:1149-55.
132. Tauman R, Gulliver TE, Krishna J, et al. Persistence of obstructive sleep apnea syndrome in children after adenotonsillectomy. J Pediatr 2006;149:803-8.
133. Shine NP, Lannigan FJ, Coates HL, Wilson A. Adenotonsillectomy for obstructive sleep apnea in obese children: effects on respiratory parameters and clinical outcome. Arch Otolaryngol Head Neck Surg 2006;132:1123-7.
134. O'Brien LM, Sitha S, Baur LA, Waters KA. Obesity increases the risk for persisting obstructive sleep apnea after treatment in children. Int J Pediatr Otorhinolaryngol 2006;70:1555-60.
135. Walker P, Whitehead B, Gulliver T. Polysomnographic outcome of adenotonsillectomy for obstructive sleep apnea in children under 5 years old. Otolaryngol Head Neck Surg 2008;139:83-6.
136. Montgomery-Downs HE, Crabtree VM, Gozal D. Cognition, sleep and respiration in at-risk children treated for obstructive sleep apnoea. Eur Respir J 2005;25:336-42.
137. de la Chaux R, Klemens C, Patscheider M, Reichel O, Dreher A. Tonsillectomy in the treatment of obstructive sleep apnea syndrome in children: polysomnography results. Int J Pediatr Otorhinolaryngol 2008;72:1411-7.
138. Villa MP, Malagola C, Pagani J, et al. Rapid maxillary expansion in children with obstructive sleep apnea syndrome: 12-month follow-up. Sleep Med 2007;8:128-34.
139. Pirelli P, Saponara M, Guilleminault C. Rapid maxillary expansion in children with obstructive sleep apnea syndrome. Sleep 2004;27:761-6.
140. Nakra N, Bhargava S, Dzuira J, Caprio S, Bazzy-Asaad A. Sleep-disordered breathing in children with metabolic syndrome: the role of leptin and sympathetic nerve system activity and the effect of continuous positive airway pressure. Pediatrics 2008;122:e634-42.
141. Downey R, 3rd, Perkin RM, MacQuarrie J. Nasal continuous positive airway pressure use in children with obstructive sleep apnea younger than 2 years of age. Chest 2000;117:1608-12.
142. Uong EC, Epperson M, Bathon SA, Jeffe DB. Adherence to nasal positive airway pressure therapy among school-aged children and adolescents with obstructive sleep apnea syndrome. Pediatrics 2007;120:e1203-11.
143. McNamara F, Sullivan CE. Obstructive sleep apnea in infants and its management with nasal continuous positive airway pressure. Chest 1999;116:10-6.
144. Mellies U, Ragette R, Dohna-Schwake C, Boehm H, Voit T, Teschler H. Long-term noninvasive ventilation in children and adolescents with neuromuscular disorders. Eur Respir J 2003;22:631-6.
145. Young HK, Lowe A, Fitzgerald DA, et al. Outcome of noninvasive ventilation in children with neuromuscular disease. Neurology 2007;68:198-201.
146. Tan E, Nixon GM, Edwards EA. Sleep studies frequently lead to changes in respiratory support in children. J Paediatr Child Health 2007;43:560-3.
147. Tunkel DE, McColley SA, Baroody FM, Marcus CL, Carroll JL, Loughlin GM. Polysomnography in the evaluation of readiness for decannulation in children. Arch Otolaryngol Head Neck Surg 1996;122:721-4.
148. Ramagopal M, Scharf SM, Roberts DW, Blaisdell CJ. Obstructive sleep apnea and history of asthma in snoring children. Sleep Breath 2008;12:381-92.
149. Gozal D. Nocturnal ventilatory support in patients with cystic fibrosis: comparison with supplemental oxygen. Eur Respir J 1997;10:1999-2003.
150. Yuan N, Skaggs DL, Davidson Ward SL, Platzker AC, Keens TG. Preoperative polysomnograms and infant pulmonary function tests do not predict prolonged postoperative mechanical ventilation in children following scoliosis repair. Pediatr Pulmonol 2004;38:256-60.