Pediatric sleep apnea-a simplified approach

Unnati Desai, Vinaya S Karkhanis, Jyotsna M Joshi

Department of Pulmonary Medicine, T.N. Medical College, B.Y.L. Nair Hospital, Mumbai, India.

Abstract

Pediatric sleep-disordered breathing in children is a spectrum of primary snoring, upper airway resistance syndrome, obstructive hypoventilation, and obstructive sleep apnea syndrome (OSAS). The prevalence of pediatric OSAS is 1%-3%. Pediatric OSAS is most commonly caused by adenoid and tonsillar hypertrophy, which is correctable by surgery. The gold standard test for diagnosis is polysomnography (PSG). OSA in children is a distinct disorder form that occurs in adults with respect to clinical manifestations, PSG diagnostic criteria, and treatment approaches. In addition, PSG has its own challenges in children. Hence, simplification of approach by appropriate use of alternative diagnostic tests including oximetry scoring systems, questionnaires, and home respiratory polygraphy is highlighted. This will ensure early diagnosis, referral for corrective surgical management versus medical therapy on basis of severity, and performance of PSG in only selective cases.

Keywords: questionnaires, oximetry, home respiratory polygraphy, polysomnography. adenotonsillectomy.

Introduction

Obstructive sleep apnea syndrome (OSAS) in children was described by Osler in 1892. The first case series was published by Guilleminault et al. in 1976[1]. Subsequently, many studies have elaborated on pediatric OSAS, but, there still remain many areas of debate. Habitual snoring is reported in 8%-12% of children but only 1-3% show associated OSAS[2]. Pediatric OSAS is a serious condition that can adversely affect the child's growth, emotional and cognitive development, and cardiovascular health. It is

most commonly caused by adenotonsillar hypertrophy, which is correctable with surgery. The approach to diagnosis and management of pediatric OSAS differs vastly from that in adults. Diagnostic polysomnography (PSG), which is the gold standard test, has its own challenges in children. Alternative diagnostic tests including oximetry scoring systems, questionnaires, and home respiratory polygraphy (HRP) simplify the approach for early diagnosis, pending PSG and referral for corrective surgical management.

Pediatric Obstructive Sleep Apnea Syndrome

Pediatric sleep-disordered breathing in children is a spectrum of primary snoring, upper airway resistance syndrome (UARS), obstructive hypoventilation, and OSAS. The American Association of Pediatrics (AAP) guideline defines OSAS in children as a "disorder of

Address for Correspondence
Dr. J. M. Joshi
Professor and Head,
Department of Pulmonary Medicine,
T.N. Medical College and B.Y.L. Nair Hospital,
Mumbai, Maharashtra 400008, India
Email:drjoshijm@gmail.com

Table 1: Symptoms and signs of OSAS

History	Frequent snoring (≥3 nights week), labored breathing during sleep, gasps/snorting noises/observed episodes of apnea, sleep enuresis (especially secondary enuresis), sleeping in a seated position or with the neck hyperextended, cyanosis, headaches on awakening, daytime sleepiness, attention deficit/hyperactivity disorder, learning problems
Physical examination	Underweight or overweight, tonsillar hypertrophy, adenoidal facies, micrognathia/retrognathia, high-arched palate, failure to thrive, hypertension

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," accompanied by symptoms or signs, as listed in Table 1[3]. The various causative factors for OSAS are enumerated in Table 2. Adenoid and tonsillar hypertrophy is the most common cause of pediatric OSAS. Children experiencing neuromuscular diseases are also at an increased risk of OSAS in addition to commoner hypoventilation and central sleep apneas. The pathophysiology[4] of childhood OSAS remains poorly understood. However, it is proposed to be caused by a combination of anatomic and neuromotor factors, i.e., by the superimposition of structural aberrations upon an integrally more collapsible upper airway. Most important contributory anatomic factor is adenotonsillar hypertrophy, particularly in the preschool age group; the other being craniofacial abnormalities. Obesity leading to changes of upper airway anatomy is more important in the older children and adolescents. Upper airway neuromotor tone and imbalance of the pharyngeal dilator and constrictor muscles are hypothesized. Subtle abnormalities in the ventilator responses are suggested. Nasal and oropharyngeal inflammation might contribute to the pathogenesis of breathing disturbances during sleep[5]. Inflammatory hypothesis suggests role of leukotrienes[6].

Neuromuscular diseases cause OSAS owing to weakness of pharyngeal muscles and adenotonsillar hypertrophy. They are at increased threat for developing pulmonary hypertension, cor pulmonale, and neurocognitive dysfunction. Sleep-related hypoventilation/hypoxemia because of neuromuscular diseases might be aggravated in the presence of OSAS. In addition, some of these children have reduced central neural chemo responsiveness[7,8].

Diagnostic Issues in Pediatric Obstructive Sleep Apnea Syndrome

Overnight PSG evaluation in sleep laboratory is the gold standard to diagnose OSA at all levels of severity. Pediatric apnea-hypopnea index (AHI) severity criteria are as follows (<12 years of age): AHI 1 to <5 is mild, 5 to <10 is moderate, and >10 is severe (Table 3)[9]. However, the definite validation of PSG criteria for OSAS is yet to be done, and there is no clarity whether primary snoring without PSG-defined OSA is benign. The first-line therapy for OSAS is adenotonsillectomy; however, it needs cautious postoperative care because of the high risk of respiratory problems[3]. The AAP guidelines recommend that clinicians as a part of regular health-maintenance visits should ask about snoring, i.e., whether the child or adolescent snores. If a child snores on a regular basis and reveal any of the problems or findings given in Table 1, clinicians should either get a polysomnogram (level A) or refer the child to a sleep specialist or otolaryngologist for a more wide-ranging evaluation (level D). If PSG is not obtainable, then

Table 2: Predisposing factors to pediatric OSA: Predisposing factors to pediatric OSA Adenotonsillar hypertrophy is the major structural factor contributing to the pathogenesis of OSA in younger children while obesity is increasingly recognized as an important contributing factor to OSA in older children and adolescents.

•	Adenotonsillar hypertrophy
•	Obesity
•	Allergic rhinitis
•	Craniofacial malformations
•	Neuromuscular diseases
•	Genetic syndromes
•	Metabolic syndromes

Table 3: OSA severity criteria in children[7] and adults

OSA severity	AHI in children	AHI in adults
None	0	0–5
Mild	1–5	5–15
Moderate	5–10	15–30
Severe	>10	>30

clinicians may direct for other diagnostic tests, such as nocturnal video recording, nocturnal oximetry, daytime nap PSG, or ambulatory PSG (level C). If a child is detected as experiencing OSAS, shows a clinical inspection consistent with adenotonsillar hypertrophy, and does not have a contraindication, then adenotonsillectomy can be suggested as the first-line therapy by the clinician. Clinical decision is mandatory to examine the benefits of adenotonsillectomy compared with other therapies in obese children with different degrees of adenotonsillar hypertrophy (level B)[3]. If the child reveal OSAS but no adenotonsillar hypertrophy, other causes such as craniofacial syndromes, neuromuscular diseases, and cardiopulmonary or metabolic disorder should be considered. These patients need evaluation of OSA with PSG. However, PSG is costly, time consuming, requires specialized expertise, has limited accessibility, and may entail long waiting periods. Pediatric centers have published average costs for each PSG ranging from US $600-2,800. The inaccuracy of oxygen saturation testing alone for the diagnosis of OSA in those children who do not have significant desaturations is also a concern. Accordingly, it is important to develop other diagnostic measures for OSAS or to improve those that now exist. There are other challenges in carrying out and interpreting PSG in children compared with a supportive adult. The laboratory and technologist must show friendly approach and comfortable with the child while not too childish to discourage adolescents. Individual nap study parameters are not very accurate in diagnosing overnight polysomnographic findings and not suggested[3]. Children reveal shorter and lesser respiratory events than adults and an increased proportion of hypopneas; so, the studies must be recorded and studied with great caution. In adults, obstructive apneas of 10 s or longer are scored, but in children, apnea is scored if the decrease in oronasal flow more than 90% is for at least two respiratory cycles in the presence of respiratory effort. For a long time, an adult model has been incorrectly used for the diagnosis

and therapy of affected children. Hence, the diagnostic algorithm needs revision to appropriately triage; integrate use of screening tools, questionnaires, and alternative tests for OSAS; and selective referral for PSG in resource-limited situations.

History and Clinical Examination

Although history and physical examinations are suitable to diagnose patients and conclude which patients require extra examination for OSAS, there is argument about their parts in finalizing which patients need treatment. The AAP guidelines recommend that clinicians as a part of routine health-maintenance visits should ask whether the child or adolescent snores and OSA was unlikely in absence of snoring. Many studies have empirically examined the utility of a standardized history alone; history and physical examinations; history or physical examination; and audiotaping or videotaping to diagnose OSAS[10,11]. As early as 1995, Carroll et al[11]. warned that clinical evaluation alone was inadequate for the diagnosis of OSAS. In meta-analysis examining the sensitivity and specificity of the history and physical examinations in diagnosing OSA, a poor correlation was noted with PSG results[12]. Audiotapes or videotapes of the sleeping child recorded by the parent can occasionally be used by health-care teams to hear and watch for noticeable apneic episodes. Studies accessing the dependability of this method of testing found variable results with mostly poor predictive values[13]. However, clinical examination has a contributory role in the diagnostic approach. The regular health-care visits should comprise sleep history screening for snoring. In children, OSAS is very unlikely with the lack of habitual snoring. More thorough history concerning labored breathing during sleep, observed apnea, restless sleep, diaphoresis, enuresis, cyanosis, excessive daytime sleepiness, and behavior or learning problems should be acquired. Findings such as malnutrition (under or overweight), adenoid facies, nasal obstruction, adenotonsillar hypertrophy, micro/retrognathia, and hypertension may be present on clinical examination. Systemic hypertension, an enhanced pulmonic component of the second heart sound representing pulmonary hypertension, and reduced growth may be observed as complication of underlying OSA. Adenotonsillar hypertrophy is graded as follows: grade I, less than 25% space between pillars; grade II, less than 50% space between pillars; grade III, less than 75% space between pillars; and grade IV, tonsils in direct

Table 4: The McGill Oximetry Scoring system

Oximetry score	Comment	AHI	Criteria			Remarks	Recommendation
			No. of drops in $SaO_2 <$ 90%	No. of drops in $SaO_2 <$ 85%	No. of drops in $SaO_2 <$ 80%		
1	Normal study/ inconclusive for OSA	0	<3	0	0	Baseline SpO2 stable, <3 desaturations with $SpO_2 < 90\%$	Additional evaluation of breathing during sleep required to rule out OSA
2	OSA, mild	1–5	≥3	≤3	0	Three or more clusters of desaturation with ≤3 desaturations of $SpO_2 < 85\%$	Recommend adenotonsillectomy on the waiting list
3	OSA, moderate	6–10	≥3	>3	≤3	Three or more clusters of desaturations with <3 desaturations of $SpO_2 < 80\%$	Recommend surgery within 2 weeks
4	OSA, severe	>10	≥3	>3	>3	Three or more clusters of desaturations with >3 desaturations of $SpO_2 < 85\%$	Recommend urgent surgery (within days)

"Desaturation," 4% fall in saturation; "cluster," 5 desaturations within a 30-min period; "positive" study, 3 clusters with 3 desaturations to <90%; "Inconclusive" study, not a positive study (i.e., <3 clusters or <3 desaturations below 90%).
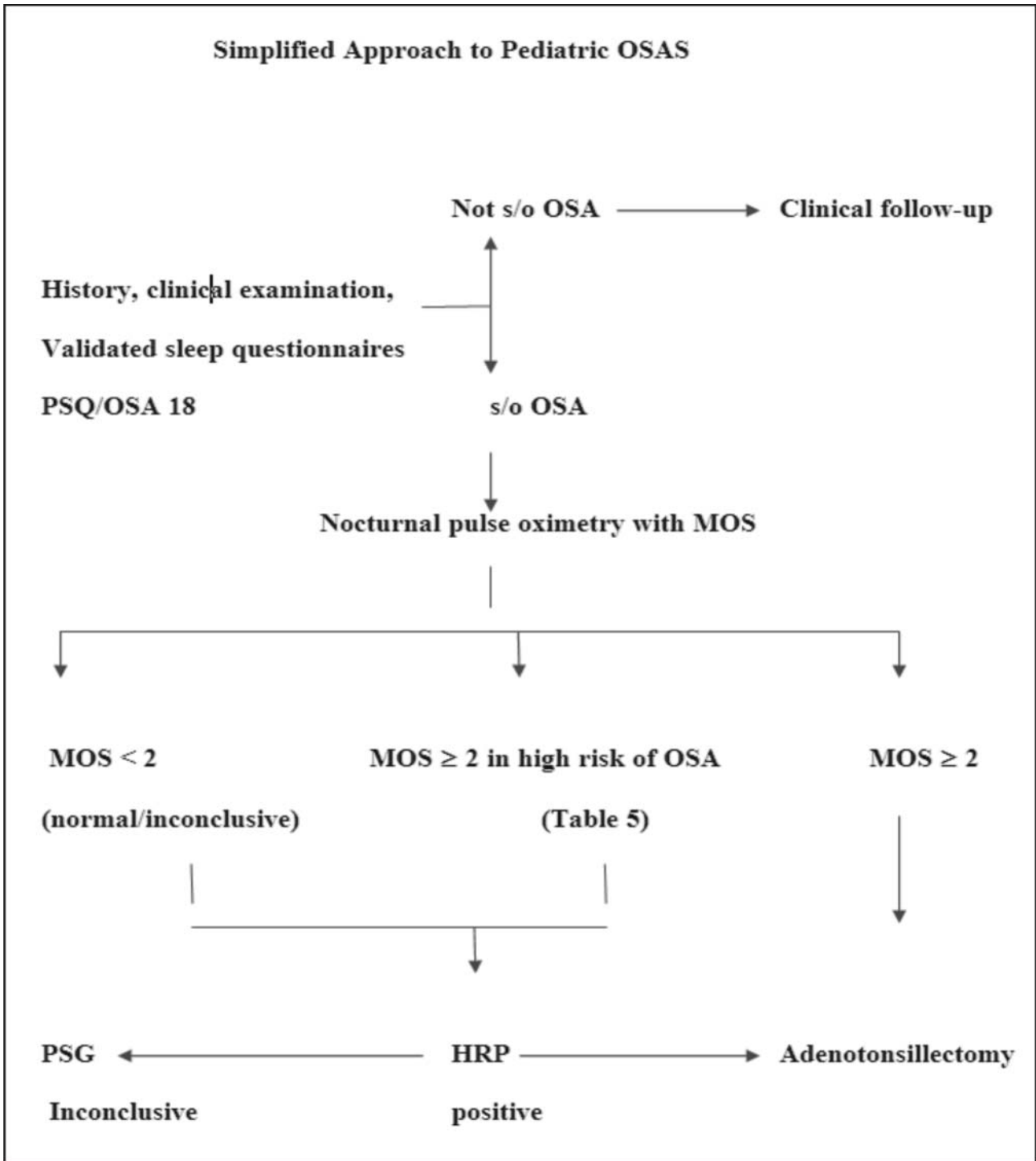
Figure 1: Simplified approach to pediatric OSAS

PSQ, Pediatric Sleep Questionnaire; OSA, obstructive sleep apnea; MOS, McGill Oximetry Scoring; HRP, home respiratory polygraphy

contact. Numerous studies have established that there is no connection between the size of the tonsils and adenoids and occurrence of OSAS[14,15].

Sleep Questionnaires

Sleep questionnaires were developed to (1) diagnose pediatric OSAS and (2) assess quality of life and response to OSAS therapy. These various questionnaires and clinical scoring scales, however, did not constantly possess accurate criteria for assessment and standardization, as recently proposed by Spruyt and Gozal[16]. Simple and suitable screening questionnaires depending on clinical history were examined for their capability to detect pediatric OSAS but did not exactly differentiate between OSA and primary snoring[10,11,17]. Subsequently, Chervin et al[18]. developed and validated a 22-item sleep-related breathing disorder (SRBD) score, called the pediatric sleep questionnaire (PSQ), which was strongly associated with diagnosis of an SRBD ($P < 0.0001$) in a logistic regression model accounting for age and gender. It is useful for research but not reliable enough for most individual patients. It forecasts OSA-related neurobehavioral illness and its reaction to adenotonsillectomy as well or enhanced than PSG[19]. The 22-item PSQ is the majorly extensively used method for evaluating OSAS owing to its psychometric properties and is particularly chosen because it has been effective in numerous languages. Montgomery-Downs et al. studied that scores obtained from parental-report questionnaires of children's snoring and other sleep-wake behaviors could be utilized as proxy predictors of snoring or sleep-disordered breathing in children. However,

Table 5: High risk factors for postoperative respiratory complications in children with OSAS undergoing adenotonsillectomy[29]

•	Younger than 3 years
•	Severe OSAS
•	Cardiac complications
•	Failure to thrive
•	Obesity
•	Craniofacial anomalies
•	Neuromuscular disorders
•	Recent respiratory tract infection

these were more useful for research purposes and not accurate enough for individual subjects. Furthermore, distinct threshold scores must be essential to predict OSAS in children from various age groups and socioeconomic backgrounds[20]. Owens and Dalzell[21] developed the "BEARS" sleep screening score that considerably enhanced the amount of sleep data recorded and the probability of detecting sleep problems in the primary-care setting[21].

Franco et al. were the pioneers in assessing quality of life improvements following adenotonsillectomy in children with OSAS. In the year 2000, they disseminated a specific questionnaire-the OSA 18-which opened the file for other authors to use this method in assessments[22]. The OSA 18 questionnaire comprise 18 items assembled into five domains, with questions about sleep disorders, the child's physical and emotional pains, daily problems for these patients, and the degree of parental anxiety. Caretakers were questioned and scored from 1 to 7 in accordance with the frequency with which issues defined by each question affected the children. Higher end OSA 18 scores were associated with more recurrent and significant clinical consequences in the quality of life of children. OSA 18 values may range from 18 to 126 points. Patients with a score below 60 experience lesser impact on their quality of life. Patients with a score between 60 and 79 are moderately affected. If the score equals or exceeds 80 points, there is higher impact on the quality of life. OSA 18 also comprise a 0 to 10 scale in which caretakers can give an total score about the patient's quality of life, with 0 representing the worst possible quality of life and 10 the best possible quality of life. de Serres et al[23]. developed a similar questionnaire-the OSD 6-which is also a strong tool for determining the quality of life in children undergoing surgery; it is also used for evaluating the postoperative development. Sohn et al[24]. performed a comparative study between the OSA 18 and the OSD 6 questionnaires and concluded that the OSA 18 was superior to OSD 6 in the correlation between its scores and polysomnographic findings and that OSA 18 could deliver dependable results in follow-up. However, the OSA 18 has been severely criticized for its ability to diagnose OSAS in several recent studies[25-27]. Subsequently, Kadmon et al. published a 6-item questionnaire[28] and an 8-item questionnaire-I'M SLEEPY[29]. There have been various other invalidated questionnaires published till date.

Oximetry Scores

The overnight oximetry was extensively studied but with variable results until in 2000 when Brouillette et al. published that nocturnal pulse oximetry could be used as an abbreviated testing modality for pediatric OSAS. In their study, oximetry was categorized as positive, negative, or inconclusive based on the following definitions and criteria: (1) A desaturation was defined as a decrease in oxygen saturation of 4% or more; (2) A cluster of desaturations was defined as five or more desaturations occurring in a 10- to 30-min period; (3) A positive oximetry trend graph showed three or more desaturation clusters and at least three desaturations to 90%; (4) A negative oximetry trend graph showed no desaturation clusters and no desaturations to 90%; and (5) An inconclusive oximetry trend graph was that which did not meet the criteria for positive or negative. They concluded that, in the situation of a child alleged of experiencing OSAS, a positive nocturnal oximetry trend graph shows at least a 97% positive predictive value. Oximetry could: (1) be the conclusive diagnostic test for straightforward OSAS ascribed to adenotonsillar hypertrophy in children older than 12 months or (2) rapidly and cheaply detect children with a history signifying sleep-disordered breathing, who would need PSG to clarify the type and severity. However, OSA cannot be ruled out with a negative oximetry result[30]. Subsequently, the 2002 AAP guidelines also recommended that single channel system, such as overnight oximetry, could aid in suggesting a history of uncomplicated OSAS by showing positive results. Nonetheless, a normal study could not eliminate OSAS and PSG continued to be the gold standard analysis[31]. However, OSAS in children is usually effectively treated by adenotonsillectomy, and the procedure may have postoperative complications in subgroup of patients. To diagnose OSA and prioritize surgery referral for adenotonsillar, several researchers have evaluated various easy algorithms. In 2004, Nixon et al[32]. developed a simple, McGill Oximetry score (MOS), based on Brouillette's oximetry score, which facilitates logical prioritization of the adenotonsillectomy surgical list and, in addition, predicts risk of postoperative complications. Their study was conducted in three phases. In phase 1, a severity score (MOS) was established by reviewing preoperative overnight oximetry in children who underwent emergency adenotonsillectomy in 1999-2000. The results were grouped as (1) normal or indecisive

oximetry recording (oximetry score 1) and, therefore, not able to eliminate OSA without further examination of breathing during sleep; (2) slightly abnormal study (oximetry score 2), predictive of OSA requiring adenotonsillectomy but not immediately; (3) evidently abnormal study (oximetry score 3), with a pattern constant with OSA, demanding surgery on an accelerated basis (subjectively defined as within 2 weeks); and (4) severely abnormal study (oximetry score 4), with a pattern constant with OSA, demanding surgery immediately (subjectively defined as within 1-2 days, with admission to hospital for stabilization before surgery). In phases 2 and 3 studies, the MOS was validated further and shown to be effective in estimating severity of OSAS. This was mainly to shorten the diagnostic and therapy process for those with more severe disease and help clinicians in prioritization of adenotonsillectomy and planning perioperative care. The MOS is elaborated in Table 4. They concluded, in children aged older than 1 year, alleged of having OSAS, a positive overnight oximetry revealed a positive predictive value of 97% and a negative predictive value of 47%. The MOS has been included in the Pediatric Society of New Zealand (PSNZ) 2005 guideline (Assessment of Sleep-Disordered Breathing in Childhood)[33]. However, its primary limitation is its low negative predictive value and low sensitivity, which may lead to missed cases. Hence, children with MOS $\geq$ 2 without high risk factors for adenotonsillectomy-related postoperative complications (Table 5)[31] need direct referral for surgery. Those with MOS <2 and $\geq$ 2 but with conditions that are high risk factors for postoperative pulmonary complications in children with OSAS undergoing adenotonsillectomy (Table 5)[31] need to be evaluated with more definitive tests such as HRP or PSG. The explanation for this limitation of oximetry is that some children with OSA desaturate at night, while others do not. The updated 2012 AAP guideline states that "Although polysomnography is the gold standard for diagnosis of OSAS, there is a shortage of sleep laboratories with pediatric expertise. Hence, polysomnography may not be readily available in certain regions of the country. Alternative diagnostic tests have been shown to have weaker positive and negative predictive values than polysomnography, but nevertheless, objective testing is preferable to clinical evaluation alone. If an alternative test fails to demonstrate OSAS in a patient with a high pretest probability, full polysomnography should be sought."[3] In 2013, Brouillette et al. published that night-to-night consistency of

nocturnal pulse oximetry, as analyzed by the MOS, as a diagnostic test and for severity evaluation of OSAS, which showed excellent agreement, and PSG was needed to rule in or rule out OSAS in children only if a single night oximetry testing was indecisive[34]. In the study by Lee et al., they used MOS as a diagnostic tool to conclude children appropriate to undergo adenotonsillectomy in peripheral hospitals or outpatient surgical centers, thus curbing surgical wait times for high-risk patients in tertiary centers. They found that an overnight home oximetry that is "normal/inconclusive" (MOS of 1) can detect patients who can be securely sent to peripheral hospitals or outpatient surgical centers for adenotonsillectomy. Thus, the MOS also enumerates respiratory difficulties postadenotonsillectomy in patients with regular or indecisive overnight oximetry[35]. Similar studies[36] reiterate that oximetry studies estimated with the MOS accelerate diagnosis and therapy of children with adenotonsillar hypertrophy referred for alleged sleep-disordered breathing. Children with atypical oximetry results were operated immediately after testing and triaged depending on oximetry results. No child with an indecisive oximetry result needed hospitalization for more than 1 night postoperatively. In a retrospective study,[37] a cohort of children with trisomy 21 was evaluated to study the potential usefulness of MOS in diagnosing OSAS among them. They concluded that MOS of 3 or 4 reliably identified patients with OSAS. The possibility of central apneas causing hypoxemia must be considered in those with MOS 2. With these caveats, MOS could be integrated for developing streamlined protocols to treat OSAS in children.

Home Respiratory Polygraphy

HRP, also known as level 3 PSG, involves unsupervised home-based recording of cardiorespiratory channels, i.e., oronasal flow, snoring, O_2 saturation, heart rate, thoracoabdominal movements, and body position during sleep. This technique has been validated in adults[38-40]. Studies in pediatric population also provide same results[41,42]. Studies on results of continuous positive airway pressure (CPAP) treatment prescribed based on diagnosis by PSG and HRP yielded similar improvements in terms of AHI, quality of life, clinical symptoms, and adherence to CPAP therapy[43-44].

Combined Modalities for a Diagnostic Approach

Although pediatric OSAS of varying severity is diagnosed definitely with PSG, few studies have explored the utility of the diagnostic algorithms with the above-mentioned tools used in combination in children. In 2006, Xu et al[45]. showed the value of combining numerous predictive factors for detecting OSAS, inclusive of clinical history data (sleep apnea, mouth breathing, nocturnal enuresis, and occurrence of daytime naps), physical examination (tonsillar hypertrophy), and lateral neck radiography. The sensitivity of the six chosen parameters was shown to be 93.5%, with 80% negative predictive value. Kaditis et al[46]. published an algorithm for the diagnoses of OSAS in children; in the step one, they suggested a "structured questionnaire" and proposed oximetry as a substitute tool in cases where PSG was inaccessible. In another publication, Alonso Álvarez et al[47]. proposed nocturnal cardiorespiratory polygraphy as a substitute even if PSG was obtainable; if AHI estimated by home polygraphy is higher than 5, then adenotonsillectomy could be proposed for the patient; but, if AHI is less than 5, PSG could be referred for the patient. Masa et al[48]. studied the utility of the PSQ and pulse oximetry (MOS) as screening tools in pediatric patients with suspected OSAS. The objective of their study was to assess these screening tools in snoring patients in whom noteworthy comorbidities were absent. They found that nearly 97% of patients who showed a positive PSQ and MOS were subsequently diagnosed with OSAS, suggesting that this group of patients may not require an overnight PSG for diagnosis. Brown et al.[49] evaluated children with sleep-disordered breathing scheduled for adenotonsillectomy with a combination of sleep-disordered breathing questionnaires and the MOS. They opined that combination assessment identifies the child with severe OSAS and provides a prediction of risk for perioperative adverse respiratory events. This approach allows excluding at-risk child from ambulatory surgical programs, triaging, and implementing risk-reduction strategies.

Thus, OSA in children is a distinct disorder from that occurs in adults with respect to clinical manifestations, PSG diagnostic criteria, and treatment approaches. A simplified multimodality diagnostic algorithm is needed for early diagnosis and treatment. In particular, when availability of PSG is an issue, a

protocol-based assessment avoids unnecessary evaluation in selected children and referring them for ambulatory adenotonsillectomy while further assessing high-risk (Table 5) children with inconclusive HRP for definitive tests such as PSG. The recommended diagnostic algorithm is as shown in Figure 1. High-risk patients should be monitored as inpatient postoperatively for 24 h. Adenoid regrowth may occur after surgical intervention, which may be associated with persistent symptoms[31]. Kheirandish et al[50]. studied the role of intranasal corticosteroids and leukotriene inhibitors such as montelukast for such residual OSA after surgery or if surgery is contraindicated. Several studies[5,6] suggest that the inflammatory mechanism in OSA comprise leukotriene expression and regulation. Treatment with montelukast results in signification decrease in adenoid size and in respiratory-related sleep parameters[51]. Anti-inflammatory therapy of childhood OSA is an encouraging method that might substitute surgical treatment in children with mild OSA. CPAP is suggested as therapy if adenotonsillectomy is not carried out or if OSAS perseveres postoperatively and weight loss, in addition to other treatments in patients who are overweight or obese. It is important for clinicians to keep in mind that children symptomatic for OSA may exhibit typical PSG results and PSG parameters may be atypical in relatively asymptomatic children. More research pertaining to pathogenesis, diagnosis, and treatment of pediatric OSA is needed to simplify the approach further.

References

1. **Guilleminault C,** Eldridge FL, Simmons FB, Dement WC. Sleep apnea in eight children. Pediatrics 1976;58:23–30.

2. **Chang SJ**, Chae KY. Obstructive sleep apnea syndrome in children: epidemiology, pathophysiology, diagnosis and sequelae. Korean J Pediatr 2010;53:863–71.

3. **Marcus CL,** Brooks LJ, Draper KA, et al. Diagnosis and management of childhood obstructive sleep apnea syndrome. Pediatrics 2012;130:576–84.

4. **Marcus CL**. Pathophysiology of childhood obstructive sleep apnea: current concepts. Respir Physiol 2000;119:143-54.

5. **Goldbart AD**, Tal A. Inflammation and sleep disordered breathing in children: a state-of-the-art review. Pediatr Pulmonol 2008;43):1151–60.

6. **Goldbart AD**, Goldman JL, Li RC, Brittian KR, Tauman R, Gozal D. Differential expression of cysteinyl leukotriene receptors 1 and 2 in tonsils of children with obstructive sleep apnea syndrome or recurrent infection. Chest 2004;126:13–8.

7. **Alves RS**, Resende MB, Skomro RP, Souza FJ, Reed UC. Sleep and neuromuscular disorders in children. Sleep Med Rev 2009;13:133–48.

8. **Hull J**, Aniapravan R, Chan E, et al. British Thoracic Society guideline for respiratory management of children with neuromuscular weakness. Thorax 2012;67 Suppl 1:i1-40.

9. **Mitchell RB**. Adenotonsillectomy for obstructive sleep apnea in children: outcome evaluated by pre- and postoperative polysomnography. Laryngoscope 2007;117:1844–54.

10. **Brouillette RT**, Hanson D, David R, et al. A diagnostic approach to suspected obstructive sleep apnea in children. J Pediatr 1984;105:10–4.

11. **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.

12. **Brietzke SE**, Katz ES, Roberson DW. Can history and physical examination reliably diagnose pediatric obstructive sleep apnea/hypopnea syndrome? A systematic review of the literature. Otolaryngol Head Neck Surg 2004;131:827–32.

13. **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.

14. **Laurikainen E**, Erkinjuntti M, Alihanka J, Rikalainen H, Suonpää J. Radiological parameters of the bony nasopharynx and the adenotonsillar size compared with sleep apnea episodes in children. Int J Pediatr Otorhinolaryngol 1987;12:303–10.

15. **Brooks LJ**, Stephens BM, Bacevice AM. Adenoid size is related to severity but not the number of episodes of obstructive apnea in children. J Pediatr 1998;132:682–6.

16. **Spruyt K**, Gozal D. Pediatric sleep questionnaires as diagnostic or epidemiological tools: a review of currently available instruments. Sleep Med Rev 2011;15:19–32.

17. **Suen JS**, Arnold JE, Brooks LJ. Adenotonsillectomy for treatment of obstructive sleep apnea in children. Arch Otolaryngol Head Neck Surg 1995;121:525–30.

18. **Chervin RD**, Hedger K, Dillon JE, Pituch KJ. Pediatric sleep questionnaire (PSQ): validity and reliability of scales for sleep-disordered breathing, snoring, sleepiness, and behavioural problems. Head Neck Surg 2003;128:344–52.

19. **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.

20. **Montgomery-Downs HE**, O'Brien LM, Holbrook CR, Gozal D. Snoring and sleep-disordered breathing in young children: subjective and objective correlates. Sleep 2004;27:87-94.

21. **Owens JA**, Dalzell V. Use of the "BEARS" sleep screening tool in a pediatric residents' continuity clinic: a pilot study. Sleep Med 2005;6:63-9.

22. **Franco RA Jr**, Rosenfeld RM, Rao M. First place-resident clinical science award 1999. Quality of life for children with obstructive sleep apnea. Otolaryngol Head Neck Surg 2000;123:9–16.

23. **de Serres LM**, Derkay C, Astley S, Deyo RA, Rosenfeld RM, Gates GA. Measuring quality of life in children with obstructive sleep disorders. Arch Otolaryngol Head Neck Surg 2000;126:1423-9.

24. **Sohn H**, Rosenfeld RM. Evaluation of sleep-disordered breathing in children. Otolaryngol Sleep Med 2000;1:21-32.

25. **Constantin E**, Tewfik TL, Brouillette RT. Can the OSA-18 quality-of-life questionnaire detect obstructive sleep apnea in children? Pediatrics 2010;125:e162–8.

26. **Borgström A,** Nerfeldt P, Friberg D. Questionnaire OSA-18 has poor validity compared to polysomnography in pediatric obstructive sleep apnea. Int J Pediatr Otorhinolaryngol 2013;77:1864–8.

27. **Ishman SL,** Yang CJ, Cohen AP, et al. Is the OSA-18 predictive of obstructive sleep apnea: comparison to polysomnography. Laryngoscope 2015;125:1491–5.

28. *Kadmon G,* Shapiro CM, Chung SA, Gozal D. Validation of a pediatric obstructive sleep apnea screening tool. Int J Pediatr Otorhinolaryngol 2013;77:1461–4.

29. *Kadmon G*, Chung SA, Shapiro CM. I'M *SLEEPY*: A short pediatric *sleep* apnea *questionnaire*. Int J Pediatr Otorhinolaryngol *2014*;78:2116–20.

30. **Brouillette RT**, Morielli A, Leimanis A, Waters KA, Luciano R, Ducharme FM. Nocturnal pulse oximetry as an abbreviated testing modality for pediatric obstructive sleep apnea. Pediatrics 2000;105:405–12.

31. **Section on Pediatric Pulmonology**, Subcommittee on Obstructive Sleep Apnea Syndrome, American Academy of Pediatrics. Clinical practice guideline: diagnosis and management of childhood obstructive sleep apnea syndrome. Pediatrics 2002;109:704–12.

32. **Nixon GM**, Kermack AS, Davis GM, Manoukian JJ, Brown KA, Brouillette RT. Planning adenotonsillectomy in children with obstructive sleep apnea: the role of overnight oximetry. Pediatrics 2004;113:e19–25.

33. **Pediatric Society of New Zealand**. Best practise evidence based guidelines (Assessment of Sleep Disordered Breathing in Childhood) guidelines. 2005. Available at: psnz@paradise.net.nz. Accessed on 10th Feb 2015.

34. **Pavone M**, Cutrera R, Verrillo E, Salerno T, Soldini S, Brouillette RT. Night-to-night consistency of at-home nocturnal pulse oximetry testing for obstructive sleep apnea in children. Pediatr Pulmonol 2013;48:754–60.

35. **Lee TT**, Lefebvre CE, Gans NE, Daniel SJ. Quantifying respiratory complications post-adenotonsillectomy in patients with normal or inconclusive overnight oximetry. J Otolaryngol Head Neck Surg 2013;42:50.

36. **Horwood L**, Brouillette RT, McGregor CD, Manoukian JJ, Constantin E. Testing for pediatric obstructive sleep apnea when health care resources are rationed. JAMA Otolaryngol Head Neck Surg 2014;140:616–23.

37. **Coverstone AM**, Bird M, Sicard M, et al. Overnight pulse oximetry for evaluation of sleep apnea among children with trisomy 21. J Clin Sleep Med 2014;10:1309–15.

38. **Alonso** Álvarez Mde L, Terán Santos J, Cordero Guevara J, et al. [Reliability of home respiratory polygraphy for the diagnosis of sleep apnea-hypopnea syndrome: analysis of costs]. [In Spanish] Arch Bronconeumol 2008;44:22.

39. **Masa JF**, Corral J, Pereira R, et al. Effectiveness of home respiratory polygraphy for the diagnosis of sleep apnoea and hypopnoea syndrome. *Thorax* 2011;66:567–73.

40. **Calleja JM**, Esnaola S, Rubio R, Durán J. Comparison of a cardiorespiratory device versus polysomnography for diagnosis of sleep apnoea. Eur Respir J 2002;20:1505–10.

41. **Tan HL,** Gozal D, Ramirez HM, Bandla HP, Kheirandish-Gozal L. Overnight polysomnography versus respiratory polygraphy in the diagnosis of pediatric obstructive sleep apnea. Sleep 2014;37:255–60.

42. **Alonso-Álvarez ML,** Terán-Santos J, Ordax Carbajo E, et al. Reliability of home respiratory polygraphy for the diagnosis of sleep apnea in children. Chest 2015;147:1020–8.

43. **Mulgrew AT,** Fox N, Ayas NT, et al. Diagnosis and initial management of obstructive sleep apnea without polysomnography: a randomized validation study. Ann Intern Med 2007;146:157–66.

44. **Whitelaw WA**, Brant RF, Flemons WW. Clinical usefulness of home oximetry compared with polysomnography for assessment of sleep apnea. Am J Respir Crit Care Med 2005;171:188–93.

45. **Xu Z**, Cheuk DK, Lee SL. Clinical evaluation in predicting childhood obstructive sleep apnea. Chest 2006;130:1765–71.

46. **Kaditis A**, Gozal LK, Gozal D. Algorithm for the diagnosis and treatment of pediatric OSA: a proposal of two pediatric sleep centers. Sleep Med 2012;13:217–27.

47. **Luz Alonso Álvarez L**, Canet T, Cubell-Alarco M, et al. [Consensus document on sleep apnea-hypopnea syndrome in children (full version). Sociedad Española de Sueño. El Área de Sueño de la Sociedad Española de Neumología y Cirugía Torácica(SEPAR)]. Arch Bronconeumol 2011;47:2-18.

48. **Peña-Zarza JA**, Osona-Rodriguez de Torres B, Gil-Sanchez JA, Figuerola-Mulet J. Utility of the pediatric sleep questionnaire and pulse oximetry as screening tools in pediatric patients with suspected obstructive sleep apnea syndrome. Sleep Disord 2012;2012:819035.

49. **Brown KA**. Evaluation of the child with sleep-disordered breathing scheduled for adenotonsillectomy. Sleep Med Clin 2013;8:149–55.

50. **Kheirandish L**, Goldbart AD, Gozal D. Intranasal steroids and oral leukotriene modifier therapy in residual sleep-disordered breathing after tonsillectomy and adenoidectomy in children. Pediatrics 2006;117:e61–6.

51. **Goldbart AD**, Goldman JL, Veling MC, Gozal D. Leukotriene modifier therapy for mild sleep-disordered breathing in children. Am J Respir Crit Care Med 2005;17:364–70.