Clinical Assessment Techniques For Pediatric Obstructive Sleep Apnea- A Review

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Abstract

Introduction

The understanding of the cells internal clock is of utmost importance inorder to diagnose a disease of sleep. Sleep disordered breathing (SDB) is one of the most common sleep disturbances among children; it represents a spectrum of symptoms from simple habitual snoring (HS) to obstructive sleep apnea (OSA). (1) Chronobiologists Hall, Rosbash, and Young received the 2017 Nobel prize in medicine for their work on the cell’s internal clock. (2) Obstructive sleep apnea (OSA) in children is an prevalent condition that imposes a large array of morbidities, some of which may have long-term implications, well into adulthood. European Respiratory Society (ERS) has defined obstructive SDB as “a syndrome of upper airway dysfunction during sleep, characterized by snoring and/or increased respiratory effort secondary to increased upper airway resistance and pharyngeal collapsibility”. (3,4)
The child will show various signs and symptoms which many most often go unnoticed or not considered important, hence its very important to have an proper clinical protocol and management techniques to provide a better adulthood.

This review article will help us understand the clinical assessments required in diagnosis of obstructive sleep apnea in pediatric patients.

CLINICAL CRITERIA FOR DIAGNOSIS OF OSA

History

Parents usually donot mention concerns about their child’s sleep issues if not asked directly. Parents will often report snoring in their children as occurring exclusively when their child is congested or with an upper respiratory tract infection. (5) On further probing questions should be asked to ascertain whether the child has any other signs of sleep disturbance, particularly signs of respiratory difficulty or apnea during sleep, and daytime concerns about school performance, behavior, focus, and sleepiness. Patients with snore concerning for OSA will generally have symptoms of audiblesnore 3 or more nights a week outside of the context of upper respiratory tract infections. (6) When parents are able to describe the concerning nocturnal breathing patterns in detail, they may describe snoring punctuated by episodes of frank obstruction or pauses in the child’s breathing. Older children and adolescents may describe waking from sleep due to cough or with a panicked, choking sensation. (7)

There may be other subtle signs of symptomatic OSA. Children may habitually sleep in positions that minimize obstruction, including with several pillows, or perhaps prone with their neck hyperextended. Older children with clinically significant OSA may present with daytime sleepiness as a core concern. (8) The Epworth Sleepiness Scale for Children and Adolescents is a helpful quantitative and trackable assessment of daytime sleepiness. (8) In addition to daytime sleepiness, the clinician should also screen for behavioral and learning issues, particularly regarding classroom performance. Children with marked nocturnal hypoventilation may report morning headache, which is related to carbon dioxide (CO2) retention. (9) Nocturnal secondary enuresis, defined as nighttime incontinence, which emerges after at least 6 months of continence, is also suggestive of SDB. (10) Obstructive sleep apnea is prevalent in children with a history of prematurity. (11) Children with chronic lung diseases, including bronchopulmonary dysplasia, are more likely to have comorbid SDB. (12) Patients with syndromes associated with macroglossia (eg, trisomy 21 and Beckwith-Wiedemann syndrome), micrognathia (eg, Pierre-Robin sequence), or palatal abnormalities may be anatomically predisposed to upper airway obstruction. (13) In children with severe gastroesophageal reflux or aerodigestive issues, reflux and subclinical aspiration during sleep may mimic the “choking” sometimes observed with obstructive events and may contribute to lung disease and nocturnal hypoxemia. (14) Patients with a history of sickle cell disease are at increased risk for disordered nocturnal breathing and are uniquely susceptible to nighttime oxygen desaturations attendant to severe OSA.

Diagnostic criteria: (15)

A. The presence of ANY 1 of the following:

1. Snoring
2. Labored, paradoxical, or obstructed breathing during the child’s sleep
3. Sleepiness, hyperactivity, behavioral problems, or learning problems

B. Polysomnography demonstrates 1 or both of the following:

1. One or more obstructive apneas, mixed apneas, or hypopneas per hour of sleep OR
2. A pattern of obstructive hypoventilation, defined as \( \geq 25\% \) of total sleep time with hypercapnia (PaCO2 > 50 mm Hg) in association with \( \geq 1 \) of the following: snoring, flattening of the inspiratory pressure waveform, or paradoxical thoraco-abdominal motion

**American Academy of Pediatrics 2012 Guideline Recommendations for the Diagnosis and Treatment of OSA in Children and Adolescents**

1. All children/adolescents should be screened for snoring.

2. Polysomnography should be performed in children/adolescents with snoring and symptoms of OSA. If polysomnography is not available, then alternative diagnostic tests or referral to a specialist for more extensive evaluation may be considered.

3. Adeno-tonsillectomy is recommended as the first-line treatment for patients with adeno-tonsillar hypertrophy.

4. High-risk patients should be monitored as inpatients postoperatively.

5. Patients should be re-evaluated postoperatively to determine whether further treatment is required. Objective testing should be performed in patients who are high risk or have persistent symptoms/signs of OSA after therapy.

6. Continuous positive airway pressure is recommended as treatment if adeno-tonsillectomy is not performed or if OSA persists postoperatively.

7. Weight loss is recommended in addition to other therapy in patients who are overweight or obese.

8. Intranasal corticosteroids are an option for children with mild OSA in whom adeno-tonsillectomy is contraindicated or for mild postoperative OSA

**SYMPTOMS OF OSA (15)**

1. Excessive daytime sleepiness.
2. Loud snoring three or more nights per week.
3. Episodes of breathing cessation witnessed by another person.
4. Abrupt awakenings accompanied by shortness of breath.
5. Awakening with dry mouth or sore throat.
6. Morning headache.
7. Difficulty staying asleep.
8. Attention problems.
9. Mouth breathing.
10. Sweating.
11. Restlessness.
12. Waking up a lot.

**MULTIPLE SCREENING TOOLS** are available for adult sleep apnea (e.g., STOP-BANG questionnaire). (16)

Such questions might include:

1. does your child snore loudly when sleeping?
2. does your child have trouble breathing while sleeping?
3. does your child stop breathing during sleep?
4. does your child occasionally wet the bed at night?
5. is your child hard to wake up in the morning?
6. does your child complain of headaches in the morning?
7. does your child tend to breathe through his/her mouth during the day?
8. have you or the teacher commented your child appears sleepy during the day?
9. does your child fall asleep quickly?
There are few questionnaires available to address OSA symptoms that are suitable for the paediatric population.

**Clinical Evaluation**

The physical examination of the patient with suspected OSA is most effective. When asleep, the muscles suspending the soft palate relax, as does the tongue. When the patient sleeps supine, these tissues may largely obstruct the upper airway.(17) Ideally, the oropharyngeal examination should note both the Mallampati classification and the tonsillar size, as both are associated with severity of OSA. The presence of tonsillar hypertrophy should be assessed by using a standard Brodsky scale from 0 to 4, wherein a score of 0 indicates the surgically induced absence of all tonsillar tissue and 4 indicates extension of the tonsils to the midline (so-called kissing tonsils). A high-arched palate may distort the architecture of the associated oropharynx and cause reduced nasal airflow. (18) Retrognathia or micrognathia reduces the space wherein otherwise normally sized tongue and soft tissues rest in the oropharynx. Malocclusion of the teeth may act in a similar way, forcing the jaw posteriorly and functionally reducing space in the mouth.(19) The nasal passages should be assessed for polyps or allergic rhinitis, which can exacerbate chronic nasal congestion. The adenoidal tissue, a midline aggregation of lymphoid tissue in the upper oropharynx, is generally not observable by standard clinical examination and is best assessed via flexible nasopharyngoscopy. (20) Truncal obesity, redundant neck tissue and other signs of metabolic syndrome or insulin resistance, such as acanthosis nigricans, suggest obesity-related OSA and comorbid metabolic disease.(21) Clubbing of the fingertips, which is often related to chronic hypoxemia and hypoventilation, may indicate underlying lung disease.

A thorough physical examination of a child suspected of having OSA must include evaluation of the child’s general appearance, with careful attention to craniofacial characteristics such as midface hypoplasia, micrognathia, and occlusal relationships. Evaluation for nasal obstruction depends on the child’s age. Septal deviation, nasolacrimal cysts, and nasal aperture stenosis must be considered in infants.(22) When examining the oral cavity, physicians should evaluate the geometry of the soft palate for size, redundancy, and clefting; document the size of the tongue and a nasopharyngoscopic examination to evaluate the size of the adenoidal tissue and the site of airway collapse.(23) Detection of tonsillar hypertrophy on routine examination should prompt physicians to question parents about snoring and other symptoms of OSA in their children.(24)

**ENT** (Ear Nose and Throat) objective examination should rule out the presence of possible tonsillar hypertrophy, which can be classified according to the Brodsky scale, evaluating the percentage of oropharynx occupied by tonsils (considered as the distance between the two anterior pillars). (25) Another classification is based on the evaluation of tonsil size (ranging from 0 to 4: 0 indicates tonsils removed surgically, 1 intravelic tonsils, 2 extravelic tonsils, 3 extravelic tonsils not reaching the midline and 4 tonsils reaching the midline). (26) Another frequently used classification has been reported by Friedman (Mallampati modified); it assesses the position of the tongue within the oral cavity, measuring how it obstructs the airway (grade 1: both uvula and tonsils are entirely visible, grade 2: uvula is visible but not the tonsils, grade 3: soft palate is visible but not the uvula, grade 4: only hard palate is visible) (27). It is also useful to observe the skeletal class (retrognathic, orthognathic or prognathic) and the facies. BMI and weight growth curve should be carefully assessed. Finally, it is also essential to measure arterial pressure and rule out eventual signs of pulmonary hypertension. (28)

There are a number of reasons why the history can be misleading. The loudness of snoring does not necessarily correlate with the degree of obstructive apnea. Thus, children may have very noticeable snoring without apnea. Children with OSAS experience obstruction primarily during rapid eye movement (REM) sleep, which occurs predominantly in the early morning hours when their parents are not observing them, thus leading to an underestimation of apnea. (29) Some children have a pattern of persistent partial upper airway obstruction associated with gas exchange abnormalities,
rather than discrete, cyclic apneas (“obstructive hypoventilation”). These children will not manifest pauses and gasps in their snoring, and therefore, the condition may be misdiagnosed as PS.

**Endoscopic Assessment**

Endoscopy with flexible optic fibres allows evaluating patency of nasal cavities (i.e. hypertrophy of the inferior turbinates, presence of septal deviations or choanal atresia, adenoid hypertrophy), tongue base tropism or the possible presence of laryngomalacia. (30)Drug-induced sleep endoscopy is performed by specialists who fully understand the upper airway anatomy dynamics contributing to OSA in the sleeping patient and is particularly useful in those with persistently elevated AHI. Sleep endoscopy serves to define the level of the obstruction and may reveal sleep state–dependent laryngomalacia or palatine collapse, facilitating more targeted intervention from the otorhinolaryngologist. Successful sleep endoscopy requires precise titration of anesthesia from an experienced anesthesiologist because the goal is emulation of the sleep state without obscuring the patient’s actual upper airway dynamics by inducing too deep a sleep state. (31)

**Polysomnography (PSG)**

Presently, PSG represents the gold standard to diagnose OSA in children. The aim of PSG is to: (i) diagnose, differentiate and quantify obstructive apnoeas, mixed apnoeas, central apnoeas; (ii) identify and classify hypopneas and high-resistance syndromes; (iii) evaluate sleep fragmentation. (32) PSG is an expensive exam; it requires specialised equipment and personnel, is time-consuming and often has long waiting lists. PSG can only be performed in a few centres, such as a sleep laboratory in a hospital setting, which allows continuous monitoring. PSG should cover at least two complete nocturnal sleep cycles, without premedication or sleep deprivation, preferably at a distance from any steroid treatment. PSG recordings in children can be longer than adults, due to their sleep times: 11 to 12 hours for small and pre-school children, 9 to 10 hours for school-age children. It is useful to extend the study time in the mornings to record REM sleep (when apnoea is usually worse). (33)

Apnoea is defined as the reduction of airflow of more than 90% for at least two respiratory cycles; it is considered obstructive if during the whole period the inspiratory effort is continued or increased, it is central if the inspiratory effort is absent, and is mixed if there is a respiratory effort present only during part of the event, especially at the end. (34)

Hypopnea is defined as reduction of airflow ≥ 30% for at least two respiratory cycles; reduction of the air flow is associated with an arousal or a desaturation > 3%. In children, the detection of a single apnoea episode or hypopneas per hour is considered pathological. Three degrees of OSA severity are identified according to the AHI: mild AHI 1-4, moderate AHI 5-9, Severe AHI ≥ 10.

The present polysomnographic classification also allows to identify children:

1) At risk of sequelae;
2) At risk of postoperative complications, which require strict clinical and instrumental follow-up;
3) At high risk of OSA even after adeno-tonsillectomy, requiring further investigations and treatments. (34)

**Nocturnal polysomnography (sleep study)** is the only diagnostic technique shown to quantitate the ventilatory and sleep abnormalities associated with sleep-disordered breathing and is currently the gold standard. Polysomnography, by definition, can distinguish PS from OAS. It can objectively determine the severity of OSA and related gas exchange and sleep disturbances. In addition, there is currently a shortage of facilities that perform pediatric polysomnography. The availability of pediatric polysomnography is expected to improve, especially with the computerized equipment currently available. (35,36). The in-laboratory sleep study, or polysomnography (PSG), is the gold standard in the diagnosis of SDB in children.
Audiotaping Or Videotaping

Studies have examined the use of audiotaping and videotaping, alone or combined with clinical findings, in establishing a diagnosis. (37,38) In these studies, sensitivity ranged from 71% to 94%, and specificity ranged from 29% to 80%. Positive predictive values (PPVs) were 50% and 73% for audiotaping and 83% for videotaping. Sounds of struggle on audiotapes were found to be more predictive of OSAS than were pauses. The negative predictive value (NPV) ranged from 73% to 88%. Although these techniques may have promise, the discrepancies in results from different centers indicate that additional study is necessary. (37,38)

Nocturnal Pulse Oximetry

Nocturnal pulse oximetry is a valid initial diagnostic test for SBD and OSA for different reasons: its high positive predictive value (97%), its easy applicability and low cost. Therefore, it represents a good screening tool.

A positive examination (3 or more desaturation clusters and at least 3 desaturations below 90%) is considered exhaustive for OSA diagnosis. According to Brouilette criteria, desaturation is defined as a decrease in SaO2 ≥ 4% and the cluster is characterised by at least 5 desaturations that occur in a period of 10-30 min.

A useful OSA severity scale is that proposed by the Canadian Brouilette group using the McGill Oximetry Score: (39)

1. Category 1, “non-conclusive examination”, no desaturation, or desaturation not meeting the subsequent criteria;
2. Category 2, mild OAS: at least 3 “clusters” of desaturation < 90%;
3. Category 3, moderate OAS: at least 3 “clusters” of desaturation < 85%;
4. Category 4, severe OAS: at least 3 “clusters” of desaturation < 80%.

The diagnostic categories 2, 3 and 4 identify, respectively, three increasing classes of priority indication for adeno-tonsillectomy, and three increasing categories of patients at high risk of developing perioperative complications (39).

Imaging And Laboratory Studies

In some cases of suspected OSA it may be beneficial to obtain lateral neck films to demonstrate upper airway narrowing. In patients with complex craniofacial abnormalities, computed tomography with three-dimensional reconstruction can be a powerful tool for oro-maxillofacial surgical consultants in staging and planning interventions. (40) An early-morning blood gas may be informative in screening for nocturnal CO2 retention. In rare cases in which a previously undiagnosed neuromuscular disorder or syndrome is suspected, referral to a specialist for targeted genetic screening should be considered. (40)

Summary

In summary, history and physical examination are poor at predicting OSAS. Most studies have shown that abbreviated or screening techniques, such as videotaping, nocturnal pulse oximetry, and daytime nap polysomnography tend to be helpful if results are positive but have a poor predictive value if results are negative. Thus, children with negative study results should undergo a more comprehensive evaluation. The cost efficacy of these screening techniques is unclear and would depend, in part, on how many patients eventually required full polysomnography. In addition, the use of these techniques in evaluating the severity of OSAS (which is important in determining management, such as whether outpatient surgery should be performed) has not been evaluated. Hence further indepth research on the pathophysiology and clinical findings with diagnostics using latest technology of OSA will help clinicians provide a better management to the patient.
REFERENCES


