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PEDIATRIC OBSTRUCTIVE SLEEP APNEA-A LITERATURE REVIEW

Pediatric Dentistry

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ABSTRACT

Pediatric obstructive sleep apnea (POSA) is an increasingly common form of sleep disordered breathing. It is caused by partial or complete obstruction of the upper airway during sleep that results in absent or diminished airflow in lungs. The treatment modalities consist of both surgical & nonsurgical methods. This article reviews the features and etiologic factors of OSA and diagnosis and possible treatment options of this disorder.

KEYWORDS

Pediatric obstructive sleep apnea, Diagnosis, Management, Pediatric Dentist

INTRODUCTION:

Sleep is a physiological need of the human body. Sleep architecture comprises two basic types of sleep called rapid eye movement (REM) sleep and non-REM (NREM) sleep. Infants spend almost 50% of the time in REM sleep, and as much as 80% of sleep is spent in REM sleep in premature infants. Physiologically, humans are most vulnerable to perturbations in breathing during REM sleep, thus often classified as sleep-related breathing disorders when there are associated disturbances in gas exchange.[1]

Sleep-disordered breathing refers to a spectrum of sleep-related breathing abnormalities that include snoring, upper airway resistance syndrome, obstructive hypopnea syndrome, and obstructive sleep apnea. Obstructive sleep apnea (OSA) is a disorder characterized by recurrent episodes of partial upper airway obstruction (hypopnea) or complete upper airway obstruction (apnea) during sleep, despite respiratory efforts, and it results in sleep disruption, usually an arousal, and ventilatory instability.[2]

Most of the time, the respiratory events are associated with snoring, oxygen desaturations and brief arousal from sleep. Polysomnography, or overnight sleep studies, remain the gold standard for the diagnosis of POSA. Pediatric OSA differs from adult OSA due to several developmental, physiological, and maturational factors related to respiration and sleep parameters.[3]

Epidemiology

The incidence of pediatric OSA peaks between 2 to 8 years of age due to the increased growth of tonsils and adenoids relative to the size of the upper airway in this age group. OSAS prevalence has 2 peak periods. The first peak occurs in children from 2 to 8 years of age, with the presence of enlarged adenoid and/or tonsils. A second peak arises during adolescence in relation with weight gain.[4] Risk factors for early-onset OSA include prematurity, Down syndrome, African American race, and daycare attendance. The severity can be increased in those with obesity, tobacco exposure, and reduced family income. Boys are at an increased risk after puberty, but the prepubertal risk is equal among boys and girls.

Etiology

Obstructive sleep apnea, 95% of diagnosed sleep apnea, is due to complete collapse of the upper airway or partial collapse, resulting in arousal from sleep or 3% or more oxygen desaturation. Anything that can decrease airway diameter or integrity can contribute to OSA, including anatomic, genetic, or neuromuscular issues.[5] The upper airway can have an increased risk of abnormal collapse due to both intrinsic and extrinsic factors. The intrinsic factors are based on the critical pressure in the airway that is needed to maintain patency. The extrinsic factors are fat deposits, hypertrophy of tissues, and craniofacial features that stray from normal anatomy that contribute to increased incidence of collapse.[6]

Pathophysiology

The pathophysiology underlying upper airway narrowing during sleep is multifactorial. OSA is most common in preschool-age children (3 to 5 years of age) because of the relative enlargement of tonsils and adenoids compared to their airway size during this age. Conditions predisposing to OSA include adenotonsillar hypertrophy, obesity, midface hypoplasia, craniofacial abnormalities, Down syndrome, neuromuscular disease, cerebral palsy, laryngomalacia, extreme preterm birth, sickle cell anemia, and metabolic disorders.[4] All of these abnormalities provide increased extrinsic factors that can lead to upper airway compression or collapsibility.

Obesity causes fat deposits to surround the upper airway and increase extrinsic pressure that causes collapse.

The presence of lymphoid hyperplasia causes tonsillar and adenoid obstruction of the airway, and the increased relaxation during sleep allows these to become significantly more problematic.

Neuromuscular dysfunction can be seen in both central and obstructive causes of sleep apnea. This is most often seen in cases like Down syndrome, where there is hypotonia, which contributes to increased susceptibility of the airway to collapse.

Additionally, patients with craniofacial abnormalities, including those with Crouzon, Pierre-Robin, or Apert syndromes, as well as those with cleft lip or palate, are at an increased risk. The presence of alteration of the normal airway anatomy as well as features such as micrognathia, micro or macroglossia, and midface hypoplasia all contribute to decreased posterior oropharynx space and the increased incidence of pediatric obstructive sleep apnea[6].

Table 1 Common Signs And Symptoms Of Pediatric Obstructive Sleep Apnea[7]

Infants, 3–12 months	Toddlers, 1–3 years	Preschool-aged children	School-aged children
Disturbed nocturnal sleep with repetitive crying	Noisy breathing or snoring	Regular, heavy snoring	Regular, heavy snoring
Noisy breathing or snoring	Restless nocturnal sleep	Mouth breathing	Restless nocturnal sleep
Nocturnal sweating	Abnormal sleeping positions	Restless nocturnal sleep	Sleepwalking
Poor suck	Nocturnal sweating	Sleepwalking	Sleep talking
Failure to thrive	Mouth breathing	Night terrors	Excessive bruxism
Delayed development	Night terrors	Enuresis	Difficulty to wake up in the morning
Apparent life-threatening event	Poor eating	ADHD-like symptoms	Morning headache
	Failure to thrive	Increased need for napping	Poor appetite
	Poor growth	Poor eating	Excessive daytime sleepiness Aggressiveness
		Growth problems	Emotional instability
			Learning difficulties

Clinical Manifestations

A wide range of symptoms and signs are associated with OSAS in children depending on their developmental stages **(Table 1).**[7] Snoring is the most common presenting complaint of children and adolescents with obstructive sleep apnea. Night terror and sleepwalking are frequently accompanied by sleep-disordered breathing during slow-wave sleep in children and adolescents with a positive family history of parasomnias.[8] Children with OSA are high risk for enuresis, which may resolve when the OSA is adequately treated.[9] Excessive daytime sleepiness is a typical symptom in adolescents with OSA, whereas hyperactivity or inattention is predominant in preadolescent children with sleep-disordered breathing. Morning headaches and poor appetite may present in OSA, which may be due to carbon dioxide retention,

sleep fragmentation, or gastroesophageal reflux.

Diagnostic Criteria

Diagnosis of OSAS in children is made on the basis of sleep history, physical examination, and polysomnographic findings **(Table 2).**[10] **The gold standard modality is overnight attended polysomnography.**[4] The parameters in these sleep studies include an electroencephalogram (EEG) to stage sleep and arousals, an electrooculogram (EOG) and chin electromyogram (EMG) to detect rapid eye movement (REM) sleep, an electrocardiogram (ECG), leg EMG to detect limb movements, oral-nasal airflow, thoracoabdominal effort, pulse oximetry, and body position. End tidal CO_2 and nasal pressure are additional parameters to be monitored.

Table 2 Diagnostic Criteria Of Pediatric Obstructive Sleep Apnea By The American Academy Of Sleep Medicine[10]

A. The caregiver of the child reports snoring, labored or obstructed breathing, or both snoring and labored or obstructed breathing during the child's sleep.
B. The caregiver reports observing at least one of the following:
1. Paradoxical inward rib-cage motion during inspiration
2. Movement arousals
3. Diaphoresis
4. Neck hyperextension during sleep
5. Excessive daytime sleepiness, hyperactivity, or aggressive behavior
6. Morning headaches
7. Secondary enuresis
C. Polysomnographic recording demonstrates 1 or more scorable respiratory events per hour (i.e., apnea or hypopnea of at least 2 respiratory cycles in duration).
Note: Very few normative data are available for hypopneas, and the data that are available have been obtained using a variety of methodologies. These criteria may be modified in the future once more comprehensive data become available.
D. Polysomnographic recording demonstrates either 1 or 2.
1. At least one of the following is observed:
a. Frequent arousals from sleep associated with increased respiratory effort
b. Arterial oxygen desaturation in association with the apneic episodes
c. Hypercapnia during sleep
d. Markedly negative esophageal pressure swings
2. Periods of hypercapnia, desaturation, or hypercapnia and desaturation during sleep associated with snoring, paradoxical inward rib-cage motion during inspiration, and at least one of the following:
a. Frequent arousals from sleep
b. Markedly negative esophageal pressure swings
E. The disorder is not better explained by another current sleep disorder, medical or neurological disorder, medication use, or substance use disorder.

Obstructive sleep apnea in children is defined as an AHI (apnea–hypopnea index) ≥ 1 event per hour based on the diagnosis of polysomnography (PSG), a criterion adopted by the American Academy of Sleep Medicine. According to the American Academy of Pediatrics obstructive sleep apnea can be mild, moderate or severe **(Table 3).**[11]

Research studies have shown that ambulatory PSGs may detect OSA in older children with moderate OSA (AHI > 5), but data are lacking regarding the value of these studies on a broad clinical scale.

Table 3 Pediatric Obstructive Sleep Apnea Syndrome Severity Scale[11]

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

The modified mallampati classification used by anesthesiologists to evaluate the upper airway has also been used to determine the risk for OSA. A Modified Mallampati class 3 or 4 airway is associated with a more difficult intubation and OSA. Hence it can be used as a screening tool in diagnosis of pediatric obstructive sleep apnea **(Figure 1).**[12]

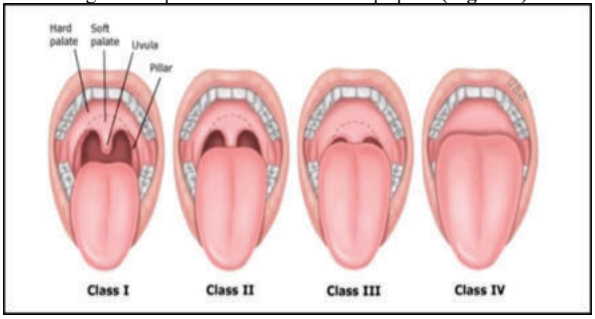

Figure 1 Modified Mallampati Scoring: Class 1 -soft palate, uvula, and pillars are visible; class II - soft palate and the uvula are visible; class III - soft palate and base of the uvula are visible; and class IV - only the hard palate is visible.[12]

In recent years, because of better diagnostic imaging, such as cone beam computed tomography (CBCT), and the integration of dental sciences with medical sciences, there has been greater understanding of the underlying causes of OSA in the pediatric population.

Treatment modalities

Paediatric OSA is a multifactorial disease and therefore requires a multidisciplinary approach for treatment. A variety of techniques and strategies can be used to address increased upper airway resistance and pediatric OSA. Treatment modalities can be widely divided into surgical and non-surgical.

Surgical Therapy

Tonsillectomy

If there is adenotonsillar hypertrophy, the most effective treatment is adenotonsillectomy (A&T). For the vast majority of children, this can be accomplished by surgical removal of both tonsils and adenoids, one of the few areas in medicine in which a more invasive approach is chosen first over a less invasive approach. The minimum age for performing adenoidectomy is three months, and for AT six months.

It recommended for most patients with an AHI more than 9 events/hour and those with mild or moderate disease with significant symptoms. A partial tonsillectomy is also an option that decreases both postoperative complications and recovery time, but it has been shown that tonsillar regrowth rates are between 7.2% to 16.6%.[11] One of the more conservative treatments, adenotonsillotomy, has not been shown to be superior to A&T in the treatment of pediatric OSA but due to increased risk of recurrence of OSA, and the possible need for repeat surgery with adenotonsillectomy should be taken into consideration.[13]

Radiofrequency ablation

Radio-frequency (RF) ablation is a new technique for shrinking the tonsils. Small probes are placed into the throat and the R-F energy is deposited, causing the tissue around the tip of the probe to heat up to a high temperature above which cells break apart and die. This technique has minimal thermal damage to surrounding tissues. The procedure is performed under a general anesthesia but the patient goes home the same day with a prescription for antibiotics[14]. The radio-frequency technique can also be used with minimal morbidity to reduce nasal turbinates enlargement which typically causes chronic nasal obstruction or a "stuffy" nose. Enlarged turbinates often cause mouth breathing and can also contribute to snoring and sleep apnea.[14]

Tracheostomy

By bypassing the site of obstruction with an artificial airway in the trachea, tracheostomy is a highly effective but end means of treating upper airway obstruction. At present, tracheotomy is rarely used in otherwise normal children who fail adenotonsillectomy, but may be needed in children with neuromuscular disorders such as cerebral palsy or severe craniofacial anomalies.[15]

Non-Surgical Therapy

Medications

This treatment modality comprises the use of upper airway anti-inflammatory medications including leukotriene receptor antagonists such as montelukast with or without intranasal steroids. These medications may be particularly effective for treatment, especially in children with nasal allergies that contribute to POSA symptoms. In mild POSA, or cases where there is adenoidal regrowth following Adenotonsillectomy, or when it cannot be performed, intranasal steroids are often indicated.[16]

Positive Airway Pressure

Nasal continuous positive airway pressure (CPAP) has been widely used in adults with sleep-disordered breathing since the report of its efficacy in virtually all cases by Sullivan in 1981[4]. Therapy with positive airway pressure (PAP) involves air that is pressurized by an electronic device and delivered during sleep via a nasal or oronasal mask, acting as a pneumatic stent of the airway. Positive airway pressure can be delivered as continuous positive airway pressure (CPAP) or as bilevel pressure (BiPAP). PAP is effective in eliminating OSAS in children, even those below 2 years of age, but adherence can be challenging even with close follow-up.[17-19]

However, even in experienced hands and with the help of a dedicated multidisciplinary team, mean use is only 5 hr/night which is clearly sub-optimal.

The challenges in treating children with OSAS using PAP include finding the appropriate equipment and achieving tolerance of that equipment. Behavioral therapy may improve adherence to PAP in children.[20-21]

Although PAP is generally well-tolerated, there are several potential complications like if masks fit poorly or are over-tightened, children may experience skin erythema or breakdown. Nasal congestion or dry nose are relatively common. Facial flattening and midface hypoplasia has also been reported.[22]

Oral Appliance Therapy

Maxillary expansion

Maxillary expansion is an orthodontic treatment adopted to correct maxillary transverse deficiency among growing patients by separating the midpalatal suture and increasing the hard palate's width. Children with OSA who have maxillary contraction, no adenotonsillar hypertrophy, and a body mass index < 24 kg/m^2 are considered to have the most favorable response to RME. Intervention with RME includes an active expansion phase (1 mm/day) for 10 to 20 days based on the original narrowness of the maxilla, and a fixed retention phase for consolidation with the device kept in place for 6 to 12 months. A significant reduction of AHI from 12.2 to 0.4 events/hr was found in a recent report using the aforementioned criteria. The improvement in OSA by RME may stem not only from augmentation of the maxillary complex, but also from modifying the resting posture of the tongue.

It is important to emphasize that the American Sleep Disorders Association states, "the presence or absence of OSA must be determined by a qualified physician before initiating treatment with oral appliances to identify those patients at risk due to complications of sleep apnea, and to provide a baseline to establish the effectiveness of subsequent treatment".[23]

Mandibular Advancement Device

Mandibular advancement (MA) with oral devices (OA), in particular mandibular advancement splints (MAS) in adults has been shown to be effective in treating OSA across a range of severity.[24,25] They enlarge the pharyngeal airway calibre predominantly at velopharynx (retropalatal) and this is mediated by an increase in its lateral dimension.[26] The improvements in airway volume are associated with soft tissue and bony structural changes. The mandibular advancers may be made of elastomeric material or hard acrylic, or thermoplastic. They can be either either onepiece [**monobloc**] or two-piece [**duobloc**] configuration. The former may be a simple vacuum formed splints with upper and lower fused together or clasped acrylic appliances, for example snore-guard, SNOAR etc. Two-piece splints, where upper and lower elements are connected by rigid or plastic lateral connectors, allow some freedom of mandibular movement.

In 2005, the American Academy of Sleep Medicine (AASM) published updated guidelines that support oral appliance therapy (OAT) "as a first-line therapy for mild and moderate OSA". [27]

The identification, diagnosis, and treatment of sleep disorders with OAT are areas where both dentists and physicians must work collaboratively for the benefit of the patient. By widening the upper airway with or without lessening the collapsibility of the airway, oral appliances can help improve opening the airway during sleep, which also improves the overall quality of the muscular tone.

Even though mandibular anterior repositioning appliance (MARA) are an established line of treatment in mild and moderate OSA cases in adults, there is still no consistent evidence showing MARA appliances' long-term effects in OSA children.

Role of a Pediatric Dentist

In providing collaborative patient-centered care, it is advisable for dentists to follow the established guidelines for the care of children with SDB, which have been promulgated by the American Academy of Pediatrics (AAP), American Academy of Pediatric Dentistry (AAPD), American Dental Association (ADA), and jointly by the American Academy of Sleep Medicine (AASM) and the American Academy of Dental Sleep Medicine (AADSM).[23]Pediatric dentists should screen patients for sleep-related breathing disorders such as OSA and primary snoring should, assess the tonsillar pillar area for hypertrophy and tongue positioning as it may contribute to obstruction, recognize obesity may contribute to.

Referal to an appropriate medical provider i.e., otolaryngologist, sleep medicine physician, pulmonologist, pediatrician for diagnosis and treatment should be made. Non-surgical intraoral appliances should be considered after a complete orthodontic/craniofacial assessment of the patient's growth and development. Pediatric dentists who perform sedation and surgical procedures in patients with OSA should be aware that these patients are more likely to experience perioperative and postoperative breathing complications. Performing an airway assessment in conjunction with the caregiver, especially when considering sedation or general anesthesia, may help identify patients at increased risk for OSA or peri-/post-operative breathing complications as part of a multi-disciplinary approach.[28]

CONCLUSION

Pediatric sleep breathing disorders (SBD) deserve attention, because it is associated with significant morbidity, potentially impacting on long-term neurocognitive and behavioral development. The presence of low-grade systemic inflammation and increased oxidative stress seen in this condition is believed to underpin the development of these OSA-related morbidities. Pediatric obstructive sleep apnea deserves specific attention by those who are positioned best to screen and detect signs and symptoms of pediatric sleep breathing disorders.

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