

Obstructive Sleep Apnea in Children: Exploring the Role of Dentists in Diagnosis and Treatment

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Abstract Among the many aspects important in a child's development is sleep. Sleep disorders can impair children's sleep and result in negative consequences. Obstructive sleep apnea (OSA) is one such disorder which involves blockage of the airway during sleep. The impact of OSA on the growth and development of a child may have detrimental effects on health, neuropsychological development and quality of life. One of the main causes of childhood OSA is hypertrophic adenoids and tonsils. As dentists often look into children's mouth, they can play an active role in identifying those with enlarged tonsils and referring them for sleep assessment. Therapeutic measures include not only surgical extraction of hypertrophic adenoids and tonsils, but also nonsurgical alternatives such as continuous positive air pressure, anti-inflammatory agents and oral appliances (OAs). Hence the therapeutic interventions that are directed at the site of airway obstruction in the maxillofacial region are within the scope of dentistry.

Keywords: *sleep disorders, obstructive sleep apnea, oral appliances*

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1. Introduction

Sleep is a key physiological drive. Pediatric sleep continues to gain significant recognition as a result of both increasing evidence for a high prevalence of sleep disorders among children and by virtue of the potential somatic and biobehavioral effects of altered sleep during development. [1] Sleep disorders can impair child's health and result in adverse consequences. Thus, respiratory disorders during sleep like obstructive sleep apnea (OSA) are of particular importance during childhood. [2] Obstructive sleep apnea (OSA) is characterized by repeated episodes of airway obstruction for more than 10 seconds during sleep, resulting in pauses in breathing that can affect both adults and children. [2,3,4] Obstructive sleep apnea syndrome (OSAS) first was reported in children in Guilleminault et al. (1976), since then recognition of abnormal breathing during sleep has gradually progressed. [5] Epidemiologic studies have shown that the incidence of sleep-disordered breathing (SDB) is about 2% among children [4,6,7] and about 2.5%–6% among adolescents [8]. Indeed, symptoms consistent with risk for SDB have been reported in 6% to 27% of children. [1,9,10,11] Children with SDB use greater health care resources [12] and display more frequent cardiovascular morbidity, [13,14] neurocognitive [15,16], metabolic complications [15,16], comorbid chronic illnesses [9,17], greater psychiatric and behavioral comorbidities [18,19,20,21,22], some of which may have long-term implications, well into adulthood. [1,2,4] Therefore, early

diagnosis and treatment of pediatric OSAS is beneficial in improving a child's long-term cognitive development, social interaction, academic achievement, cardiovascular health and overall wellbeing [16].

The pathogenesis of childhood OSA is multifactorial. In most children with OSA, the primary factor that leads to the upper airway obstruction is adenotonsillar hypertrophy [1,4]. Amongst the physicians treating children, dentists are most likely to detect adenotonsillar hypertrophy at an early age; thus, it may be most beneficial to these patients, if dentists act as "gatekeepers" in diagnosing children with adenotonsillar hypertrophy [16,23,24]. This review is attempted to provide an overview of OSA including the role of the oral health professional in the diagnosis and management of OSA.

2. Etiology

Increased upper airway resistance during sleep in children with OSA is most likely due to a combination of soft tissue hypertrophy, craniofacial dysmorphism, neuromuscular weakness or obesity [15,16]. Recently, the association between maxillofacial malformation and malocclusion to OSA has triggered more interest. Most common facial anatomic abnormalities associated with airway obstruction include deviated nasal septum, narrow maxillary arch and retro-micrognathia [15,16].

The most common cause of pediatric OSA is adenotonsillar hypertrophy [25]. A study by Konno et al reported that during sleep, pediatric patients with enlarged tonsils had an esophageal pressure that was 4–6-fold

higher than that of a control group [26]. The researchers explained that this elevation was caused by nasal obstruction due to adenoid hypertrophy. In another study, infants and children with enlarged tonsils were monitored for sleep apnea. All were found to have obstructions during sleep and 21 were found to have obstructive sleep apnea. Six of the 22 patients were clinically diagnosed with failure to thrive [27,28].

Risk factors for development of OSA in children comprise a family history of snoring or OSA, physical abnormalities, muscular dystrophy, Down's syndrome, syndromes that involve craniofacial and upper airway abnormalities, such as Pierre Robin, Apert's and Crouzon syndromes, cerebral palsy, sickle-cell disease, mouth breathing etc. [29,30,31] According to Tauman and Gozal, an important general risk factor for OSA is obesity among children, as the condition has been shown to be positively correlated with body mass index. [4,32] The degree of OSA is known to be proportional to the degree of obesity. However, this remains a controversy as most children with OSA are not obese [2,33].

3. Pathophysiology of Obstructive Sleep Apnea

Understanding obstructive sleep apnea in children necessitates a thorough knowledge of the physiology of sleep and breathing. There is an immediate increase in upper airway resistance with sleep onset and an initial 'overshoot' in this resistance that decreases very quickly. Still, this resistance during deep sleep is mildly higher than during wakefulness. [23,34] The size of the pediatric airway is dependent on craniofacial and soft tissue structures. [15] The available airway space may be decreased by abnormalities of oral cavity, larynx, nose, nasopharynx and oropharynx. [34] Pediatric OSA is the result of a complex interaction between an airway predisposed toward collapse and neuromuscular compensation [23]. During wakefulness, there is robust activation of pharyngeal dilator muscles and a stable ventilatory pattern. During sleep onset, there is a marked reduction in the activation of airway muscles, an increased ventilatory variability, and the appearance of an apneic threshold close to eupneic levels. Arousal from sleep contributes to ventilatory instability and therefore exacerbates obstructive cycling [23,34].

This phenomenon occurs through the night when the tongue falls back and blocks the airway. This means that every hour of every night there are multiple periods of O₂ deprivation to every cell in the body. These deoxygenated periods range between five to 100+ times an hour, for 10 to 90 seconds an episode. With inadequate oxygen, cellular regeneration is unable to transpire during sleep. Poor cellular regeneration and nightly cellular breakdown results in an individual being predisposed to numerous comorbidities anywhere in the body [35,36,37,38].

Obesity is a major factor and yields an interesting relation, which is the circumferential measure of the neck. Male obese patients with OSAS will frequently have a >40 cm/17in. neck, females >16in. This brings up the issue of "fat infiltration" into the pharyngeal space that some believe helps collapse the airway; however, it seems that this extra fat around the pharyngeal walls does not

compress the airway as suspected, but rather this fat infiltration "softens" the soft palate, tongue, epiglottis and pharyngeal walls setting them up for collapse during sleep [39,40].

4. Clinical Features of Pediatric Obstructive Sleep Apnea

OSA in children typically appears between the ages of 2 and 7 years. [2] OSA has a bimodal age of occurrence with the first peak coinciding with the developmental peak of adenotonsillar hyperplasia (2-5 years). The second peak appears in middle to late adolescence. [23,41] Although it was thought that boys and girls were equally affected, [6,7] Goodwin et al [3] observed that boys are more likely to have OSA, which is consistent with the inclination for overweight adult males to have OSA.

Clinical features concomitant with OSA in children include frequent snoring, excessive daytime sleepiness, pauses in breathing while asleep, restless sleep, bizarre sleeping positions, paradoxical chest movements parasomnias, nocturnal arousals, cyanosis, bedwetting hyperactivity, stunted growth and disruptive behaviour in school [4,6,32,42].

5. Diagnostic Criteria and Investigation

Sleep apnea in children and how it affects them in day-to-day life has been described in ample literature. Yet the dentists as well as general physicians usually fail to diagnose it. The dental office can play an important role in screening patients who may have OSA by taking a detailed medical history. However, Carroll et al in 1995 reported the inability of clinical history alone to differentiate primary snoring from OSAS in children [43].

Dentists are in a great position to provide further airway screening through cephalometry. The "Muller Maneuver" describes how to take a cephalometric film, to evaluate the airway in a dynamic fashion; prior to taking this radiograph the patient is asked to make a "forced" inspiratory effort with the mouth closed and nose plugged, the idea being that this should mimic an apneic event. If there is a dysfunction in the pharyngeal anatomy, soft palate, tongue, epiglottis or hyoid bone this can be evaluated on the radiographic film. [39,40] Adenoid space is another finding that is noticeable in a child's lateral film. The range of adenoid hypertrophy blocking the epipharyngeal space can be anywhere from none to a near complete blockage [40,44].

Nocturnal attended polysomnography, which requires an overnight stay in a sleep facility, is the standard diagnostic modality in determining if a patient has OSA. [23,45] The polysomnogram records parameters including electroencephalography (brain waves), electrooculography (eye movement), electrocardiography, electromyography (chin and leg movement), sleep positioning, respiratory activity and oxygen saturations. [46] The polysomnographic results for children with OSA show lower apnea indices compared to adults. [40,47] However, compared to adults, very little data are available regarding normal values of respiratory parameters during sleep in healthy children and adolescents. However, interpretation of abnormal

polysomnography and accurate diagnosis of disorders remains one of the challenges to pediatric sleep medicine and research, largely as a result of a historic lack of normative reference values. Because polysomnography is expensive and labor-intensive, and demands on resources are generally taken up by evaluation of disordered subjects or patients, few studies report normative values [1,48].

The American Academy of Pediatrics (AAP) has established clinical guidelines for better recognition and management of young patients with OSA. Dentists should also be aware of these guidelines. The guidelines recommended that for the diagnosis of OSA [49].

1. Compulsory screening is must in all children with snoring.
2. Referral to a specialist for high complex high-risk patients.
3. Elective evaluation for patients with cardiorespiratory failure.
4. Diagnostic evaluation is useful in discriminating primary snoring and OSA, with the gold standard being polysomnography.
5. The first line of treatment remains adenotonsillectomy for most children, and continuous positive airway pressure is an option for those who are not candidates for surgery or who do not respond to surgery
6. High-risk patients should be monitored as inpatients postoperatively.
7. All patients should undergo clinical reevaluation postoperatively to determine whether additional treatment is required.

6. Management of OSA in Children

Management of OSA can be surgical or nonsurgical. The potential contributing factors identified by the history, clinical examination, investigations and severity of the patient's condition must be considered in formulating a treatment plan. OSA severity is classified on the basis of the patient's AHI score, into three categories: [23]

Mild - AHI score between 5 and 15

Moderate - AHI score between 15 and 30

Severe - AHI score > 30.

Among children, an AHI score more than one and oxygen desaturations more than 4% are indicators of mild OSA.

The various treatment options available for OSA are as follows:

Surgical procedures

The traditional surgical procedure for treating OSAS in children is tonsillectomy with adenoidectomy. The goal of this procedure is to remove the obstruction by resection of all the lymphoid tissue of the tonsils. [50] Adenotonsillectomy is found to be an effective treatment for upto 80% of children diagnosed with OSA. [8] Chan et al., reported that adenotonsillectomy decreased symptoms associated with OSA in children. [51] Elsherif et al., in 1999 found a marked improvement in both daytime and nighttime symptoms in children after adenotonsillectomy. [47] In 2003, Uruma et.al, also clearly demonstrated a marked improvement in clinical symptoms such as snoring, sleep apnea attacks and mouth breathing after adenotonsillectomy [44].

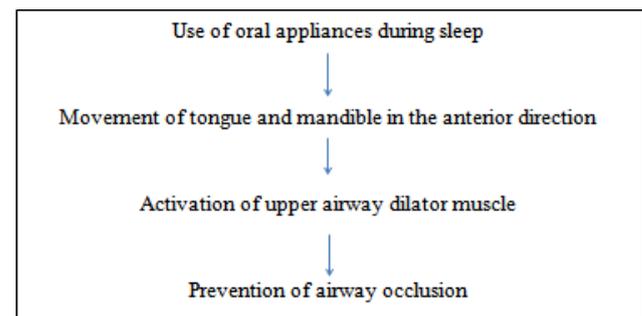
Continuous positive airway pressure (CPAP):

Since its initiation in 1981, it has become the gold standard treatment of OSA. CPAP therapy is indicated in children with specific surgical contraindications, minimal adenotonsillar tissue, or persistent OSA after adenotonsillectomy or for those who prefer nonsurgical alternatives. This treatment involves wearing a mask overnight that exerts pressure by acting as a pneumatic splint on the upper airway to prevent collapse. CPAP has better tolerance in older children and requires frequent clinician assessment of adherence and efficacy [52].

Diet and medications: For obese children, weight loss and healthy diet might prove to be the ultimate treatment. [33,38] A short course of broad spectrum antibiotics and underlying nasal pathology (if any) should be treated [23,53].

Oral Appliances:

Finally, oral appliances, which are provided primarily by dentists, have become increasingly popular within the past few years in the treatment of OSA. Oral appliances are of particular interest to people who opt not to have surgery and cannot tolerate continuous positive airway pressure treatment. [54,55] According to Cozza et al., a modified monobloc, is not only effective in reducing apneic events during sleep, but also improves subjective sleep quality and daytime performance among children [56,57].



Hoffstein et al concluded that oral appliances, although not as effective as CPAP in treating OSA, have a definite role in the treatment of snoring and sleep apnea. [58]

7. Role of Dentist in Management of OSA

Dentists are most likely to identify adenotonsillar hypertrophy at an early stage through a skillfully extracted history and meticulous examination. [2] The dental patient history and questionnaire should elicit responses to specific questions about sleep patterns, snoring, night or daytime bruxism, episodes of breath cessation, kicking/moving around during sleep, choking/gasping, abrupt awakening from sleep, excessive daytime sleepiness etc. Once dentists identify children with OSA, they should inform the parents about the risk of OSA and further inform their family physician. [59] This therapeutic use of oral appliances has involved dentists in the treatment of OSA among both adults and children [4,23].

Dentists should exercise cautiousness while performing routine dental procedures on patients with OSA. Surgical extractions in patients undergoing CPAP should be avoided as it may predispose the patient to subcutaneous emphysema. [60] Such patients should not be prescribed intravenous sedation because of compromised airway [61].

8. Conclusion

The impact of OSA on the growth and development of a child may have detrimental effects on the general health, neuropsychological development, quality of life and economic potential. The informed dentist can play a significant role in the initial diagnosis, the proper referral and in the likely supportive therapy of this population of patients.

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