Sleep Disordered Breathing in Children – A Review and the Role of a Pediatric Dentist

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Among the many factors important in children's development is sleep. Sleep disorders can impair children's sleep and lead to negative consequences. The most common sleep disordered breathing (SDB) in children is obstructive sleep apnea (OSA). One of the main causes of childhood SDB is enlargement of the tonsil tissues and, in most cases, their removal serves as an ultimate treatment of SDB. However, it remains unclear what proportion of children with enlarged tonsil tissue suffer from SDB. Dentists are becoming increasingly aware of the issue of SDB as they are sometimes involved in treatment of this condition using oral appliances. Moreover, as dentists often look into children's mouths, they can play an active role in identifying those with enlarged tonsils and referring them for sleep assessment. This review focuses on the diagnosis and treatment of SDB and also on the utility of oral appliances in the management of this disorder.
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INTRODUCTION

Sleep is a major physiological drive. The average child spends almost one-half of his or her life asleep.[1] Breathing disorders during sleep or sleep disordered breathing (SDB) can cause significant health problems that are associated with a high morbidity and a high risk of mortality.[2,3] The most widely studied breathing disorders during sleep are obstructive sleep apnea syndrome (OSA), central hypoventilation and Cheyne-Stokes respiration. Obstructive sleep apnea, or OSA, the most common type, is characterized by repeated episodes of upper-airway obstruction during sleep.[3,4]

OSA is characterized by repeated episodes of airway obstruction for more than 10 seconds during sleep, resulting in pauses in breathing. Within the past decade increasing attention has been placed on OSA among children. Epidemiologic studies have shown that there is a prevalence of about 2% among children[5,6] and about 2.5%–6% among adolescents.[7] OSA in children usually occurs between the ages of 2 and 7 years. Initially though it was thought that both the genders were equally affected,[8,9] it was later observed that boys are more likely to have OSA, which is consistent with the tendency for overweight adult males to have OSA.[10]

SDB in children is a timely public health concern, given the increasing rates of obesity in this population. In this review article we will discuss on the pathophysiology of SDB, etiology, the diagnosis of OSA and the management of the condition with emphasis on the role of the pediatric dentist in the diagnosis and referral and also the management of the same using oral appliances.

Changes in Respiration during Sleep – Differences between Children and Adults

We all breathe better awake than asleep. During sleep, there is a decrease in minute ventilation. In adults, minute ventilation decreases by approximately 13–15% compared with the value during wakefulness; respiratory rate tends to remain constant and the decrease is due primarily to a decrease in tidal volume.[11] In contrast, studies of infants, children, and adolescents have shown that the respiratory rate decreases during sleep.[12,13] Data on sleep-related changes in tidal volume in the pediatric age group are scarce, although one study in adolescents confirmed a decrease in tidal volume.[13] The functional residual capacity (FRC) decreases with sleep, and upper airway resistance doubles.[14] The ventilatory drive decreases, particularly during rapid eye movement (REM) sleep.[13,14] During REM sleep, breathing is erratic, with variable respiratory rate and tidal volume

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and frequent central apneas. REM sleep is also associated with a decrease in intercostals and upper airway muscle tone. Thus, breathing is impaired during sleep compared with wakefulness, and is further impaired during REM sleep. This is especially important in children, as they sleep more than adults, and have relatively more REM sleep. In neonates, active sleep (a REM-like state) can occur for up to two-thirds of total sleep time, [15] as compared with 20–25% of sleep time in adults.[16]

The way children with SDB present is different than in adults. Children tend to have fewer nighttime symptoms since their obstructive spells are prone to be brief and periods of arousal less obvious. Likewise, they present with more subtle behavioral changes in the daytime and do not have the degree of daytime somnolence seen in adults.[1] Unlike adults, children with SDB tend to be of normal weight or thin and may fail to thrive. Boys and girls are equally affected in this age group.[16] Table 1 summarizes differences between children and adults with SDB. Although SDB is relatively common in children, and can result in significant impact on development, it is not well understood.

Table 1: Summary of differences in SDB between children and adults

Variables	Adults	Children
Gender	Males/Females = 8-10/1	Boys/Girls = 1/1
Obstruction type/pattern	Obstructive apnea	Obstructive hypoventilation/ hypopneas
Snoring	Intermittent with pauses	Often continuously
Weight	Commonly obese	Underweight
Mouth breathing	Less common	Common
Severe sleepiness (somnolence) during the day	Basic symptom	Minimal percentage
Hypertrophy of tonsils-adenoids	Not usual	Very usual
Awakening at the end of apnea episode	Often	Not often
Sleep architecture	Fragmented	Preserved
Sequelae	Daytime sleepiness, cardiovascular disease	Behavioral changes, neurocognitive deficits
Primary treatment	Adenotonsillectomy	CPAP therapy

Etiology

The pathophysiology of SDB in children is similar to that seen in adults. During sleep, the ventilatory drive and upper airway muscle tone decrease. The inspiratory force collapses the pharyngeal airway that is already narrowed from other anatomic causes.

The collapse of the pharyngeal airway leads to partial airway obstruction producing hypopnea, or total airway obstruction resulting in apnea. Apneic and hypopneic events are terminated by arousals, in which natural defense mechanisms, the pharyngeal dilators, are activated. The whole cycle may repeat itself when the child returns to a deeper sleep stage with decreased ventilatory drive and upper airway muscle tone.[17]

The causes of Obstructive Sleep Apnea syndrome in children are discerned as follows: [18, 19, 20]

(a) **Anatomic anomalies**, such as hypertrophic tonsils and adenoids (the most common cause in childhood), choanal atresia or stenosis, anomalies of the nasal septum (mainly scoliosis) and soft tissue thickening of the upper respiratory system (polysaccharidosis, achondroplasia, Prader-Willi syndrome, obesity syndromes).

(b) **Neuromuscular diseases**, such as cerebral palsy, myotonic dystrophy, congenital myopathies and the Arnold-Chiari syndrome. These diseases may coexist with decreased muscle tone of the upper respiratory muscles and defective breathing control by the Central Nervous System.

(c) **Craniofacial anomalies and syndromes**. Most of these conditions coexist with midfacial hypoplasia, micrognathia, hypotony, and sometimes mental retardation.

Diagnosis

Polysomnography (PSG), as in adults, is the gold standard for diagnosis of OSA in children.[21] The standard parameters provided from polysomnography include sleep architecture, respiration, cardiac rhythm, muscle activity, gas exchange, and snoring. The most important index of polysomnography in defining the severity of OSA is the apnea/hypopnea index (AHI) which is defined as the number of apneas and hypopneas per hour of total sleep time. Apnea in children is defined as absence of airflow with continued chest wall and abdominal wall movement for duration longer than 2 breaths, [22] whereas obstructive hypopnea is defined as a decrease in nasal flow between 30% and 80% from baseline with a corresponding decrease in oxygen saturation of 3% and /or arousal.[21] An AHI > 1 event/h in children is considered abnormal.[23] With the use of appropriate equipment and an experienced technician, polysomnography can be performed successfully in infants and children of all ages. There are several polysomnographic differences in OSA between children and adults.

1. Children with OSA frequently do not have cortical arousal associated with obstructive apnea and are less likely to have fragmented sleep than adults. Consequently, sleep architecture is preserved and daytime sleepiness is uncommon.[24, 25]
2. In children, the majority of obstructive apneas occur during rapid eye movement (REM) sleep, particularly in later REM sleep.[26] As a result, OSA may be missed if the REM stage is decreased or absent on screening studies, e.g. nap studies.
3. Children may present with persistent obstructive hypoventilation, rather than cyclic obstructive apnea.[21, 27] Clinically, these children manifest constant snoring and labored breathing instead of breathing pauses or gasps.

Clinical Presentation

The three main nighttime symptoms of OSA in infants and children are snoring, apnea with noisy resumption of

breathing, and difficulty in breathing with an inward movement of the upper chest during inspiration.[28] Snoring occurs in almost all children with SDB and is the main reason many parents seek medical advice. However, only a proportion of snoring children have OSA (habitual snoring: OSA = 10%: 2% in the childhood population).[29] Besides, children with severe OSA may manifest without clear snoring because of prolonged breathing pauses. Consequently, snoring alone is an insensitive indicator of OSA and it is difficult to make a diagnosis of OSA based on a history of snoring alone. In addition to snoring, the majority of children with SDB who are referred to otolaryngologists have mouth breathing and adenotonsillar hypertrophy. The relationship between mouth breathing and adenoidal hypertrophy is straightforward. A study revealed that mouth breathing is a significant predictor for suspecting OSA with a specificity and positive predict value of 100%, and warrants early polysomnography.[29] Mouth breathing has also been found to be a cause of abnormal facial development such as adenoid faces and dental malocclusion.[30, 31]

Restless sleep and persistent body movements are frequently observed in children with SDB. Odd sleep positions (kneeling or upright sitting) are not rare in childhood with SDB. These unusual behaviors are thought to be a compensatory mechanism to extend the neck and improve airway obstruction during sleep. Enuresis, particularly if secondary in etiology, is associated with SDB in children. It is thought that fragmentation of sleep architecture by apnea and arousal may affect normal secretion of anti-diuretic hormone, and contributes to enuresis. Nocturnal enuresis can be the principal clinical picture in some older children with SDB.[31]The manifestation can be reversed by adenotonsillectomy in most cases.[32] Excessive daytime sleepiness, the most prominent clinical symptom of OSA in adults, is not a common complaint in pediatric SDB.[33] However, it is seen in some children with severe OSA and is more common in adolescents, particular if they are morbidly obese. In contrast, younger children often become hyperactive rather than sleepy. Children with OSA can display daytime behavior

disorders such as inattention, hyperactivity, aggressiveness, and social withdrawal.[34] There is good evidence that SDB leads to daytime disturbance closely mimicking attention-deficit/hyperactivity disorder (ADHD) in children.[35] Furthermore, snoring and other SDB-related symptoms are strong risk factors for the future emergence or exacerbation of hyperactive behavior in a four-year prospective cohort study.[36] In addition, learning problems can be at the forefront of the clinical picture in school-age children.[37] It is therefore recommended that a sleep study be performed to identify SDB in children diagnosed with ADHD.

Treatment of SDB in children

Treatment for children with SDB can be either surgical or non-surgical. The classification of the treatments has been summarized in Table 2. The choice of treatment is made based on the underlying etiology.

Table 2. Treatment options in the treatment of SDB

Non- Surgical	Surgical
Treatment of nasal allergy	Adenotonsillectomy (AT)
Treatment of acute inflammation	Uvulopalatopharyngoplasty (UPPP)
Continuous Positive Airway Pressure (CPAP)	Revision of posterior pharyngeal flap
Rapid Maxillary Expansion	Distraction Osteogenesis
Weight Reduction	Tracheotomy
Sleep hygiene (Sleep modification)	

Adenotonsillectomy (AT)

Because adenotonsillar hypertrophy is a significant contributing factor to OSA among children, for many, removal of the tonsils is the ultimate treatment of their OSA. In fact, adenotonsillectomy is found to be an effective treatment for up to 80% of children diagnosed with OSA.[38] Others reported that adenotonsillectomy decreased snoring, OSA, weight problems, enuresis and behavior problems in children with OSA. Authors[39] also studied the outcomes among 76 children, aged 3–12 years, with large tonsils or large adenoids (or both) who underwent surgery to relieve upper airway obstruction. These children were studied over 1 year. Following surgery, almost all patients experienced an alleviation of all sleep difficulties, except for enuresis (bedwetting). In conclusion, the authors[39] expressed the strong view that all children with large tonsils should consider adenotonsillectomy as well as a polysomnography recording. Another study found that adenotonsillectomy significantly reduced health care utilization by children with OSA.[40]

Uvulopalatopharyngoplasty (UPPP)

UPPP has been successfully used to strengthen hypotonic pharyngeal musculature in children in whom abnormal upper airway neuromuscular tone contributes to OSA, such as those with cerebral palsy or Downs syndrome.[41, 42] It has also been reported to be helpful in treating an otherwise normal child with OSA.[43] Currently, UPPP is considered for cases of pediatric OSA with obesity or redundant oropharyngeal soft tissue. Nevertheless, the muscular structure of the soft palate must be preserved and the procedure done as conservatively as possible in children to prevent troublesome velopharyngeal insufficiency.

Continuous Positive Airway Pressure (CPAP)

Nasal CPAP has been widely used in adults with SDB since the report of its efficacy in virtually all cases by Sullivan in 1981.[44] While the widespread use of CPAP in children with SDB has been hampered by the lack of masks adapted to children, this is slowly being corrected in recent years. Several reports have highlighted the ability to use CPAP in children and infants with SDB,[32] especially in children with gross craniofacial anomalies (e.g., Pierre Robin sequence, Treacher-Collins syndrome), Down syndrome, morbid obesity or upper airway muscle weakness (e.g., cerebral palsy). Hence, CPAP increasingly replaces surgery and tracheotomy

in severe SDB children. With the recognition of persisting SDB in at least 20% of children after AT, CPAP is increasingly used after AT.[34, 35] While daily CPAP treatment is highly effective in SDB children, it is not curative and has often to be used on a long-term basis.[37] Compliance in children is a genuine hurdle. Behavioral techniques are of help for getting accustomed to the mask and ventilatory equipment.[45] 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. While the use of automated CPAP, which allows continuous adjustment to varying upper airway resistance, may increase compliance, specific studies on the subject are needed. Finally, the use of nasal CPAP has been associated to mid-face hypoplasia in one report[46] and could conceivably increase SDB further. In conclusion, CPAP can be used in children with severe SDB or persisting SDB after AT. Significant remaining problems are related to the lack of optimal equipment for young children and frequent difficulties with regards to initial adaptation to the nasal mask and compliance to treatment.

Radiofrequency ablation

One of the causes of sleep apnea and snoring is enlarged tonsils or a thickened soft palate. 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. Unlike the traditional tonsillectomy where the entire tissue is excised from the base, this R-F procedure involves ablation via a probe that is put into contact with the surface of the tonsil, removing tissue layers one at a time. The procedure is performed under a general anesthetic but the patient goes home the same day with a prescription for antibiotics.[63]

The radio-frequency technique can also be used with minimal morbidity to reduce nasal turbinates—the small, shelf-like, bony structures covered by mucous membranes that protrude into the nasal airway and help warm and cleanse the air as it is inhaled. The

enlargement of nasal turbinates typically causes chronic nasal obstruction or a "stuffy" nose. Enlarged turbinates often cause mouth breathing and can also contribute to snoring and sleep apnea.[63, 64]

Tracheotomy

Tracheotomy is the ultimate treatment of OSA. Bypassing the pharyngeal obstruction can relieve obstructive apnea but not central apnea. However, a tracheotomy in children is associated with many side effects including impediments in speech and learning, chronic tracheitis, and interference with social activity. Fortunately, the introduction of continuous positive airway pressure (CPAP) and other treatment modalities have decreased the requirement for tracheotomy in pediatric OSA. 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.[17]

Orthodontic treatments in children with SDB

Oral Appliances

Oral appliances, which are provided primarily by dentists, have become increasingly popular within the past few years for treatment of OSA.[47] Oral appliances are of particular interest to people who opt not to have surgery and cannot tolerate continuous positive airway pressure treatment. Oral appliances provide effective treatment for many patients and, in one study, were shown to be effective in as many as 50% of patients with OSA.[48] Researchers investigating the use of oral appliances for children[46] have found certain types to be particularly effective in treating OSA. For example, according to Cozza and colleagues,[47, 48] a new orthodontic appliance, a modified monobloc, is not only effective in reducing apneic events during sleep, but also improves subjective sleep quality and daytime performance among children.[49]

Rapid Maxillary Expansion (RME)

RME is an orthodontic procedure that uses a fixed appliance with an expansion screw anchored on selected teeth. It is aimed at skeletal expansion of the upper jaw by the application of orthopedic force to the midpalatal suture resulting in maxillary widening.[50]

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.[51] 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.[50] A significant reduction of AHI from 12.2 to 0.4 events/hr was found in a recent report using the aforementioned criteria.[51] 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.[52]

Complications of untreated SDB

As in adults, OSA among children can lead to various adverse health-related consequences. For instance, OSA places increasing strain on the cardiovascular system due to the acute increases in blood pressure and arrythmias associated with apneas and intermittent hypoxia.[53-55] Those who suffer from OSA may snort awake, thus experiencing fragmented sleep. Among children, particularly, OSA can lead to severe developmental problems including failure to thrive, enuresis, attention deficit disorder, behavior problems, decreased academic performance and cardiopulmonary disease.[54] Studies have reported excessive daytime sleepiness in children with OSA, and other studies have shown that children with OSA are more aggressive, have higher rates of inattention, are more moody and have impaired visual perception and working memory.[56] Researchers have further reported that a large percentage of children with attention

deficit hyperactive disorder had underlying sleep disorders including OSA.[8]

In another study inattention and hyperactivity were found to be associated with increased daytime sleepiness and, particularly in young boys, there was an association with snoring and other symptoms of sleep-disordered breathing. The large sample size in this study (n = 866), as well as the use of questionnaires for both the children and their parents, increases the applicability of these results to general pediatric patients.[35] Furthermore, others found that the relation between sleep problems and attention deficit hyperactive disorder depends on the type of sleep problem as well as the confounding factors.[57] authors also found that children with SDB had significantly lower mean scores on IQ-like tests. In addition, children with SDB scored significantly lower than a control group on a test of phonological processing, a skill that is very important for literacy.[58] Finally, this study found that total arousal index was negatively correlated with neurocognitive abilities, suggesting a role for sleep fragmentation in SDB-induced cognitive dysfunction in children.[58]

Role of a Pediatric Dentist in the diagnosis or referral and the management of these children

As adenotonsillar hypertrophy is one of the main causes of OSA among children, investigating the prevalence of OSA among children with adenotonsillar hypertrophy is an important research question. Although previous studies have looked at adenotonsillar hypertrophy in children suffering from OSA, the reverse question (frequency of OSA among children with adenotonsillar hypertrophy) needs more attention. There is evidence that physicians may not always recognize childhood OSA.[59] According to authors,[60] an average delay of 23 months occurred between identification of pediatric patients with large tonsils and their referral to a sleep clinic. As sleep disorders in children might lead to various behavioral and learning problems, such problems may be misdiagnosed in a child with undiagnosed OSA; identifying and treating children with OSA would significantly benefit several facets of their lives. The first step in this process is to identify children with adenotonsillar hypertrophy, then refer them to a sleep clinic. Among the physicians treating children, dentists and especially pediatric dentists are most likely to identify adenotonsillar hypertrophy; thus, it may be in the patient's best interests if we pediatric dentists act as guards in identifying children with adenotonsillar hypertrophy. The Fig 1[8] just gives us an idea on the factors to look out for in the diagnosis of adenotonsillar hypertrophy. As discussed above, dentists are becoming increasingly aware of sleep apnea in adults, as some are involved in using oral appliances to treat this disorder.[61,62] Once we pediatric dentists identify children with adenotonsillar hypertrophy, they should inform the parents about the risk of OSA and further inform their family physician about the importance of sleep assessment in children with enlarged tonsils. Involvement of dentists in this process can contribute significantly to the health of patients, as OSA, with such significant developmental consequences, can be diagnosed and treated at an early stage, preventing later problems and complications.

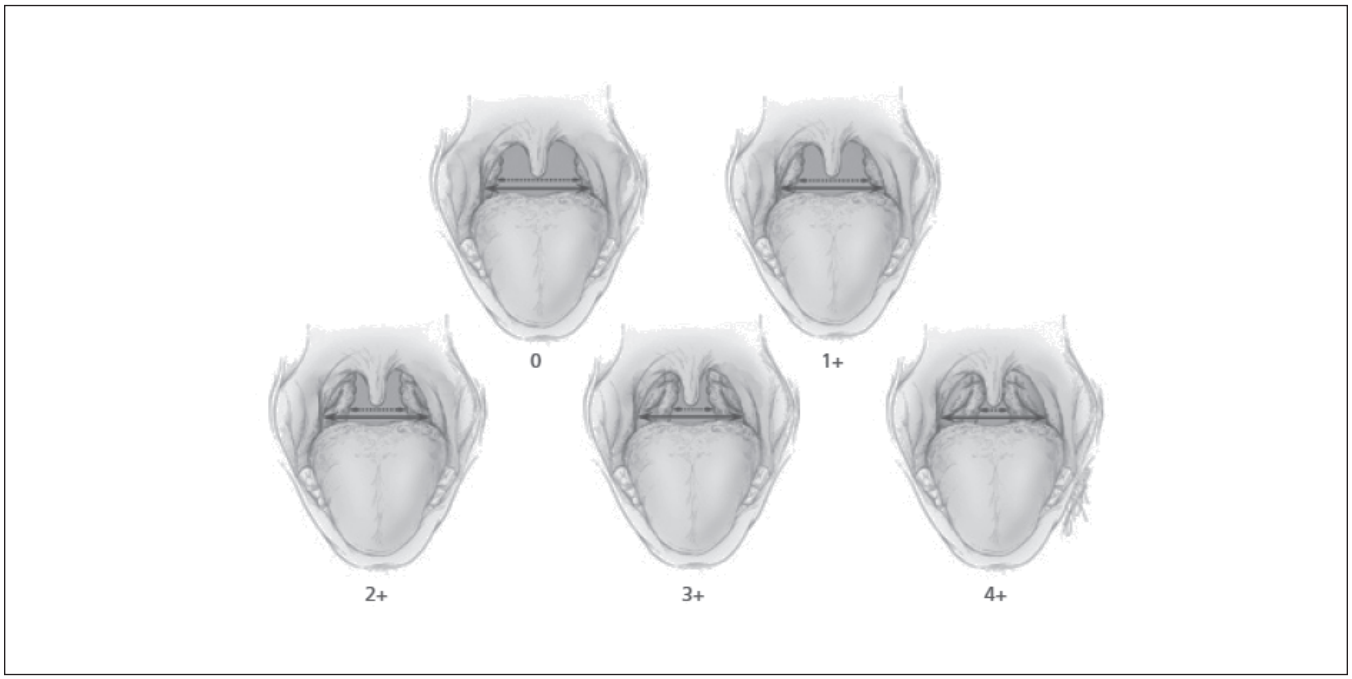

Figure 1. Standardized tonsillar hypertrophy grading scale.
(0) Tonsils are entirely within the tonsillar fossa.
(1+) Tonsils occupy less than 25 percent of the lateral dimension of the oropharynx as measured between the anterior tonsillar pillars.
(2+) Tonsils occupy less than 50 percent of the lateral dimension of the oropharynx.
(3+) Tonsils occupy less than 75 percent of the lateral dimension of the oropharynx.
(4+) Tonsils occupy 75 percent or more of the lateral dimension of the oropharynx.

CONCLUSIONS

OSA has significant life-threatening consequences, particularly for developing children. Furthermore, OSA among children can be the underlying cause of behavioral and attention problems as well as learning difficulties. One of the main causes of childhood OSA is adenotonsillar hypertrophy or enlargement of the adenotonsillar tissue. It is still unclear what proportions of children with adenotonsillar hypertrophy have OSA. However, in many cases, removal of the tonsils has been shown to relieve OSA symptoms. In identifying and treating OSA among children with adenotonsillar hypertrophy, Pediatric dentists can play an important role by noting the size of the tonsils when looking into children's mouths and informing the child's parents and the primary care physician when enlarged tonsils are observed and also the treatment of the same can be carried out by us using simple orthodontic procedures. But, first of all to comprehend the concept of SDB in children and its importance to dentistry needs to be stressed upon.

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