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REVIEW ARTICLE

Obstructive Sleep Apnea in Children

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ABSTRACT
Obstructive sleep apnea (OSA) is the consequence of a partial or complete intermittent cessation of airflow and brings on recurrent oxygen desaturations during sleep. The severity of OSA depends, in large part, from the patency of the high airway. In children, the major contributor to high airway obstruction is hyperplasia of pharyngeal tonsils and adenoids; craniofacial disharmony is also largely associated. Tonsil hypertrophy is considered the leading cause of OSA and tonsillar removal is the optional treatment. Hence; in the present review, we have summarized some of the important aspects of Obstructive sleep apnea.

KEY WORDS: Obstructive sleep apnea, Pediatric

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INTRODUCTION

Pharynx is a tube shaped structure formed by muscles and membrane which is divided into three parts: Nasopharynx, oropharynx and laryngopharynx. Nasopharynx and oropharynx have significant location and function because both of them form a part of unit in which processes like respiration and deglutition are carried out.[1-3]

Pediatric obstructive sleep apnea (OSA) was initially described in 1976. In 1981, Guilleminault et al. published a review of 50 pediatric patients and emphasized that pediatric OSA was different from the clinical presentation reported in adults. Obstructive sleep apnea (OSA) is the consequence of a partial or complete intermittent cessation of airflow and brings on recurrent oxygen desaturations during sleep. The severity of OSA depends, in large part, from the patency of the high airway. In children, the major contributor to high airway obstruction is hyperplasia of pharyngeal tonsils and adenoids; craniofacial disharmony is also largely associated. Most studies showed a prevalence of sleep-disordered breathing (SDB) between 1% and 4% in children.[4]

PATHOPHYSIOLOGY OF OBSTRUCTIVE SLEEP APNEA IN CHILDREN

The pathophysiology of OSA in children is a complex interaction between an airway predisposed toward collapse and neuromuscular compensation. Anatomic measures of the airway lumen, soft tissue, and skeleton are of critical importance to the development of OSA, although they do not completely account for the pattern of sleep-disordered breathing. This indicates a role for other determinants of airway patency such as neuromuscular activation, ventilatory control, and arousal threshold. At sleep onset, airway muscle activity is reduced, ventilatory variability increases, and an apneic threshold slightly below eupneic levels is observed in non-REM sleep. Airway collapse is offset by pharyngeal dilator activity in response to hypercapnia and negative lumenal pressure. Ventilatory overshoot results in sudden reduction in airway muscle activation, contributing to obstruction during non-REM sleep. Most children with severe OSA will be able to sustain stable breathing during a large portion of sleep, consistent with successful neuromuscular compensation. Respiratory control mechanisms modulate ventilation and pharyngeal dilator activation. Arousal from sleep contributes to ventilatory

instability and, thus, obstructive cycling. Paroxysmal reductions in pharyngeal dilator activity related to central REM sleep processes likely account for the disproportionate severity of OSA observed during REM sleep.[5]

To appreciate the factors that contribute to airway collapse, it is helpful to contrast the breathing patterns during sleep in children with and without OSA. Normative data from several, large samples of non snoring, normal children indicate that (1) obstructive apneas and hypopneas rarely occur; (2) inspiratory flow limitation and respiratory effort–related arousals are uncommon; (3) oxygen saturation rarely drops below 90%, even during normal 10 to 15 second respiratory pauses following sighs or movements; and (4) during non-REM sleep, respiratory rate decreases and end-tidal CO2 increases 3-4 mm Hg. Thus, despite ethnic differences in these studies the findings were remarkably similar, indicating the rarity of sleep-disordered breathing in normal children.[6, 7]

CLINICAL MANIFESTATIONS OF OBSTRUCTIVE SLEEP APNEA

The presenting problem in children with sleep disordered breathing depends on the child's age. In children younger than five years, snoring is the most common complaint. Other night time symptoms frequently reported by parents include mouth breathing, diaphoresis, paradoxic rib-cage movement, restlessness, frequent awakenings, and witnessed apnoeic episodes. Children five years and older commonly exhibit enuresis, behavior problems, deficient attention span, and failure to thrive, in addition to snoring. Compared with adults, fewer children with OSA report excessive daytime somnolence, with the notable exception of obese children. In extreme cases of OSA in children, cor pulmonale and pulmonary hypertension may be the presenting problems.[8]

CONSEQUENCES OF OBSTRUCTIVE SLEEP APNEA

We have begun to understand that sleep disorders in general, and more particularly, sleep-disordered breathing, can lead to substantial morbidities affecting the central nervous system (CNS), the cardiovascular and metabolic systems, and somatic growth, ultimately leading to reduced quality of life. On the basis of a series of elegant studies using rodent models of OSA, it is highly plausible that many if not all of these end-organ consequences impart common pathogenic mechanisms triggered by the interactions of intermittent hypoxia and hypercapnia, repeated intrathoracic pressure swings, and episodic arousal.[8]

- Neurobehavioral consequences
- Excessive daytime sleepiness
- Cardiovascular consequences
- Quality of life and depression
- Insulin resistance, type 2 diabetes, and metabolic syndrome
- Somatic growth impairment

MECHANISMS FOR INCREASED RISK OF OSA IN OBESE CHILDREN AND ADOLESCENTS

There are multiple factors that interact to significantly increase the risk of OSA among obese children and adolescents. Similar to non obese children, airway obstruction by adenotonsillar hypertrophy is a fairly common cause of OSA among obese children affecting approximately 45% of all obese children with OSA. However, alarmingly, following adenotonsillectomy, OSA persists in about 50% of obese children which is significantly higher than the observed persistence rate of 10–20% amongst non obese children. Another additional interesting observation is that the prevalence of adenotonsillar hypertrophy among obese children is higher than among non obese children, which indirectly suggests that adenotonsillar hypertrophy in obese children could be a consequence of another distinct mechanism. Possible explanations include endocrine mediated somatic growth that results in larger and/or heavier fat pads, soft palate, and tongue among adults with obesity. It is possible that similar mechanisms operate in children and adolescents as well. Functional factors that operate to promote upper airway obstruction OSA in obese individuals during sleep include altered neuromuscular tone resulting in greater upper airway collapsibility during sleep. Indeed, measurements of airway flow and mechanics have shown that in obese children, there is a positive critical closing pressure of the pharynx causing the airway to collapse during sleep with even mild negative inspiratory pressure.[9- 11]

PREVALENCE OF MALOCCLUSION WITH OBSTRUCTIVE SLEEP APNEA

There is a significantly higher prevalence of malocclusion in OSA children compared to the normal children. The results of Angela Galeotti et al. demonstrate a significantly higher prevalence of posterior cross bite in OSA children compared to controls and a strong association between posterior crossbite and OSA. This finding is consistent with previous studies that show an association between snoring and cross bite. As previously reported, the presence of a posterior cross bite is related to the altered equilibrium between the tongue and cheeks. The low and anterior position of the tongue related to mouth breathing results in a lack of internal pressure, leading to a reduction of transversal growth of the upper arch with the development of lateral and posterior crossbite. On the other hand, mouth breathing is associated with decreased nose prominence and width dimensions compared to normal children. These facial characteristics could lead to a reduced upper airway space resulting in obstructive apnoea events.[10- 12]

METHODS OF DIAGNOSIS OF OBSTRUCTIVE SLEEP APNEA

Diagnostic methods that have been scientifically evaluated include history and physical examination, audiotaping or videotaping, pulse oximetry, abbreviated polysomnography, and full polysomnography. The goals of diagnosis are to 1) identify patients who are at risk for

adverse outcomes; 2) avoid unnecessary intervention in patients who are not at risk for adverse outcomes; and 3) evaluate which patients are at increased risk of complications resulting from adenotonsillectomy so that appropriate precautions can be taken.[13]

- History and physical examination
- Nocturnal polysomnography
- Audiotaping or videotaping
- Abbreviated polysomnography

MANAGEMENT OF OBSTRUCTIVE SLEEP APNEA

Treatment options for pediatric OSA fall into four main categories: AT, anti-inflammatory agents, continuous positive airway pressure (CPAP) and dental/orthodontic treatments. The choice of treatment will depend on the severity of the disorder, the presence of comorbid conditions, and the physical examination and history findings.[14]

ADENOTONSILLECTOMY (AT)

Past studies confirm results from earlier research that overwhelmingly concur that AT significantly improves the severity of OSA in children with tonsillar hypertrophy, but that there are still significant numbers of children with residual OSA post-AT, especially obese children.[14]

CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP) TREATMENT

CPAP treatment is based on the premise that a column of air pressure acts as a splint to the upper airway, preventing collapse. It is delivered non-invasively using a mask applied to the nose and/or mouth and is used as a treatment in children when AT or other therapies have not resulted in sufficient clinical improvement, or in cases when surgery is not indicated. Children who are obese, or those with craniofacial abnormalities or neuromuscular disorders are the most common candidates for CPAP treatment. CPAP was first used in adults with OSA in 1981 and in children a few years later. CPAP pressure needs to be individualized, usually by manual adjustment during a polysomnographic study until airway obstruction is resolved.[15]

CORTICOSTEROIDS & LEUKOTRIENE MODIFIERS

Corticosteroids have been shown to reduce tonsillar proliferation in vitro, in tissue samples collected from children with OSA during AT. Administration of systemic corticosteroids has been shown to lead to a reduction in the size of lymphoid tissues due to anti-inflammatory and lympholytic effects; however, a short course of systemic prednisone was found not to have a significant effect on adenoidal size or the severity of OSA. Furthermore, adverse effects preclude the long-term use of this therapy for OSA in children.[16]

DENTAL/ORTHODONTIC TREATMENTS

A common phenotype in children with OSA is a narrow upper airway with maxillary constriction, a high arched palate and/or retrognathia and increased posterior facial height. Mandibular advancement splints (MAS) are indicated in adults with mild-to-moderate OSA as an alternative to CPAP and are a particularly attractive option in children with retrognathia. For this treatment, as for MAS, careful clinical assessment, including of the dentition and involvement of a multidisciplinary team, are more likely to lead to the best treatment outcomes.[17]

MYOFUNCTIONAL APPLIANCES IN MANAGEMENT OF OBSTRUCTIVE SLEEP APNEA

The most common non-surgical types of treatment include devices of air pressure (CPAP or BPAP), however, they are expensive and little accepted by children. Recurrence of the clinical condition can happen after adenotonsillectomy, and it is believed to be due to concomitant craniofacial problems, among others. These alterations can be easily recognized and treated by the orthodontist. The persistence of Obstructive Breathing(OB) and Persistant Snoring(PS) during the growing and developmental period may lead or exacerbate dental skeletal changes. The incurred changes coupled with genetic predisposition make the OSA even more severe, allowing the development of a vicious circle. Orthodontic appliances can be used before or after surgery as preventive or curative.[17]

The Funtional Appliances are widely used in children to promote mandibular growth and to improve craniofacial changes. The mandibular advancement devices protrude the mandible and the tongue, increasing the passage diameter of the UA, improving the tonicity of the muscles in the region, particularly the genioglossus muscle, and consequently preventing the collapse of the soft tissue.[17]

EFFECTS OF TWIN BLOCK IN OBSTRUCTIVE SLEEP APNEA

Myofunctional appliances are the choice of treatment where Class II skeletal pattern is due to retrognathic mandible. These appliances reposition the tongue and mandible to a new position leading to different changes in airway dimensions. Several studies, have reported that modifications of myofunctional appliances are effective in treatment of obstructive sleep apnea by improving the airway. Twin Blocks designed by Clark are traditionally used in treatment of children with class II skeletal relationships which proved to be the best tolerated as well as most robust of all functional appliances. It allows the mandible to be gradually advanced to a position of maximum comfortable protrusion which is associated with optimal reduction in OSA symptoms. The Twin Block (TB) appliance is a well-accepted approach in correcting Class II division 1 malocclusion with mandibular retrusion in recent years. Previous studies indicated that TB is effective in mandibular forward repositioning and thereby achieves a more harmonious facial profile. The upper airway changes in adult patients

with OSA were measured after mandibular repositioning treatment, and showed a significant enlargement in pharyngeal volume.[18, 19]

EFFECTS OF ACTIVATOR IN OBSTRUCTIVE SLEEP APNEA

Functional orthopedic treatment with activator in growing children is known to be an effective method to enhance sagital mandibular growth and correct skeletal Class 2 problems due to retrognathic mandible. It was previously reported that these appliances can also alter airway dimensions positively and prohibit possible obstructions in the future. Still studies have limitations due to the lack of control groups or reporting only short-term effects. Retention is desirable in functional therapies until the end of adolescence and can be made by night-time wearing of the appliance either in the original way or cut down. Some studies were set up to evaluate the effects of activator treatment on the pharyngeal airway morphology on a long-term basis and compare it with an untreated control group. Their results showed that prominent skeletal changes were mainly occurred in the retention period with respect to the observation period. Although activator treatment induced favorable changes in nasopharyngeal area, no significant difference was found when compared with the normal children neither in short nor in the long-term. This might be attributed to the fact that the patient was at the beginning of pubertal growth at the beginning of activator treatment, therefore significant changes in the airway size related to the growth process were not expected. However, a combination of growth changes and the effects of functional appliance might be considered as favorable in nasopharyngeal and oropharyngeal area throughout the retention period. Additionally, cases with retrognathic facial structures and/or patients with sleep disorders due to relatively smaller pharyngeal dimensions would have demonstrated greater advancement of airway dimensions and reveal greater intrinsic stimulus to increase their capacity. Snoring and OSA are described as two aspects of the same basic disorder, namely sleep-related narrowing of the upper airways which differ only in severity. The patency of the upper airway depends on the balance between the negative intrapharyngeal pressure developed during inspiration and its counteraction by dilating muscles. It is clear that upper airway collapse most often results from a combination of anatomical factors that predispose the airway to collapse during inspiration, plus neuromuscular compensation that is insufficient during sleep to maintain airway patency. Therefore, it is possible that small pharyngeal dimensions established early in life may predispose to OSA and snoring later when subsequent soft tissue changes caused by age, obesity, or genetic background further reduce the available oropharyngeal airway.[20, 21]

EFFECTS OF BIONATOR IN OBSTRUCTIVE SLEEP APNEA

The bionator, a functional appliance considered to stimulate mandibular growth in growing patients with retrognathia, is widely used in daily orthodontic practice. The development of the bionator is credited to Wilhelm Balters, who emphasized that equilibrium between the tongue and circumoral buccinator mechanism was responsible for the shape of the dental arches and intercuspation. His main objective of Angle class II malocclusion treatment was to bring the tongue forward by stimulating the distal part of the dorsum of the tongue and developing the mandible in an anterior position to achieve an Angle class I relationship. The modified bionator had different influences on the pharyngeal airway in growing patients. During bionator treatment, because of the connection of the lateral wall of the soft palate to the base of the tongue through the palatoglossal arch, mandibular advancement stretched the soft palate and stiffened the velopharynx, which caused the oropharyngeal airway dimension to appear not to have changed during growth. In one of the previous study, reported that mandibular-advancement oral appliances increased the lateral dimension of the velopharynx more than the anteroposterior dimension in adult OSAHS individuals.[18, 19]

EFFECTS OF FIXED FUNCTIONAL APPLIACES IN OBSTRUCTIVE SLEEP APNEA

The aim of fixed functional therapy in young adults is to achieve a neutral bite by dentoalveolar compensation for the skeletal discrepancy; mainly, the upper dentition is moved to the distal and the lower dentition is moved to the mesial with a clockwise rotation of the occlusal plane and without creating pronounced vertical skeletal changes, only inhibiting the sagittal growth potential of maxilla. The minimal skeletal mandibular effects depend on the device used and the age and growth capacity of the patient. The lower incisors procline and intrude, while the lower molars move significantly in a mesial and vertical direction. As there are studies reporting on changes of the posterior airway after dental movements only, the amount of dental movement induced by these appliances in post-peak patients is large enough to justify the investigation of related changes of the airway. Most patients undergoing fixed functional therapy have retrognathic mandibles, and because this is noted as one of the risk factors for sleep apnea, it is logical to investigate how these appliances potentially affect the airway. Recent research suggests that airway changes induced by orthodontic movements affect the room for the tongue, thereby affecting the position of the hyoid bone and causing a subsequent change in the dimensions of the posterior airway.[20, 21]

MYOFUNCTIONAL THERAPY AND OBSTRUCTIVE SLEEP APNEA

Myofunctional therapy (MT) and proper tongue positioning in the oral cavity have been described since 1918 to improve mandibular growth, nasal breathing, and facial appearance. In most of the studies reviewed, the therapeutic program for adults lasted three months, with a weekly visit and training at home for three to five times a day. The authors adopted systems of control of adherence

to treatment considering the frequency of supervised sessions and a diary indicating home practice. Only one pilot study lasted two months, with training at the clinic for 5 min twice a day and four times per week.[22]

The principle of OMT (oropharyngeal exercises) is repetitive muscle training, with specific gains in the coordination, tonicity, and endurance of the muscles, considering the specificity of the exercises adopted (isotonic and isometric). The exercises may improve the condition of muscle fatigue in subjects with OSA and perhaps act on the equilibrium of contraction between the different muscles that involve the velopharyngeal, oropharyngeal, and hypopharyngeal segments. Further, they can decrease the volume of specific structures and fat in the pharynx-dilating muscles, thus also reducing the potential upper airway collapse in apneic subjects. However, these hypotheses have not been verified so far.[23, 24]

CONCLUSION

OSA has a negative impact on child growth, affecting their quality of life. If the condition persists, it may affect the quality of life in their adulthood. Tonsil hypertrophy is considered the leading cause of OSA and tonsillar removal is the optional treatment. Studies have shown that the use of FA can eliminate or reduce the symptoms of the OSA, promoting a better long-term quality of life.

REFERENCES

1. Filho DI, Ravell DB, Ravell RB, Loffredo LDCM, Gandini LG. A comparison of nasopharyngeal endoscopy and lateral cephalometric radiography in the diagnosis of nasopharyngeal airway obstruction. Am J Orthod Dentofacial Orthop. 2001;120:348-52.
2. Isono S, Tanaka A, Sho Y, Konno A, and Nishino T. Advancement of the mandible improves velopharyngeal airway patency. J Appl Physio. 1995;79(6):2132-2138.
3. Uliel S, Tauman R, Greenfeld M, Sivan Y. Normal polysomnographic respiratory values in children and adolescents. Chest 2004;125:872-878.
4. Chervin RD, Burns JW, Subotic NS, Roussi C, Thelen B, Ruzicka DL. Correlates of respiratory cycle–related EEG changes in children with sleep-disordered breathing. Sleep 2004;27:116-121.
5. Brouillette RT, Manoukian JJ, Ducharme FM, Oudjhane K, Earle LG, Ladan S, Morielli A. Efficacy of fluticasone nasal spray for pediatric obstructive sleep apnea. J Pediatr 2001;138:838-844.
6. Gozal D, Wang M, Pope DW Jr. Objective sleepiness measures in pediatric obstructive sleep apnea. Pediatrics 2001;108:693-7.
7. Almendros I, Acerbi I, Puig F, Montserrat JM, Navajas D, Farre R. Upper-airway inflammation triggered by vibration in a rat model of snoring. Sleep 2007;30:225-227.
8. Gozal D, Crabtree VM, Sans Capdevila O, Witcher LA, Kheirandish- Gozal L. C-reactive protein, obstructive sleep apnea, and cognitive dysfunction in school-aged children. Am J Respir Crit Care Med 2007;176:188-193.
9. Crabtree VM, Varni JW, Gozal D. Health-related quality of life and depressive symptoms in children with suspected sleep-disordered breathing. Sleep 2004;27:1131-1138.
10. Ye J. Emerging role of adipose tissue hypoxia in obesity and insulin resistance. Int J Obes 2009;33:54-66.
11. Cazzolla AP, Lacarbonara V, Pellegrino B, Testa NF, Fidanza F, Lacaita MG. Sleep-disordered breathing in a sample of 495 children in Southern Italy. Eur J Paediatr Dent. 2010;11:189-192.
12. American Thoracic Society. Cardiorespiratory sleep studies in children: establishment of normative data and polysomnographic predictors of morbidity. Am J Respir Crit Care Med. 1999;160:1381-1387
13. Carroll JL. Obstructive sleep-disordered breathing in children: new controversies, new directions. Clin Chest Med 2003;24:261-82.
14. Pirttiniemi P. Cephalometric evaluation of children with nocturnal sleep-disordered breathing. Eur J Orthod 2010;32:662-71.
15. Pracharktam N, Hans MG, Strohl KP, Redline S. Upright and supine cephalometric evaluation of obstructive sleep apnea syndrome and snoring subjects. Angle Orthod 1994;64:63-73.
16. O'Donnell AR, Bjornson CL, Bohn SG et al. Compliance rates in children using noninvasive continuous positive airway pressure. Sleep 2006;29(5):651-658.
17. Nixon GM, Brouillette RT. Obstructive sleep apnea in children: do intranasal corticosteroids help? Am. J. Respir. Med. 2002;1(3):159-166.
18. Johal A, Battagel JM. An investigation into the changes in airway dimension and the efficacy of mandibular advancement appliances in subjects with obstructive sleep apnoea. Br J Orthod 1999;26:205-10.
19. Preston BC, Tobias VP, Salem HO. Skeletal age and growth of the nasopharynx in the sagittal plane: a cephalometric study. Semin Orthod 2004;10:16-38.
20. Zhou L, Zhao Z, Lu D. [The analysis of the changes of tongue shape and position, hyoid position in Class II, division 1 malocclusion treated with functional appliances (FR-I)]. Hua Xi Kou Qiang Yi Xue Za Zhi 2000;18:123-5.
21. Jena AK, Singh SP, Utreja AK. Effectiveness of twin-block and Mandibular Protraction Appliance-IV in the improvement of pharyngeal airway passage dimensions in Class II malocclusion subjects with a retrognathic mandible. Angle Orthod 2013;83:728-34.
22. Quinn GW. Airway interferences and its effect upon the growth and development of the face, jaws, dentition and associated parts. N C Dent J 1978;60:28‐31.
23. Fransson AM, Tegelberg A, Svenson BA, Influence of mandibular protruding device on airway passages and dentofacial characteristics in obstructive sleep apnea and snoring. Am J Orthod Dentofacial Orthop 2003;122:371‐9.
24. Li Y. Early orthodontic treatment of skeletal Class II malocclusion may be effective to prevent the potential for OSAHS and snoring. Med Hypotheses 2009;73:594-5.