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Review Article

Pediatric Obstructive Sleep Apnea Syndrome: "Wake Up Before It's Too Late"

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Abstract

Pediatric Obstructive Sleep Apnea is a part of the spectrum of Sleep-Disordered Breathing that is characterized by frequent arousals, apneas, and hypopneas. It can be associated with reduction in blood oxygen saturation and hypoventilation during sleep in children. If left untreated or adequately treated, it can lead to long-term sequelae involving cardiovascular, endothelial, metabolic, endocrine, neurocognitive, and psychological consequences affecting the overall growth and development of children. For this study, an online search of databases such as PubMed, Scopus, Science Direct, ProQuest, Medline and Google Scholar has been done and as a result of this search, scientific evidences describing the etiopathogenesis, pathophysiology, clinical manifestations, and management of such serious illnesses have been found. This article focuses on the several aspects of pediatric obstructive sleep apnea with special emphasis on the identification and management of the condition.

Keywords: Myofunctional therapy, obstructive sleep apnea, oral appliance therapy, pediatric obstructive sleep apnea, sleep disordered breathing

Introduction

Sleep is an active and cyclic physiological process that has a critical impact on health. During the first years of life, a human experiences a number of important changes which affect his/her development. The result of these changes can lead to the expected pattern of sleep or wakefulness in adults.[1] Sleep Disordered Breathing (SDB) and Pediatric Obstructive Sleep Apnea (POSA) have started to attract increased public attention in recent years and they now can be considered as major medical concerns and research fields.[2] The terms can be correlated with a spectrum of sleep-related breathing abnormalities that include snoring, upper airway resistance syndrome, obstructive hypopnea syndrome, and Obstructive Sleep Apnea (OSA). SDB in children is a significant public health concern due to the high frequency of comorbidities such as neurocognitive impairment, cardiovascular problems, and obesity. Obstructive Sleep Apnea syndrome (OSAS) is defined as a disorder of breathing during sleep and its characterizations are prolonged partial airway obstruction and intermittent complete obstruction (obstructive apnea) that interrupts normal ventilation during sleep and normal sleep patterns.[2] The American Academy of Pediatric Dentistry (AAPD) recognizes that OSAS occurs in the pediatric population.[3]

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Epidemiology

POSA, the most severe form of SDB, is a quite common disease, affecting 1% to 4% of all the children worldwide.[3] There are two peaks in the prevalence of OSAS. The first peak occurs in youngsters between the ages of 2 and 8 when the adenoid and/or tonsils are swollen. During adolescence, a second peak appears with weight growth. Although there are no gender differences in the prevalence of OSAS among prepubescent children, certain studies have found that adolescent boys are more afflicted than girls.[4] In adults, most studies have found that men had a twofold to threefold higher risk than women.[5] POSA affects approximately 7 to 9 million children, leading medical and dental professionals to try to raise awareness about proper screening, diagnosis, and treatment.[3] Children of African-American and Asian descent, children with respiratory diseases such as allergic rhinitis and asthma, obese children, and children with a family history of OSA had higher rates of POSA.[6] Although adenotonsillar hypertrophy remains the most prevalent cause of POSA, the rise in childhood obesity has resulted in a large increase in the prevalence of POSA in all children, including teenagers.

Etiopathogenesis

The etiology of POSA in children is multifactorial and it includes an intricate interaction between hypertrophy of the tonsils, craniofacial limitations and alterations in tonicity of the neuromuscular system.

Various other possible causes of childhood sleep apnea include (Fig. 1):

- Enlarged structures obstructing the back of the throat, such as the tongue and tonsils, or in the back of the nose, such as adenoids.
- Hay fever and long-term allergies, childhood obesity and low muscle tone or weak muscles.
- Craniofacial Disorders which result in growth deformities in the upper jaw or airway, such as a narrow palate or small patent airway. These can also block airflow to the lungs.
- Neuromuscular Disorders.
- Premature Births.[7,8]

The pathophysiology of childhood OSAS is influenced by Adenotonsillar Hypertrophy. Obesity, craniofacial disease, and neuromuscular disease are other causes of childhood OSAS, but they are less common.[9] Large tonsils and adenoids are most likely present in the vast majority of children with OSAS. It was discovered before that when it comes to the children with OSAS, the site of upper airway closure is at the level of the tonsils and adenoids, whereas it was determined to be at the level of the soft palate in normal youngsters.[10] The most obvious symptom is that patients with OSAS do not have any obstruction issues during wakefulness when the tone of the upper airway muscles is increased. However, many studies have failed to show a correlation between upper airway or adenotonsillar size and OSAS.[11,12]

Although the overall ventilatory drive in children with OSAS appears to be normal, the central augmentation of upper airway neuromotor function is probably aberrant. Upper airway muscles are respiratory accessory muscles that are stimulated by hypoxemia, hypercapnia,[13] and upper airway sub atmospheric pressure, among with other stimuli. The level of activity of the upper airway dilator muscles[14-16] is inversely related to the upper airway's tendency to collapse. With this knowledge, higher upper airway neuromotor tone could be considered as an appropriate approach to adjust for patients with a constricted upper airway. Eventhough this compensatory mechanism could be observed in adults while awake, it was lost during sleep.[17]

Children, unlike adults, have a less compressible upper airway. The central ventilator drive, chemoreceptor afferents, upper airway pressure and flow receptors, pulmonary mechanoreceptors, and sleep state are all influencing factors for upper airway neuromotor activation in children. The central nervous system is thought to be important in maintaining upper airway patency. Experiments have shown that the pediatric upper airway has the ability to modify airflow in response to stimuli such as sub atmospheric pressure and CO_2. Furthermore, the results demonstrate that children have a higher basal upper airway tone during sleep, as well as the ability to enhance the tone even more in response to a stimulus. To compensate for a physically smaller upper airway, pharyngeal muscle activity appears to play a significant role in keeping upper airway patency in children during sleep.[18]

Children and adults have quite different arousal responses to obstructive apnea. Arousals are seldom experienced by children. In babies, less than 20% of obstructive apnea is related to arousal.[19] The lack of sleep fragmentation resulting with daytime somnolence in pediatric patients is likely due to this lack of cortical activation which occurs in response to airway constriction. It could also be thought as an explanation for children suffering from obstructive hypoventilation for long periods. Children's arousal thresholds are generally higher than adults'; the younger the child, the higher the arousal threshold is.[20]

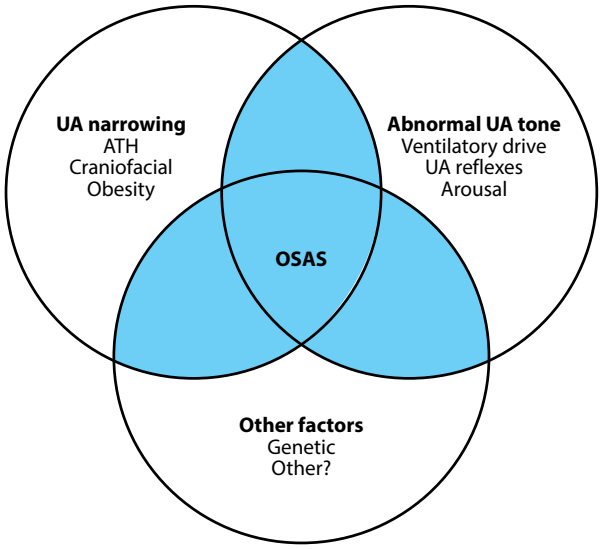

Figure 1. Pathophysiology of OSAS

OSAS: Obstructive Sleep Apnea syndrome; UA: Upper airway; ATH: Adenotonsillar hypertrophy

Table 1. According to American Academy of Pediatric Dentistry (2012)[21]

• Excessive daytime sleepiness.
• Loud snoring three or more nights per week.
• Episodes of breathing cessation witnessed by another person.
• Abrupt awakenings accompanied by shortness of breath.
• Awakening with dry mouth or sore throat.
• Morning headache.
• Difficulty staying asleep.
• Attention problems.
• Mouth breathing.
• Sweating.
• Restlessness.
• Waking up a lot.

Signs and symptoms

According to AAPD Guidelines Revised (2021), OSA symptoms include habitual snoring to occur in three or more nights per week (often associated with intermittent pauses, snorts, or gasps), disturbed sleep, nocturnal enuresis, sleeping in an unusual position with a hyperextended neck and mouth breathing (Table 1). Classic symptoms of daytime sleepiness may occur, but this is uncommon in young children. Neurocognitive dysfunction, such as aggressive behavior, symptoms that resemble attention deficit hyperactivity disorder, decreasing school performance, bedwetting, developmental delay, and reduced quality of life are all markers of untreated POSA in school-aged children according to the AAPD. Patients with POSA are also more likely to develop respiratory problems such as respiratory arrest followed by the need of general anesthesia or sedation (Table 2).[21]

Diagnosis

Early identification and treatment of pediatric OSA is required in order to prevent long-term complications and a proper diagnosis is a basis for correct and effective treatment. The American Academy of Pediatrics recommends that all children and adolescents should be screened for snoring. A thorough history and physical examination are integral to the initial evaluation of patients with suspected SDB. Controversy exists concerning the need for any type of test to be performed. Some of the measures used for this testing include questionnaires and scales, home monitoring and Polysomnography (PSG).[22-25] Although questionnaires may be helpful in directing the attention of parents to the diurnal and nocturnal symptoms of SDB (Table 3),[26] the sensitivity and specificity of the questionnaires are not sufficient for affirming the presence of SDB.[27-29]

Home monitoring with or without videotaping has also been used. Ambulatory monitoring with a recording of cardiac and respiratory variables has been suggested as the first diagnostic step in testing for SDB. These devices can detect the presence of drops in oxygen saturation, apneas and hypopneas; can affirm the diagnosis of SDB, and lead to treatment.[30-32] Polysomnography is the only test that may help with the diagnosis of SDB. The test includes monitoring of sleep/wake states through electroencephalography (EEG), electrooculography, chin and leg electromyography, electrocardiography, body position and appropriate monitoring of breathing.[18]

The suspicion of SDB indicates not only the need for a general pediatric evaluation but also for a thorough evaluation of the upper airway anatomy. Clinically, it involves a comprehensive examination of its successive segments.:

1. Some indicatives within the nose, such as asymmetry of the nares, a large septal base, the collapse of the nasal valves during inspiration, a deviated septum or enlargement of the inferior nasal turbinates should be looked for.
2. The oropharynx should be examined regarding the position of the uvula and its relation to the tongue. The scale developed by Mallampati et al may help to evaluate this position.
3. The size of the tonsils should be compared with the size of the airway.

Table 2. Signs and symptoms of POSA vary with the age[18]

Infants 3 to 12 months of age	Toddlers 1 to 3 years of age	Preschool-aged children	School- aged children
• Disturbed nocturnal sleep with repetitive crying	• Noisy breathing or snoring	• Regular heavy snoring	• Regular heavy snoring
• Poorly established day/ night cycle	• Agitated sleep or disrupted nocturnal	• Mouth breathing	• Agitated sleep
• Noisy breathing or snoring	• Crying spells or sleep terrors	• Drooling during sleep	• Abnormal sleeping positions
• Nocturnal sweating	• Grouchy or aggressive daytime behaviour	• Agitated sleep	• Insomnia
• Poor suck	• Daytime fatigue	• Nocturnal awakenings	• Delayed sleep phase syndrome
• Absence of normal growth pattern or failure to thrive	• Nocturnal sweating	• Confusional arousals	• Confusional arousal
• Observation of apnoeic events	• Mouth breathing	• Sleep walking	• Sleep walking, sleep talking
• Report of apparent life-threatening events	• Poor eating	• Sleep terrors	• Persistence of bed wetting
• Presence of repetitive earaches or URI	• Failure to thrive	• Nocturnal sweating	• Nocturnal sweating
	• Repetitive URI	• Abnormal sleeping positions	• Hard to wake up in the morning
	• Witnessed apnoeic episodes	• Persistence of bed wetting	• Mouth breathing, drooling
		• Abnormal daytime behaviour	• Morning headache
		• Aggressiveness	• Daytime fatigue
		• Hyperactivity	• Daytime sleepiness with regular napping
		• Inattention	• Abnormal daytime behaviours
		• Daytime fatigue	• Pattern of attention deficit/ hyperactivity disorder
		• Hard to wake up in the morning	• Aggressiveness
		• Morning headache	• Abnormal shyness withdrawn and depressive presentation
		• Increased need for napping compared with peers	• Learning difficulties
		• Poor eating	• Abnormal growth patterns
		• Growth problems	• Delayed puberty
		• Frequent URI	• Repetitive URI
			• Dental problems appreciated by dentist
			• Crossbite
			• Malocclusion (II, III)
			• Small jaw

POSA: Pediatric Obstructive Sleep Apnea

4. An examination should be made in case a high and narrow hard palate, overlapping incisors, a crossbite and an important (2 mm) overjet (the horizontal distance between the upper and lower teeth) are present since these are indicatives of a small jaw and/ or an abnormal maxilla-mandibular development.

The polysomnography test is the gold standard for diagnosing OSA in both children and adults. Currently, a variety of surgical and non-surgical treatments are being used to treat POSA. Although it often improves symptoms, adenotonsillectomy (AT), the first line of treatment for POSA in children, may not be completely

Table 3. Diagnostic criteria of OSAS in children[32]

Frequent signs	Infrequent signs
• Nocturnal snoring	• Daytime sleepiness
• Mouth breathing	• Decreased appetite
• Restless sleep with or without arousals	• Failure to thrive without arousals
• Respiratory pauses	• Frequent vomiting
• Respiratory infections	• Swallowing dysfunction
• Chronic rhinorrhoea	• Behavioral problems
• Nocturnal sweating	• Otitis media
	• Enuresis

OSAS: Obstructive Sleep Apnea syndrome

Table 4. Surgical therapies for OSAS in children[36]

Therapy	Population	Benefits	Risks/challenges
Adenotonsillectomy (AT)	Children with enlarged tonsils and/ or adenoids	Highly effective; well-tolerated in most children	Common: Pain, decreased oral tolerance, rarely hemorrhage, respiratory complications, etc.
Partial tonsillectomy (PT) and adenoidectomy	Children with enlarged tonsils±adenoids	Shorter recovery time than extracapsular tonsillectomy	Efficacy in treating OSAS less-established; effect of tonsillar regrowth on OSAS unknown
Lingual tonsillectomy	Persistent OSAS after AT with enlarged lingual tonsils	Definitive therapy for residual OSAS	Concentric scarring in airway; efficacy/ideal population not well-established in OSAS
Tracheostomy	Children with severe OSAS and no other therapeutic option	Highly effective	Requires increased monitoring at home; increased risk of significant complications
Bariatric Surgery	Select obese teenagers that have failed other therapies	Small studies show high short term success rate in select populations	Significant complications; no long-term efficacy data, success varies by center/type of surgery
Craniofacial surgery	Select children with craniofacial conditions	Highly effective in select populations	Minimal long-term follow-up data; success varies by center/ type of surgery; significant morbidity

OSAS: Obstructive Sleep Apnea syndrome

curative. According to the American Association of Sleep Apnea, depending on the situation, therapeutic options include positive airway pressure (PAP), weight loss in obese children, and additional treatment measures such as dental therapies. The diagnosis of suspected sleep disturbances in children and infants is hampered because they are frequently ignored and underreported. Physicians should follow the clinical practice recommendations developed by the American Academy of Pediatrics (AAP) in detecting and managing young children with OSA. Dentists should also be aware of the guidelines. For the diagnosis of OSA, these guidelines' recommendations are as follows:[33]

1. All children should be screened for snoring.
2. Complex, high-risk patients should be referred to a specialist.
3. Patients with cardiorespiratory failure cannot await elective evaluation.
4. Diagnostic evaluation is useful in discriminating between primary snoring and OSA, with the gold standard being polysomnography.
5. Adenotonsillectomy remains the initial treatment 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. Patients should be re-evaluated postoperatively to determine whether additional treatment is required.

The use of polysomnography in children has been studied. Pang and Balakrishnan[34] questioned the routine need for mandatory overnight polysomnogra-

Table 5. Non-surgical therapies for OSAS in children[36]

Therapy	Population	Benefits	Risks/challenges
Positive airway pressure (PAP)	Any child	Strong evidence for efficacy, even if OSAS is severe	Some will have trouble tolerating; few mask options for some children
Nasal steroids and leukotriene receptor antagonists	Children with mild-moderate OSAS	Minimally invasive	Weak evidence; length of therapy needed/ideal population unclear
Rapid maxillary expansion/ oral appliances	Children with narrow maxilla or constricted maxillary arch, non-obese, without adenotonsillar enlargement	Therapy is short term, minimally invasive, may be easier to tolerate than PAP	Few studies showing efficacy; unclear which children will benefit most; discomfort; potentially expensive
Supplemental oxygen	Unclear; possible infants or those with no other therapeutic options	May prevent hypoxemia	Dose not treat airway obstruction; risk of hypercapnia
Weight loss	Older, obese children	Non-invasive, good for overall health; can be done in conjunction with PAP	Difficult, no evidence for sustained resolution of OSAS

OSAS: Obstructive Sleep Apnea syndrome

phy prior to an adenotonsillectomy. Objective testing with polysomnography before adenotonsillectomy, however, has been recommended for SDB children by the AAP. Katz et al[35] found little clinical significance in night-to-night variability in pediatric polysomnography and found no first night effect. Caution must be used when diagnosing children with symptoms suggestive of OSA since another study found that half or fewer of these children had the condition when they were examined with polysomnography.

Polysomnographic differences between children and adults with OSAS

The American Thoracic Society has defined its criteria for an abnormal PSG in children as follows:[26]

- Apnea index (AI) 1/hour.
- Apnea-hypopnea index 5/hour.
- Peak end-tidal carbon dioxide of 53 mm Hg or an end-tidal carbon dioxide tension of 50 mm Hg for 10% of the sleep period.
- A minimum hemoglobin oxygen saturation of 92%.

Management

For purposes of simplicity, treatment for POSA is divided into surgical and non-surgical methods through this research. The decision as to which modality is pursued is dependent on the severity and etiology of the patient's POSA and in the case of non-surgical treatment, the patient's compliance. Thus, it may involve a combination of both surgical and non-surgical techniques. The main focus of the various treatment modalities is to uphold the upper-airway patency, especially during sleep. To achieve this, various surgical procedures have been attempted for improving the anatomy of the upper airway such as CPAP or the other devices that help in maintaining the patency of an upper airway mechanically during sleep.

Surgical considerations

Generally, the first line of therapy in the treatment of POSA consists of surgically removing the hypertrophic adenoids and tonsils. If POSA has been diagnosed in a child, and if a child has adenotonsillar hypertrophy, unless contraindicated, Adenotonsillectomy (AT) is considered as the first-line therapy. Tonsillectomy is a surgical procedure where the peritonsillar space between the tonsillar capsule and muscular wall is dissected and the tonsil is completely removed. When performed in conjunction with the removal of the adenoids, this procedure is referred to as AT. If a child has OSAS and has adenotonsillar hypertrophy, AT is considered as the first-line therapy. AT is the most common treatment modality for OSAS in children. In the literature, either tonsillectomy and adenoidectomy procedures have been reported to give superior results with a success rate of 80% when performed alone (Table 4).[36]

Adenotonsillectomy (AT)

Most children will benefit from adenotonsillectomy. However, individuals who are not candidates for surgery or who do not respond to surgery may benefit from continuous positive airway pressure. Uvulopalatopharyngoplasty, tracheotomy, and other surgical procedures are less frequently indicated.

Tracheostomy

Tracheostomy is the most aggressive of the aforementioned techniques but is suggested as a cure for OSA as

it evades the upper airway. However, it is associated with significant social and morbidity risks including infection of the stoma (36%), hemorrhage (5%), tracheal stenosis (2%), and psychosocial difficulties. This surgical method is more of a temporary solution for protecting the airway in patients with severe sleep apnea due to comorbidities including morbid obesity and craniofacial syndromes. It may be used as a last resort for patients who have undergone other unsuccessful surgical and non-surgical procedures.[37]

Uvulopalatopharyngoplasty (UPPP)

The UPPP procedure is estimated to be only 40% to 50% successful in improving mild to moderate OSA. Its effectiveness decreases over time, adding substantial postoperative complications including velopharyngeal insufficiency (reported at 2%), postoperative hemorrhage (reported at 1%), continued dryness of the airway, dysphagia, voice change (reported at 0.6%), narrowing of the nasopharyngeal walls (reported at 0.8%), and death (reported at 0.2%).[3] A revision of this technique is called a uvulopalatal flap and is preferred over UPPP in most cases because it lowers the risk of velopharyngeal insufficiency as it creates a potentially reversible flap that can be altered during the initial postoperative period. Since there are no sutures involved along the free edge of the palate, in comparison with UPPP, the reported pain was less. However, this technique still imposes the same risks of complications as seen with UPPP and is typically performed on adults only. Ablation has an advantage over electrocautery and laser applications because of its lower temperature, safety, and overall precision without affecting speech or swallowing. The only downside to this technique is that it requires multiple treatments over weeks, and although uncommon, may result in ulceration and infection.[38]

Mandibular distraction osteogenesis

Mandibular distraction osteogenesis is another treatment alternative for grown up children in whom fused cartilage has been seen and also for the patients who do not require maxillomandibular advancement. Distraction osteogenesis, by a method of rapid maxillary distraction combined with an AT, has proved to be successful in treating children with OSA who has the following facial features: High arched palate and a unilateral or bilateral posterior crossbite in patients who are at least 5 to 16 years of age (before the cartilage becomes bone).[3] As discussed previously, syndromic patients may benefit from mandibular distraction osteogenesis by expanding the bones in the mandible via internal or external fixation (external fixation results in more scarring). These distraction strategies work by allowing increased space for the tongue to lie so that it does not fall posteriorly and obstruct the oropharynx. Rapid maxillary distraction may be performed in congruence with mandibular distraction osteogenesis when the patient has reached the age of adolescence (approximately 12 to 13 years).[38,39]

Non-surgical medical considerations

As measures to diagnose POSA have evolved, so have the treatment modalities including several non-surgical approaches which, in some cases, replace or serve as an adjunctive to AT. Since obesity is the second most common cause of POSA, weight reduction is a common non-surgical recommendation for these patients. However, other therapeutic strategies may be used concurrently to address the patient's POSA while weight reduction is being pursued. Non-surgical treatment options include PAP, myofunctional therapy, and if indicated, maxillary expansion along with other dental/orthodontic treatments (Table 5).[36] This highlights the need for multidisciplinary approaches for the treatment of POSA. Teams consisting of primary care physicians, otolaryngologists, sleep medicine physicians, myofunctional therapists, and dentists (including general dentists, pediatric dentists, and orthodontists) have become much more common.

Continuous positive airway pressure via nasal mask

Mechanically bypassing the obstruction with Continuous Positive Airway Pressure (CPAP) and Bilevel Positive Airway Pressure (BiPAP) has been used successfully in children, although difficulty in finding appropriately fitting equipment may contribute to problems with adherence. It should be noted that this method is only palliative in nature and does not cure the underlying cause of the obstruction. Downey et al[40] showed that CPAP can be effectively used in children younger than 2 years of age. Palombini et al[41] found that, in children 8 months of age to 12 years of age, Auto-CPAP was safe and effective in an attended environment. Auto-CPAP, however, did not eliminate all abnormal respiratory events.

Nasal steroids

Another non-surgical 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 in cases where adenoidal regrowth following AT is present, or when AT cannot be performed, intranasal steroids are often indicated. The rationale for these approaches centers on the premise that these therapies reduce the inflammatory proliferation of adenotonsillar tissue, which often leads to a reduction in their size. A potential limitation of this treatment modality is that children are at risk for growth and adrenal disturbances if intranasal steroids are used for extended periods, and cause unwanted side effects including nasal irritation and epistaxis. Studies utilizing combination therapy with montelukast and intranasal steroids, such as budesonide in patients with unresolved POSA following AT, have had positive results, showing even further reductions in AHI.[36]

Oral appliance therapy (OAT)

Rapid maxillary distraction (RMD) is an orthodontic technique that is based on the bone formation process. This technique pushes the soft tissues laterally, decreases the height of the soft palate, and enlarges the nasal orifices. Rapid maxillary distraction may be associated with the distraction of the mandible, but because no mid cartilage is present, there is very limited widening. Slow maxillary distraction is based on similar principles and optimizes the degree of widening at the different growth periods that occur in prepubertal children. Rapid and slow maxillary distractions are performed between 5 and 11 years of age.[36] Distraction results in widening of the palate and the nose; thus, these procedures remedy nasal occlusion related to a deviated septum, for which little can be done before 14 to 16 years of age.[36]

Appliances for habits such as bruxism fall under "occlusal guards by report" or "lab occlusal guard-nightguard". Utilizing Oral Appliance Therapy appliances for myofunctional purposes or bruxism is typically not a current covered benefit for patients, making it difficult for parents to endure an out-of-pocket expense for their child. Cozza et al[42] found that a modified monobloc device may be useful in children with mild to moderate OSA. The authors suggest further long-term studies prior to wide use of these devices, however, because of concerns with the effects on growth patterns and compliance issues.

Nasal temporizing measures

In infants and children with craniofacial conditions and with obstructive sleep apnea, measures to stent the nasal passages may be used as temporizing measures while the patient grows or until definitive surgical therapy. Potential therapies include stents, nasopharyngeal airways, balloons, nostril retainers, and nasoalveolar molding devices. The use of these devices is limited to specialized populations and in general outcomes specific to OSAS, which have not been well studied yet.[36]

Supplemental oxygen

While not reducing the airway obstruction, supplemental oxygen can be used to normalize oxyhemoglobin saturation during sleep in children with OSAS. With the paucity of data available, supplemental oxygen therapy for OSAS should be used with caution due to the concerns for hypercapnia, which has been described, and the potential for oxygen toxicity and its inability to treat upper airway obstruction.

Weight loss

Weight loss has been shown to be an effective means of improving, but not curing, OSAS in adults. In a study where 49 obese children with OSAS who were between 10 to 18 years of age were observed, weight loss was associated with the resolution of OSAS in 71% of the children.[43] However, sustained weight loss is difficult without continued intervention. The limited data available suggests that weight loss could be considered as an adjunctive therapy for older children and teenagers with OSAS, especially considering the fact that adenotonsillectomy is less likely to be successful in this group. CPAP could be used simultaneously until OSAS seems to be resolved. The need for proper screening, and the need for a team approach in the screening and treatment of OSA has been identified by pediatric dentists. If they knew the most common signs and symptoms of OSA, with proper training, they could have become a valuable asset to sleep medicine. Although OSA can be detrimental to children, it is treatable. Early recognition of this condition through screening is of utmost importance.

Conclusion

Pediatric Obstructive Sleep Apnea has commonly remained undetected and misdiagnosed both by parents and by medical professionals until now. There has been a necessity for sensitization of pediatricians, respiratory physicians and pediatric dentists regularly so as to help them, and to promptly diagnose and intervene the condition at an early stage. There has been a continuous progress in the field of pediatric sleep medicine that helps for having a better understanding of the Obstructive Sleep Apnea, especially in children.

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