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Pediatric obstructive sleep apnea: A Pedodontist's perspective on causes, consequences and care

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Abstract
Sleep is considered to be an important key factor for the healthy growth and development of a child. Sleep disorders usually impair a child's sleep and can lead to negative consequences on the general and dental health. In children, Obstructive sleep apnea syndrome is reported to be the most common sleep disordered breathing. The enlargement of the adenoids and tonsil tissues is considered to be the major cause of pediatric OSA. Currently, Pedodontists play a pivotal role in the early recognition, diagnosis and prompt treatment of OSA with the help of oral appliance thereby referring the high risk patients for sleep assessment. Inter-professional teamwork is very much essential to safeguard from long-term consequences. The primary health care provider, sleep physician, sleep study center, anesthesiologist, surgical team, pedodontist and caregivers effectively provide the successful outcome for patients with OSA. Pedodontists must identify the sleep issues and educate caregivers so as to communicate with the specialists for further screening and treatment of pediatric OSA. Thus, this review encompasses on etiology, clinical features, diagnosis, treatment, complications and the role of a Pedodontist in treating OSA.

Keywords: Children, obstructive sleep apnea, pedodontist, sleep disorder

Introduction

Sleep-disordered breathing (SDB) in children spans a broad range of conditions, from primary snoring (PS) as the mildest clinical manifestation to obstructive sleep apnea syndrome (OSAS) as it is most severe form. Early identification, diagnosis, and treatment are crucial due to their significant impact and potential serious consequences on general health [1].

OSAS was first described in 1965 by Bickelmann *et al.* as Obesity Hypoventilation syndrome (OHS) and currently OSA is the most prevalent and clinically significant SDB [2]. According to the American Academy of Sleep Medicine, OSAS is characterized by recurrent episodes of partial or complete airway obstruction during sleep, resulting in a reduction in (hypopnea) or in the total cessation (apnea) of airflow, despite ongoing respiratory efforts, leading to oxygen desaturation and arousal resulting in sleep fragmentation, intermittent hypoxia, and hypercapnia with increased sympathetic nervous system activity [2].

The lack of deep sleep leads to daytime napping, cognitive impairment, and low quality of life in association with various diseases such as obesity, myocardial infarction, global cardiovascular morbidity, atrial fibrillation, hypertension, diabetes mellitus and other endocrine disorders [3]. OSAS can have dramatic effects on childhood behaviour, neurodevelopment and overall health. Thus, early recognition, evaluation, and treatment are utmost important to prevent long-term consequences [4].

This review provides a concise overview of the current state of OSA, its importance in the medical and dental field, summarizing the most effective diagnostic and treatment tools and the role of a Pedodontist in the therapeutic approach in Paediatric OSAS.

Epidemiology

Epidemiologic studies report high prevalence of OSAS ranging from 0.8% to 24% but true prevalence ranges between 1% to 5% of all children [5]. Approximately 12-15% of children are affected by SDB, with highest prevalence in preschool children of 3 to 5 years. In 2% of children and 2.5% to 6% of adolescents, incidence peaks at 2 to 8 years reason being increased growth of tonsils and adenoids relative to the size of the upper airway in this age [6, 7, 8]. Unlikely, in adults, habitual snoring (snoring >3 nights/week) is common, affecting 3% to 12% of children [5, 9]. Equal prevalence in both sexes but predominant in males following puberty [9].

ANATOMIC FACTORS	NON ANATOMIC FACTORS	MALOCCLUSIONS
• Adenoid and tonsillar hypertrophy • Retrognathia • Micrognathia • Maxillary constriction • Short cranial base • High arched palate • Steep mandibular plane angle • Decreased cranial base angle • Mid face deficiency • Dolicofacial pattern • Infraposition of hyoid bone • Narrow nasal cavities	• Obesity (BMI≥30Kg/m^2) • Male • Heredity • Supine sleeping position • Behavioral aspects (smoking, alcohol, hypnotics, sedative medications)	• A narrow maxillary dentition • Ashort lower dental arch • Mandibular crowding • Anterior openbite • Lateral crossbite • Increased overjet

Fig 1: Risk Factors [1, 10, 11, 12, 13]

Additional medical disorders (Based on various observational studies) [8, 14, 15, 16, 17, 18, 19, 20, 21, 22]

Endocrine disorders (Diabetes mellitus, metabolic syndrome, acromegaly, hypothyroidism)

Neurological disorders (Stroke, spinal cord injury, myasthenia gravis)

Frader willi syndrome

Congestive heart failure

Downs syndrome

Atrial fibrillation

Obesity hypoventilation syndrome

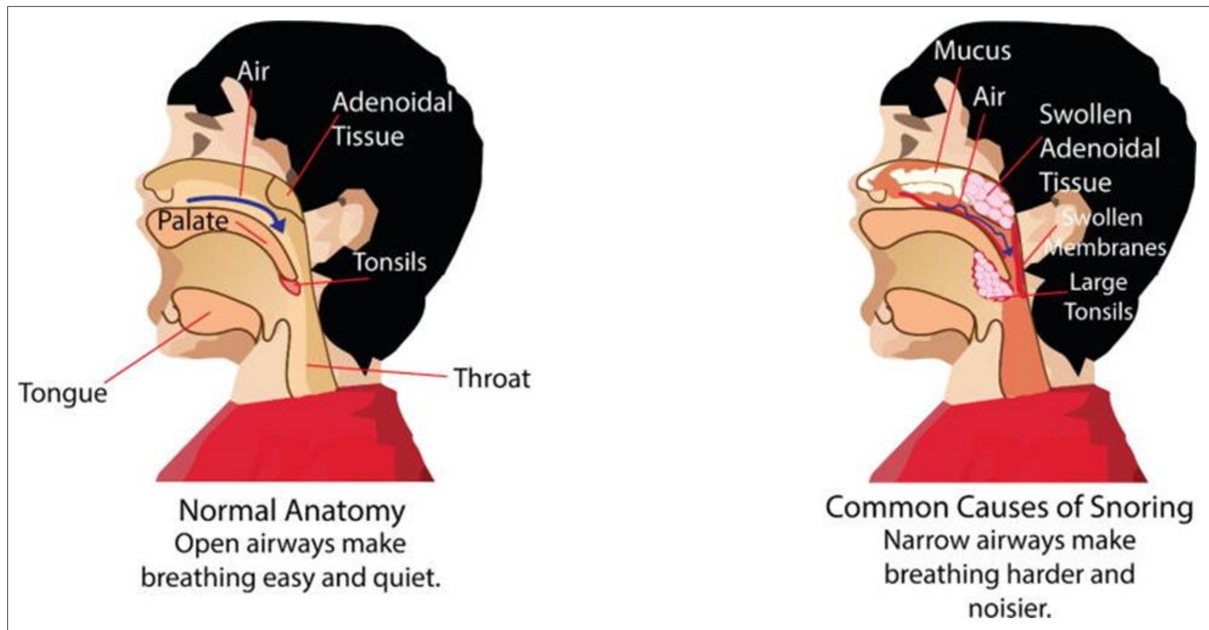

Fig 2: Normal Anatomy and Common causes of snoring [14]

Pathophysiology [23]

The Conceptualization of Pressure-Flow Behaviour Through the Collapsible Tubes

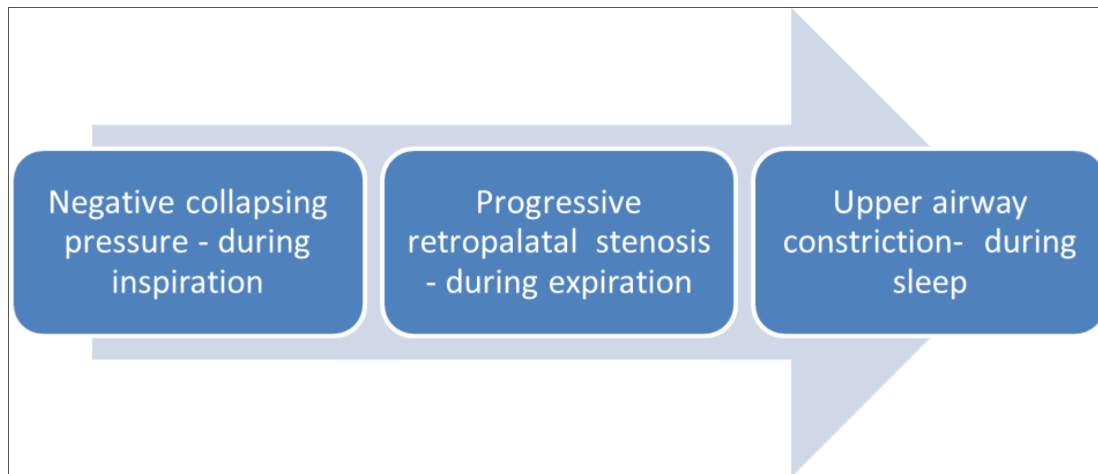

Fig 3: Anatomical and neuromusculoskeletal factors contribute to upper airway obstruction and the intensity of airway occlusion during sleep is most often related to body mass index (BMI).

Table 1: Clinical Features [14, 24]

Infants	Toddlers	Pre-school	School going
Disturbed nocturnal sleep Repetitive crying Snoring Nocturnal sweating Poor sucking reflex Apparent life-threatening event Delayed development Failure to thrive	Snoring Restless nocturnal sleep Abnormal sleeping position Nocturnal sweating Mouth breathing Night terrors Poor eating Failure to thrive Poor growth	Regular heavy snoring Mouth breathing Restless nocturnal sleep Sleep walking Night terrors Enuresis ADHD like syndrome Increased need for napping Poor eating Growth problems	Regular heavy snoring Restless nocturnal sleep Sleep walking Sleep talking Excessive bruxism Difficulty walking up in the morning Morning headaches Poor appetite Excessive daytime sleep Aggressiveness Emotional instability Learning difficulties

Dentoskeletal Changes [14]: Distinctive dentoskeletal characteristics of hard and soft tissues reported by various authors:

Tangugsorn *et al.* (1995): Reduced airway space, inferior positioned hyoid bone, soft palate (thick and long), and tongue (large and long).

Nuckton *et al.* (2006): Adenotonsillar hypertrophy followed by Mallampati deviation classes III and IV.

Marino *et al.* (2009): Elongated soft palate.

Pirila-Parkkinen *et al.* (2009): Predominantly class II malocclusion in children.

Cohen-Levy *et al.* (2009): Mandibular retrognathism, mid-face hypoplasia or both, which ultimately force the tongue to fall back into the upper airway.

Pirila-Parkkinen *et al.* (2009): Mandibular crowding is directly proportional to the apnea–hypopnea index (AHI).

Iwasaki *et al.* (2009): Class III malocclusion show more intraoral airway space and larger oropharyngeal airway compared to those with class I malocclusion, making them less prone to OSA.

More *et al.* (2011): Increased overjet, decreased overbite, unilateral or bilateral open bite/cross bite and mandibular crowding.

Marcus *et al.* (2012) [5]: High and narrow arched palates and long face profiles.

Deng and Gao (2012): Retrognathic mandible, a long lower face and a deficient chin – important cause of childhood OSA.

Katyal *et al.* (2013): Hypertrophy of both palatal and lingual tonsils.

Diagnosis [14]

A thorough medical and sleep history along with proper examination of oral cavity is imperative for accurate and timely assessment of Obstructive sleep apnea with appropriate treatment and prevention of further complications.

Dentist should examine the following:

Rosen (1999): Polysomnography (PSG) is considered as a gold standard and nocturnal oximetry is indeed another tool to assess OSA patients.

Friedman *et al.* (1999): Examining the etiologic factors associated with OSA, namely mostly tonsilloadenoid hypertrophy succeeded by a Mallampati score.

Li *et al.* (2002): Tonsillar size and OSA have positive correlation i.e, Greater the tonsillar size, worse the intensity of OSA.

Marcus *et al*. (2012) [5]: Clinical features such as changes in sleep posture and snoring.

Marcus *et al*. (2012) [5]: Children presenting with a high degree of suspicion for OSA, (i.e, a history of regular snoring, and higher grade of tonsils and/or a high Mallampati score) should be referred for a PSG to confirm diagnosis.

Kumar *et al*. (2014): one-unit increment in the Mallampati index will enhance the likelihood of developing Obstructive sleep apnea by 6-fold.

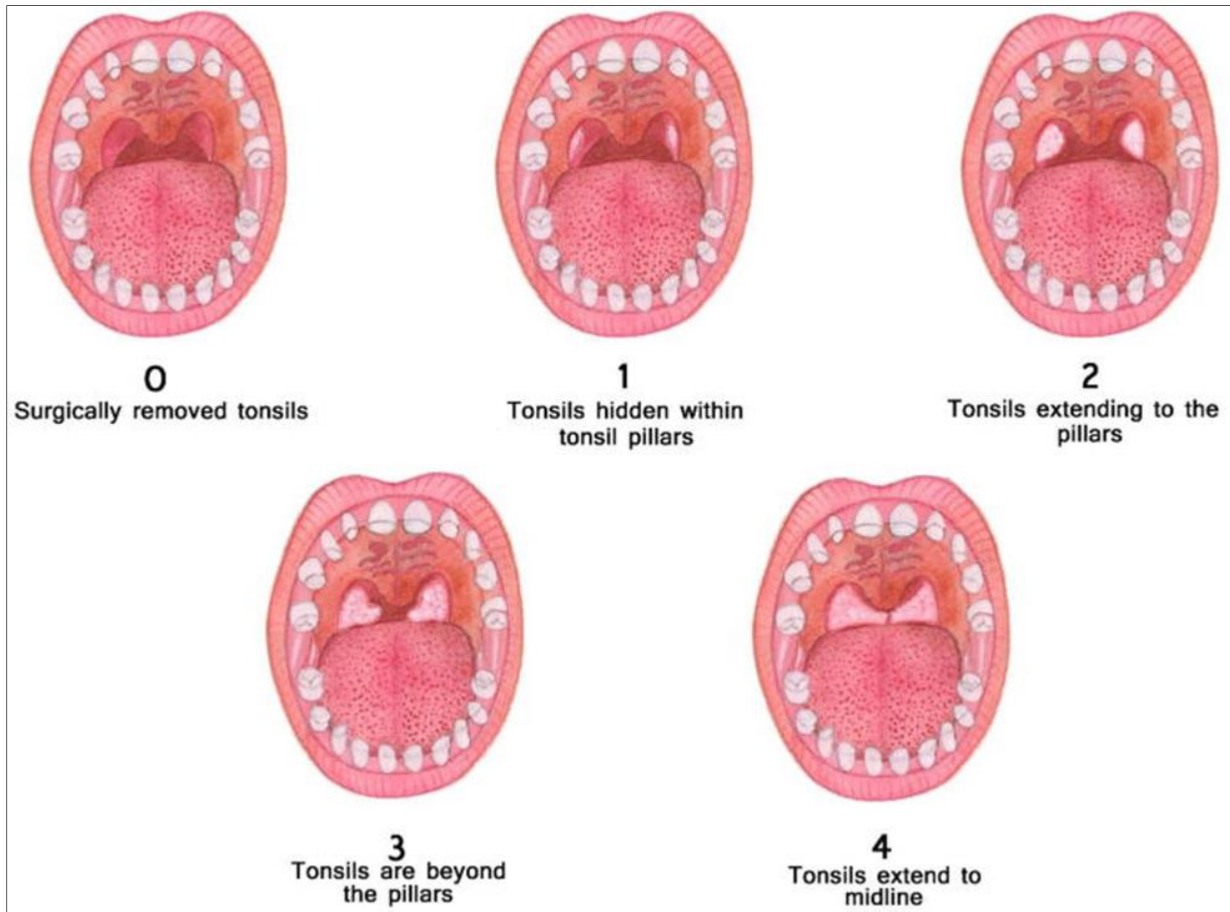

Fig 4: Friedman classification of tonsils [14]

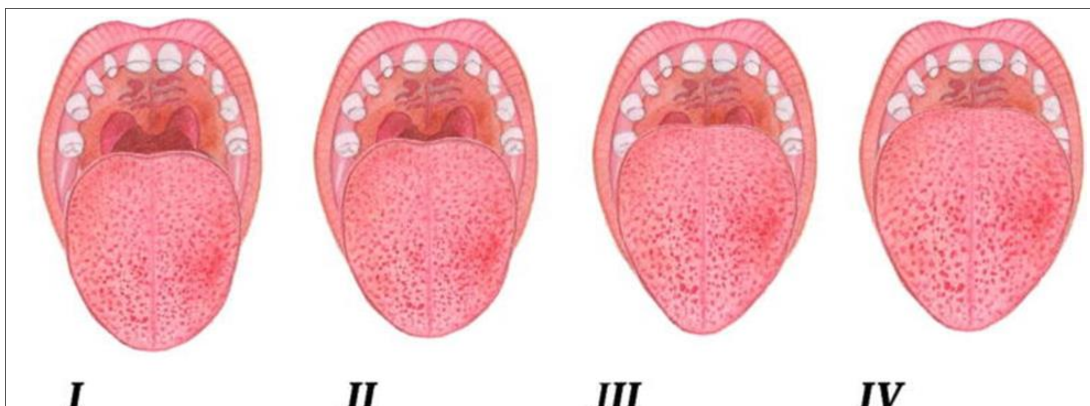

Fig 5: The Mallampati classification [14]

Detailed history and a validated survey, both to be considered before advising a PSG for the conclusive diagnosis of OSA.

Ahmed *et al*. (2018): Newly designed questionnaire (IMP-Q) was compiled and assembled from other widely used five questionnaires. Of these questionnaires, two of which focused on the quality of life (1) PedsQL – Parent Report (2) PedsQL – Child Report and three others focused on sleepiness and its effects (1) OSA-18 (2) Modified Epworth Sleepiness Scale and (3) Pediatric Sleep Questionnaire.

IMP-Q Questionnaire derived from (1) PedsQL- Parent Report (2) PedsQL- Child Report, (3) OSA-18 (4) Modified Epworth Sleepiness Scale (5) Pediatric Sleep Questionnaire

Table 2

Dataset	Question
CHQ	Do you have trouble sleeping?
CHQ	Can other kids do things you cannot?
SLS	Chance of Dozing or Falling Asleep: Sitting and reading
PSL	*Always snores?
PSL	*Have trouble breathing, or struggle to breath?
PSL	Have you ever seen your child stop breathing during the night?
PSL	*Tend to breathe through the mouth during the day?
PSL	Does your child complain of headache in the morning?
PSL	*Is your child overweight?
OSA-18	**Breath holding spells or pauses in breathing at night?
OSA-18	*Choking or gasping sounds while asleep?
OSA-18	Mouth breathing because of nasal obstruction?

Oxygen Saturation Cut off values
Oxygen Saturation < 85% - Adults
Oxygen Saturation < 92% - children
The severity of OSA in children can be determined by the AHI index (Haviv *et al*., 2014, Stauffer *et al*., 2018, Behrents *et al*., 2019) [9, 11].

AHI 1.5 to 5 – mild OSA
AHI > 5 and <10 - moderate OSA
AHI >10 - severe OSA

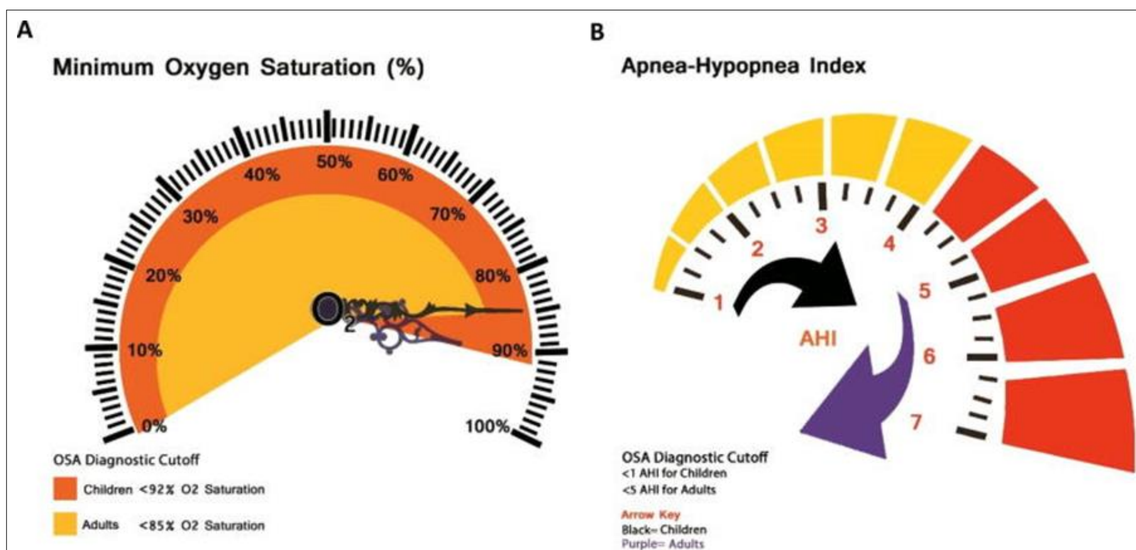

Fig 6: A. Oxygen Saturation Cut off values for Adults and Children
B. AHI Cut Off Values for Adults and Children[14]

Differential Diagnosis [8]

Allergic rhinitis: Snoring associated with nasal congestion could be due to allergic rhinitis.

Attention deficit disorder: Hyperactivity, distractibility and inattention in children could be misdiagnosed to behaviour changes seen in OSA.

Developmental milestones delay: OSA may lead to learning disabilities due to compromised attention and focus which may be sometimes misguided as a delay in developmental milestones.

Gastroesophageal reflux: OSA and gastro esophageal reflux are mutually exclusive. Occurrence of reflux at night results in brief cessation in breathing which may be mistaken as OSA.

Morning headaches: Overnight retention of carbon dioxide can also lead to early morning headaches. But a typical headache from OSA is dull and generally resolves on its own shortly after waking up without using any caffeine or medications. So, the patient should be screened for the qualities of headaches (worsens when lying supine, awakening at night, etc).

Narcolepsy: Hypersomnia in younger children is not a feature of OSA. Patients frequently falling asleep during the day time after age five should be screened for narcolepsy.
Polysomnography is the only gold standard diagnostic tool to distinguish between OSA and any of these.

Management [14]

The American Association of Orthodontists strictly recommends that a definitive diagnosis should be made by the appropriate physician and the treating orthodontist should be well versed with the signs and symptoms of OSA and its risk factors.
Management can be broadly categorized into:
1) Non-Dental Treatments
2) Dental Treatments

1. Non-Dental Treatments

Table 3

Therapy	Population	Benefits	Risk/challenges
Surgical [9, 25]			
Adenotonsillectomy (AT)	Children with enlarged tonsils and/or adenoids	Highly effective: well-tolerated in most children	Common: pain, decreased oral tolerance, rarely haemorrhage, 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 growth 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 the 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 follows up data; success varies by center/type of surgery; significant morbidity.
Non-Surgical [9,25]			
Positive airway pressure (PAP)	Any child	Strong evidence for efficacy, even if OSAS 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
Supplemental oxygen	Unclear; possible infants or those with no other therapeutic options	Hypoxemia may be prevented	Does 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

2. Dental Treatments

Dental treatments typically involve interventions aimed at modifying growth in the oromandibular region, such as rapid maxillary expansion and mandibular growth activators, along with mandibular advancement appliances and tongue retraining devices (Ngiam, 2015).

Rapid Maxillary Expansion (RME) [14]

In pediatric patients with obstructive sleep apnea, there is often observed a constricted maxilla, a high-arched palate, midface hypoplasia in the transverse dimension, and maxillary crowding.

Baratieri *et al*. (2011) and Ribeiro *et al*. (2012) illustrated that rapid maxillary expansion treatment led to an enlargement of the transverse dimension of the nasal airway.

Iwasaki *et al*. (2013) highlighted the comprehensive outcomes of RME, which encompassed widening of the pharyngeal space, reduction of nasal obstruction, and elevation of tongue posture. In a classification system proposed by Angelieri *et al*. (2013), RME stages are categorized from A to E, with stage A indicating no fusion of the mid palatine suture and stage E representing complete fusion. This classification system aims to guide clinicians in avoiding unnecessary surgical interventions and enhancing the success rates of RME treatments.

According to Villa *et al*. 2015, early initiation of treatment is crucial for maximizing its benefits. Vale *et al*. 2017, in their meta-analysis, found a significant decrease in the Apnea-Hypopnea Index (AHI) following rapid maxillary expansion treatment in children. They suggested that RME could serve as a viable alternative treatment for pediatric sleep apnea syndrome.

Mandibular advancement [14]

Lowe *et al*. (1995) indicated a correlation between a skeletal class II relationship and mandibular retrognathism with sleep apnea syndrome.

Cozza *et al*. (2004) reported a notable decrease in the AHI among children diagnosed with sleep apnea syndrome attributable to class II malocclusion following treatment with a customized monobloc device.

Hanggi *et al*. (2008) in a cohort study spanning 22 years, observed increased pharyngeal airway dimensions in children treated with activator-headgear.

Xiang *et al*. (2017) a meta-analysis was conducted to evaluate the effectiveness of functional appliances on upper airway dimensions in growing children diagnosed with class II malocclusion and retruded mandibles. The study concluded that functional appliances have the capability to enhance airway dimensions, thus potentially reducing the risk of obstructive sleep apnea (OSA) associated with mandibular retrusion. This guided therapeutic approach using functional appliances is beneficial in mitigating future OSA risks in growing children, as well as offering treatment possibilities for established OSA patients.

According to Stauffer *et al*. (2018) [9] myofunctional therapy involves educating patients on correcting tongue posture and orofacial muscle function.

Lin *et al*. (2020) conducted a systematic review and network meta-analysis, which determined that surgery yielded the best results in terms of AHI outcomes. Additionally, rapid maxillary expansion (RME) was found effective in achieving the highest arterial oxygen saturation (SaO_2). However, none of these interventions have been definitively established as sole treatments capable of completely resolving pediatric sleep apnea syndrome.

Oral Appliances [8]

For children diagnosed with mild to moderate obstructive sleep apnea syndrome (OSA) who have suitable dental conditions and cannot use CPAP therapy, custom-fitted and titrated oral appliances or mandibular advancement devices (MAD) are recommended. These devices advance the lower jaw to alleviate airway obstruction. Recent research, including a long-term randomized clinical trial with a 10-year follow-up, has shown comparable improvement in self-reported neurobehavioral outcomes between CPAP and MAD therapies.

Guidelines developed jointly by the American Academy of Dental Sleep Medicine (AADSM) and the American Academy of Sleep Medicine (AASM) outline the appropriate use of MAD for OSA:

- Oral appliances may be considered for patients with snoring (without OSA) or those with OSA who are unable to tolerate CPAP or prefer alternative treatments.
- A qualified dentist should provide a custom fitted, titratable oral appliance for patients with sleep apnea syndrome.
- Regular follow-up with a dentist is necessary after initiating intraoral appliance therapy in patients with OSA to monitor for any dental-related side effects.
- Periodic sleep testing follow-up is recommended to confirm the efficacy of the treatment.

Role of Pedodontist

Pedodontists leverage their clinical expertise to assess and predict the likelihood of obstructive sleep apnea (OSA) in patients. They consider various craniodental indicators such as large tonsils, high Mallampati score, crossbite, overjet, elongated soft palate, high-arched palate, short lingual frenulum, retruded mandible and dental crowding. A comprehensive sleep history is also crucial in their evaluation process. Based on these assessments, pedodontists can accurately diagnose OSA or determine the risk of its development. In suspected cases of OSA, polysomnography (PSG) may be recommended for definitive diagnosis and further management. Pedodontists also play a significant role in managing maxillary and mandibular growth in specific age groups. This growth changes can effectively treat OSA and potentially obviate the requirement of surgical interventions or continuous positive airway pressure (CPAP) therapy [14].

The AAPD emphasizes the importance of recognizing potential consequences of untreated obstructive sleep apnea (OSA) and encourages healthcare professionals to

- Screen patients for sleep-related breathing disorders, including OSA and primary snoring.
- Evaluate the tonsillar pillar area for signs of hypertrophy.
- Assess tongue positioning, as it can contribute to airway obstruction.
- Acknowledge the role of obesity in contributing to OSA.
- Recognize that craniofacial anomalies may be linked to OSA.
- Refer patients suspected of having OSA to an appropriate medical specialist (e.g., otolaryngologist, sleep medicine physician, pulmonologist) for diagnosis and treatment.
- Consider the use of intraoral appliances after conducting a comprehensive orthodontic and craniofacial assessment of the patient's growth and development, within a multidisciplinary approach [26].

Complications of Untreated Osa

Untreated OSA in children, coupled with insulin resistance and obesity, not only exacerbates previously mentioned comorbidities such as cardiovascular issues, impaired growth, learning difficulties, and behavioral problems, but also predisposes them to developing heart disease and endocrinopathies.

Pediatric dentists who administer sedation or perform surgical procedures on children with OSA should be aware of the possible risk of perioperative and postoperative respiratory complications. Conducting a thorough airway assessment in collaboration with caregivers,

especially before procedures involving sedation or general anesthesia, can help identify patients at increased risk of OSA or complications during and after surgery. Referral to a medical specialist for further assessment, diagnosis, and management may be beneficial for these individuals [27].

Conclusion

Pediatric OSA is a significant medical concern that has evolved over time and can profoundly impact a patient's health and quality of life in the long term. It is crucial for pediatric dentists to be knowledgeable about the signs and symptoms of OSA. They should conduct a comprehensive assessment including a detailed history, intraoral and extraoral examination, use of relevant questionnaires, and consideration of comorbid conditions. Timely diagnosis and effective management of OSA are essential to mitigate potential long-term health consequences. Adopting an interdisciplinary treatment approach often benefits the patient's overall well-being. Therefore, pediatric dentists play a critical role in early detection and implementing interventions to modify growth patterns, which can facilitate the future management of OSA.

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