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Review Article

Pediatric Obstructive Sleep Apnoea

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Introduction

Paediatric obstructive sleep apnoea (OSA) is a condition in which there is complete or partial airway obstruction during sleep,leading to decreased oxygen saturation or arousal from sleep. It can have marked effects on the behaviour of the child, neurodevelopment, metabolism, and overall health (1) . When Paediatric OSA is not treated it is associated with changes in behavioural pattern and learning disabilities; in severe cases, it can be associated with impaired growth,including failure to thrive and cardiovascular complications. The prevalence of morbidity of paediatric OSA varies but is between 1%−5%, with the peak prevalence occurring at 2−8 years of age (2,3,4). Early diagnosis and treatment can decrease the chances of morbidity (5). Guilleminault et al. first described the precise characteristics of OSA in 1976, noting that upper airway obstruction during sleep could be intermittent, partial, or complete. Osler and Hill had first reported it 120 years ago in these terms: "The stupid and lazy child who suffers from headaches in school breathes through his mouth rather than his nose, snores during his agitated sleep, and awakens with his mouth dry, deserves better treatment by the school nurse" (6).

There is no gender predilection reported for the prevalence of OSA in children except among adolescent boys. Higher levels of weight gain may be responsible for the increased prevalence of OSA in adolescent boys (7,8).

Redline et al., suggested that an increase in body mass index (BMI) 1 kg/m2 above the mean increases the risk of developing OSA in children by 12%. Additionally, these children have larger neck circumferences than healthy children (9). Children who continued breastfeeding for more than a month showed a lower risk of witnessed sleep apnoea compared to children who never breastfed or breastfed for less than a month (10).

Etiology

Sleep apnoea is divided into two categories based on the causes: central or obstructive. Central sleep apnoea is due to central nervous system pathology. Obstructive sleep apnoea, 95% of diagnosed sleep apnoea, is due to complete collapse of the upper airway or partial collapse, resulting in arousal from sleep or 3% or more oxygen desaturation (11,12).

Anything that can decrease airway diameter or integrity can contribute to OSA, including anatomic, genetic, or neuromuscular issues (11). Upper airway can have an increased risk of abnormal collapse due to both intrinsic and extrinsic factors. The intrinsic factors are based on the critical pressure in the airway that is needed to maintain patency. The extrinsic factors are fat deposits, hypertrophy of tissues, and craniofacial features that stray from normal anatomy that contribute to increased incidence of collapse. (1)

Risk factors for early-onset OSA include prematurity, African, American race, and day-care attendance. The severity of OSA is increased in those with obesity, tobacco exposure, patients with craniofacial abnormalities, including those with Crouzon, Pierre-Robin or Apert syndromes, as well as those with cleft lip or palate. Boys are at an increased risk after puberty, but the prepubertal risk is equal among boys and girls. The presence of alteration of the normal airway anatomy as well as features such as micrognathia, micro or macroglossia, and midface hypoplasia all contribute to decreased posterior oropharynx space and the increased incidence of paediatric OSA (13,14). Many genetic disorders and syndromes can also cause OSA, such as Trisomy 21, Achondroplasia, Pierre Robin Anomaly, Apert syndrome, Crouzon syndrome, Treacher Collins syndrome, Turner syndrome, cleft palate, and cerebral palsy (15). Children with autism spectrum disorder (ASD) exhibit significantly more sleep disorders, including OSA and parasomnias, than preschool children of the same age (16) . If the child's snoring is not related to hypoxia or apnoea, it does not require any further intervention or treatment (17,18,19).

Epidemiology

Paediatric obstructive sleep apnoea (OSA) is a highly prevalent but often neglected disorder due to ignorance among general physicians and paediatricians. The prevalence in children is estimated in the range of 2%–4% in Western countries but data from India is lacking (20). The prevalence is increasing and is probably under represented given the paediatric obesity epidemic.

A study by Goyal et al in 2018 showed a high prevalence (9.6%) of OSA among Indian children in the age group of 5–10 years (21).

Clinical Features

History

Sleep quantity and quality of the paediatric patient must be evaluated and questions about frequent night awakenings, unusual sleep positioning, and significant disruption of bed coverings as signs of increased night-time movements to evaluate sleep quality (14).

Parents often give a history of snoring, laboured breathing, paradoxical breathing, mouth breathing, witnessed apnoea's, frequent night-time awakenings, secondary nocturnal enuresis, nocturnal sweating, fragmented, agitated sleep, drooling while sleeping, sleep terrors, and sleepwalking (8, 22).

In addition to snoring, children with OSA have hypoxemia and experience hypoventilation, sleep disruption, and poor gas exchange (2) . It is important to interview parents/caregivers systemically to obtain the best clinical picture given that the children themselves can only recognize and report the complaints and symptoms at an older age (22,23).

Physical examination

A thorough physical examination of a child suspected of having OSA must include an evaluation of the child's general appearance, with careful attention to craniofacial characteristics such as midface hypoplasia, micrognathia, and occlusal relationships. Septal deviation, choanal atresia, nasolacrimal cysts, and nasal aperture stenosis must be considered in infants. In older children, nasal polyps and turbinate hypertrophy must be ruled out.

On physical exam, patients may appear tired or may exhibit a hyperactive state. An examination may reveal "allergic shiners," swollen nasal mucosa, micrognathia, macroglossia, high-arched palate, adenoidal facies, or hypertrophied tonsils. Additionally, hypo nasal speech and nasal congestion can often be appreciated (22).

During an intra-oral examination, evaluate the geometry of the soft palate for size, redundancy, and clefting; document the size of the tongue and tonsils; and perform lateral neck radiography or a nasopharyngoscopic examination to evaluate the size of the adenoidal tissue and the site of airway collapse. Detection of tonsillar hypertrophy on routine examination should prompt physicians to question parents about snoring and other symptoms of OSA in their children (24).

The most common cause of OSA in children is hypertrophy of the tonsils, both palatal and lingual (25,26,27,28). Adenotonsillar hypertrophy is followed by Mallampati deviation classes III and IV. The Mallampatti score is a score based on anatomical structures that are visualized by opening the mouth and protruding the tongue, and it is widely used as a predictor of OSA. The higher the Mallampati score, the greater the severity of OSA, provided the Mallampati score is measured accurately (29). Some children with OSA also exhibit an elongated soft palate (30,31,32), a reduced airway space, a longer and thicker, soft palate, a long and large tongue, and an inferior positioned hyoid bone (31, 33, 34).

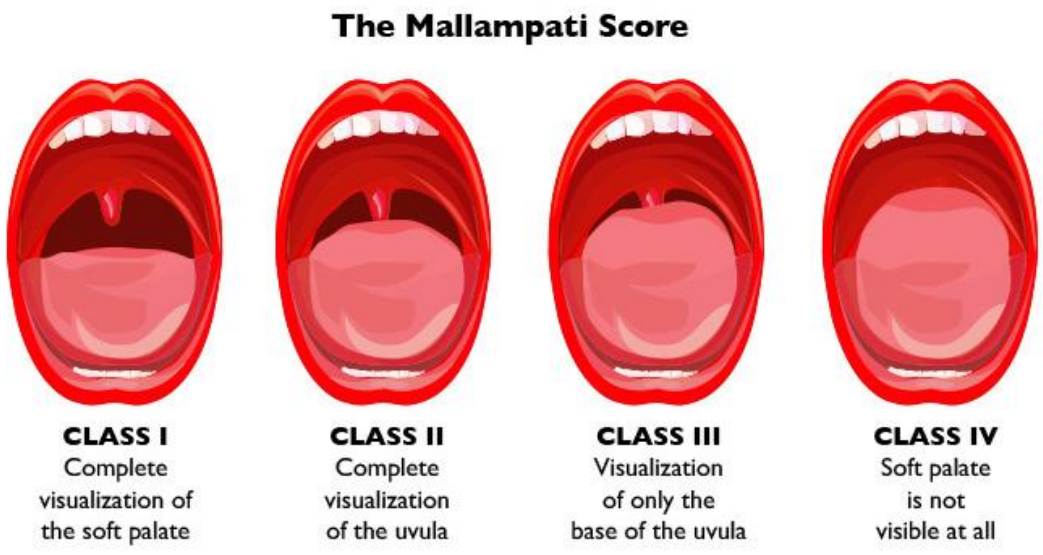

Figure 1: The Mallampati Score

Children diagnosed with OSA predominantly showcase a class II malocclusion pattern (25, 32, 35,36) with high arched and narrow palates, long face profiles, greater overjet, less overbite, unilateral or bilateral open bite/crossbite, and mandibular crowding (26,37,38,39,40) and demonstrate retrognathism of the mandible, mid-face hypoplasia, or both, which ultimately force the tongue to fall back into the upper airway. Deng et al. reported retrognathism of the mandible, a long lower face, and a deficient/short chin as a few important causes of childhood OSA (41). Mandibular crowding is directly proportional to the apnoea–hypopnea index (AHI) (25). Interestingly, patients with class III malocclusion show more intraoral airway space and larger oropharyngeal airway compared to those with class I malocclusion, thus making them less prone to OSA (42).

Diagnosis Of OSA

The American Academy of Paediatrics (AAP) recommends screening for OSAS at routine medical visits. The diagnosis should be considered in children with typical symptoms (e.g. snoring, restless sleep, or daytime hyperactivity) or risk factors (e.g. craniofacial, neurologic, or genetic disorders) and is confirmed with overnight polysomnography. A detailed medical and sleep history as well as an oral cavity examination is needed when the patient presents in the dental clinic. Specific information about signs and symptoms must be elicited from the parents, such as snoring and frequent changes in sleep posture (40).

The gold standard for the diagnosis of OSA, as recommended by the AAP, is a nocturnal, in-lab polysomnography (PSG) study (43,44). There is no universally accepted classification for OSAS severity in children, although many studies use the Apnea Hypopnea Index (AHI) to categorize OSAS as mild (AHI 1–4.9), moderate (AHI 5–9.9), or severe (AHI > 10). This approach provides an objective, quantitative evaluation of disturbances in respiratory parameters and sleep patterns, thus allowing patients to be stratified into disease severities and thereby enabling clinicians to tailor clinical management accordingly. As of yet there is no international consensus regarding the AHI cut off values for therapy initiation. The current accepted practice has consisted of the use of an arbitrary cut off for AHI corresponding to >3 standard deviations beyond the mean of the normative AHI in healthy children.

Most clinicians would agree that a child with an AHI >5/hour TST requires treatment and that a child with an AHI <1/hour TST does not have significant OSA (43).

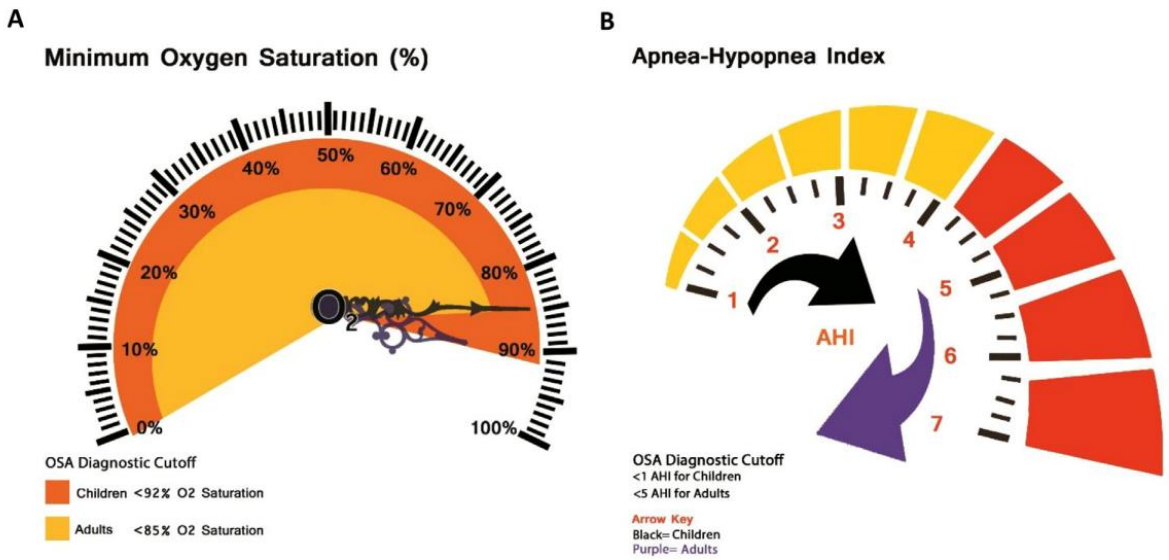

Figure 2: A. Oxygen Saturation Cutoff values for Adults and Children

B. AHI Cut Off Values for Adults and Children

Moin Anwer HM, Albagieh HN, Kalladka M, Chiang HK, Malik S, McLaren SW, Khan J. The role of the dentist in the diagnosis and management of pediatric obstructive sleep apnea. Saudi Dent J. 2021 Nov;33(7):424-433. Epub 2021 Feb 26.

Differential Diagnosis of OSA in Children

i. Adeno tonsillar hypertrophy

ii. Naso-septal obstruction

iii. Enlarged soft palate or uvula

iv. Macroglossia

v. Hypotonic pharynx

vi. Lingual tonsils

vii. Laryngeal abnormality (e.g., lymphatic malformation)

viii. Micrognathia

ix. Maxillary hypoplasia

Management of Pediatric OSA

Management of OSA can be divided into two broad categories:

i. Non-surgical treatments

ii. Surgical treatments

Non-surgical treatments

a) Continuous positive airway pressure (CPAP): children with small tonsils/adenoids and those who are not surgical candidates or whose parents decline surgery should be managed with PAP therapy during sleep (45).

b) Medical therapy: proposed as an alternative to surgery, especially in children with mild OSAS. A six-week course of once-daily intranasal budesonide has been shown to improve quality of life and respiratory parameters measured on polysomnography with a sustained effect two months after discontinuation (46, 47), and a recent meta-analysis found that montelukast improved the AHI by 55% when used alone and by 70% when used in conjunction with intranasal corticosteroids in children with mild OSAS (48).

c) Dental treatment: Various dental treatments primarily including growth modifiers of the oro-mandibular region (rapid maxillary expansion [RME] and mandibular growth activators), mandibular advancement appliances, and tongue retaining devices move the tongue and mandible forward and

away from the posterior pharynx to improve upper airway patency (49).

Surgical treatments

a) Adenotonsillectomy: Adenotonsillectomy refers to surgical resection of the tonsils and adenoids, it is generally indicated for otherwise healthy children who have adeno tonsillar hypertrophy and severe OSA (eg, apnea-hypopnea index [AHI] >10, in the setting of relevant clinical symptoms). In obese children, OSA usually improves following adenotonsillectomy, although the outcome may be less satisfactory than in lean children. In 2013, a randomized childhood adenotonsillectomy trial demonstrated an advantage of adenotonsillectomy over watchful waiting for 5-9 year-old children with uncomplicated OSA (AHI 1 to 30) and showed improvements in daytime behaviour, sleep apnoea symptoms, subjective sleepiness, and quality of life (50,51,52) .

b) Tonsillectomy: The first reported tonsil surgery is documented in 700 BC (53,54) in an Indian Sanskrit document Atharva-Veda and described again in 50 AD by Celsius (55,56); however, it was not practiced due to the risk of haemorrhage (54,56). In Total tonsillectomy (TT) the entire palatine tonsil and capsule are removed and in Intracapsular Tonsillectomy (IT) the capsule or portion of the tonsil is left in situ(53,57,58, 59, 60,61,62). Complication rates of partial tonsillectomy (IT) are lower compared to standard tonsillectomy (63,64). Complications following tonsillectomy in children should be monitored and they could range in severity from mild oxygen desaturation to postoperative pulmonary oedema, throat pain, bleeding, nausea/vomiting, dehydration, delayed feeding, velopharyngeal insufficiency, and rarely death (65). Most tonsillectomies are performed as outpatient procedures although children with age < 3 or severe OSAS (AHI > 10 or oxygen saturation < 80%) are at increased risk for complications and should be observed overnight (65).

Types of tonsillectomy procedures:

a) Cold methods

- Cold Steel Tonsillectomy: Tonsillectomy is completed with reusable, re-sterilizable metal surgical instruments.

- Guillotine tonsillectomy: described in 1827 to remove the portion of the tonsils (IT only) by tonsillotome (54,56) .

- Micro debrider: powered soft tissue shavers, were introduced in 2002 (54). It uses a single-use disposable handpiece and is used for IT techniques removing 90–95% of the tonsillar tissue leaving a physiologic "bandage" decreasing post-operative haemorrhage and pain (56, 66).

- Plasma Blade: A newer single-use device that uses radiofrequency to cut and coagulate tissue. It creates a highly ionized plasma field around an electrode using the surrounding tissue electrolytes causing less thermal damage, tissue damage, less post-operative pain, and faster wound healing (61,67).

b) Hot Methods

- **Electrocautery:** It delivers radiofrequency energy via an instrument providing kinetic energy that heats the intracellular and extracellular fluids and ruptures localized tissue (54,57). It generates heat that may reach 300–440C and may cause more thermal damage to the surrounding tissue that may contribute to postoperative pain (54).

- Coblation or radiofrequency-controlled ablation uses an electrically powered single-use handpiece with saline irrigation to create an ionized plasma field that has energetic charge-carrying ions with sufficient energy to break organic molecular bonds resulting in the breakdown or "ablation" of the tissue (68,69,70). The heat generated in this method is less than in electrocautery at 40–70C (57).

- **Laser:** It was introduced as a concept for a bloodless tonsillectomy in 1994. Both CO2 and KTP lasers have been used in either TT or IT.

c) Lingual tonsillectomy: Lingual tonsillar hypertrophy and hypopharyngeal obstruction is the cause of persistent airway obstruction in children post tonsillectomy (72). In children with lingual tonsil hypertrophy, the removal of most of the hypertrophied tissue can alleviate the point of obstruction (73). In 2017, a systematic review and meta-analysis of children with persistent OSA had a reduction in the AHI and improvements in their oxygen saturations following lingual tonsillectomy (72).

d) Targeted Nasal Surgery: In patients with persistent OSA, particularly for patients with nasal obstruction, nasal allergies, or those who cannot tolerate CPAP, nasal surgery may be beneficial. This typically includes revision adenoidectomy, inferior turbinate reduction, septoplasty, or some combination of these procedures aiming at the reduction of obstruction to facilitate both medical management of any underlying allergies and improve nasal airflow (73).

e) Supraglottoplasty and Epiglottopexy: Collapse of the supraglottis on inspiration is seen in laryngomalacia. Congenital laryngomalacia is the most common cause of neonatal stridor and may present with failure to thrive and respiratory distress with feeding or sleep (73,74). The anatomic area of obstruction can be addressed with surgery. Epiglottopexy is done by denuding the mucosa of the base of the tongue with a partial lingual tonsillectomy and denuding the lingual surface of the epiglottis to promote scarring and to move the epiglottis anteriorly. Zalzal et al. showed lower AHI post-operatively in patients with PSG-proven OSA and supraglottic collapse following epiglottopexy (74).

c) Uvulopalatopharyngoplasty (UPPP) Uvulopalatopharyngoplasty was first described in 1991 (75). This procedure is reserved for children with neurologic impairment and moderate to severe OSA on PSG (73). This is the extensive restructuring of the soft palate and pharyngeal walls. In this procedure, the palatine tonsils are removed entirely (TT) if still present, with portions of the anterior pillar musculature (palatoglossus) removed. The uvula is resected, and a portion of the soft palate mucosa and muscle (levator palatini) are resected. The soft palate is reapproximated and the tonsillar pillars are sutured close. This results in the posterior tonsil pillar (palatopharyngeus muscle) being pulled anteriorly and superiorly (75). Post-operative complications include edema, airway obstruction, velopharyngeal insufficiency, and nasopharyngeal stenosis (75).

d) Mandibular advancement or mandibular distraction osteogenesis: Alleviates upper airway obstruction and avoid tracheostomy dependence. When airway obstruction is severe due to craniofacial disorders restricting the upper airway, more extensive surgical approaches are used (76,77,78,79).

e) Other Surgical Options: Tongue-base reduction and tongue-base suspension are rare procedures in children and are reserved for children with persistent OSA after adenotonsillectomy and other failed medical options. In addition to the peri-operative risks of bleeding and airway obstruction, neurovascular damage of the tongue is also a risk (75).

f) Hypoglossal nerve stimulation is done with an implantable device that stimulates the tongue in time to respiratory effort. This results in protrusion of the tongue with inspiration. This procedure is uncommon and effective in selected paediatric populations, typically in children with Trisomy 21 with macroglossia with persistent OSA post-adenotonsillectomy who cannot tolerate CPAP (75).

g) Tracheotomy: is the definitive surgical treatment for OSAS. Most patients who require tracheotomy have either severe craniofacial abnormalities or neuromuscular conditions causing hypotonia. Although many children remain tracheostomy-dependent, the procedure can also be used as a temporary measure to manage severe OSAS while awaiting other surgical treatment (80).

Conclusion

The role of an Oral and Maxillofacial Surgeon and paediatric dentist in the timely diagnosis and treatment of paediatric OSA has evolved over the years. As Paediatric OSA is a serious medical condition that can have long-term consequences on the overall health and quality of life of a patient, dental specialists should be familiar with the signs and symptoms of OSA and should conduct a thorough history, intra/extra oral examination, questionnaires and understand the role of comorbid conditions. A timely diagnosis and management of OSA can eradicate potential long-term negative effects on the health of a patient. If paediatric OSA is suspected a proper referral should be made for the definitive diagnosis and treated accordingly. An interdisciplinary treatment team consisting of a Paediatrician, Oral and maxillofacial surgeon, Pedodontist, and may often serve in the best interest of the patient.

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