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Review Article

Obstructive Sleep Apnoea in Children: A Review

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Abstract

Obstructive sleep apnea (OSA) is one of the most common causes of sleep-disordered breathing (SDB) in children. It is associated with significant morbidity, potentially impacting on long-term neurocognitive and behavioural development, as well as cardiovascular outcomes and metabolic homeostasis. The significant variance in degree of end organ morbidity in patients with the same severity of OSA highlights the importance of the interplay of genetic and environmental factors in determining the overall OSA phenotype. Dentists who practice sedation dentistry should exercise extra precautions when treating patients with risk of sleep apnea. This review seeks to summarize the current understanding of the aetiology and mechanisms underlying OSA, its risk factors, diagnosis and treatment.

Keywords: Paediatric Obstructive Sleep Apnea (Paediatric OSA); Sleep-disordered Breathing in Childhood (SDB in Childhood); Oral Appliances; Management; Role of Peadodontist

Abbreviations

OSA: Obstructive Sleep Apnea; SDB: Sleep Disordered Breathing; ATS: American Thoracic Society; PSG: Polysomnography; AHI: Apnea-Hypopnoea Index; ADHD: Attention Deficit/Hyperactivity Disorder; CPAP: Continuous Positive Airway Pressure; BiPAP: Bilevel Positive Airway Pressure

Introduction

Sleep is a necessary and vital biological function which is essential for a person's physical and emotional well being. Every child has a definite sleep pattern which is rarely disturbed except through sickness or experimental conditions. More than 10% of preschool aged children snore regularly. Obstructive sleep apnea syndrome is a pathology related to snoring with possible serious complications [1]. Sleep apnea, and particularly obstructive sleep apnea, is a common disorder that is characterized by repetitive partial or complete cessation of air flow, associated with oxyhemoglobin desaturation and increased effort to breathe. It takes its name from the Greek word "apnea", which means "without breath" Sleep apnea.

Obstructive Sleep Apnea (OSA): OSA is defined by the American Thoracic Society (ATS) as ''a disorder of breathing during sleep characterized by prolonged partial upper airway obstruction and/or intermittent complete obstruction (obstructive apnea) that disrupts normal ventilation during sleep and normal sleep patterns" [3]. Our understanding of pediatric obstructive sleep apnea syndrome and its significance on general health is in a rapid state of development. There is a great increase in research of this disorder and its complex etiology. Despite increasing recognition of childhood OSA as a significant public health problem, treatment of the condition remains inconsistent. Ongoing research of obstructive sleep apnea syndrome continues to demonstrate the significance of dentofacial support of the airway in relation to this disease. Dental practitioners especially pediatric dentist can play a significant role in the treatment of OSA in children. It is imperative that the dental community continues to participate in the research and treatment of this serious and pervasive health problem.

Epidemiology

A meta analysis of published studies found the prevalence of snoring to be 7.45%. The prevalence of SDB estimated from parental reports with additional diagnostic testing ranges from 0.1% to 13.0%, but most studies suggest a prevalence of 1% to 4% [4]. Based on a questionnaire survey the prevalence of pediatric OSA in India is estimated to be 9.6% in children between 5 and 10 years of age [5].

Etiology

Children with craniofacial syndromes have fixed anatomic variations that predispose them to airway obstruction, while in children with neuromuscular disease, obstruction is caused by hypotonia. Other risk factors include morbid obesity, prematurity, and the use of drugs that depress the reticular activating system, reduce the central ventilatory drive or directly depress upper airway muscle tone, such as sedatives, general anesthesia, and alcohol. Etiology of OSA in children may result from a complex interplay between Aden tonsillar hypertrophy and loss of neuromuscular tone [6]. Genetic and hormonal factors also found to have a role in the etiopathogenesis of the disease [7].

The adenoids, situated on the posterior wall of the nasopharynx, enlarge from infancy through adolescence in healthy children and then decrease again during adult life. The fastest period of adenoidal growth is in the first year of life [8]. Some of the systemic conditions with associated Obstructive Sleep Apnea include Down's syndrome, Cerebral palsy, Hypothyroidism, Osteopetrosis, Pierre Robin syndrome, Treacher Collins syndrome [9].

Clinical features

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 apneic episodes. These episodes are followed by gasping, choking, movement or arousal. Cyanosis is occasionally noted. Children may often sleep in positions promoting airway patency, such as seated or with their neck hyperextended. Children five years and older commonly exhibit enuresis, behavior problems, deficient attention span, and failure to thrive, apart from snoring [10].

In extreme cases of OSA in children, cor pulmonale and pulmonary hypertension may be the presenting problems. Poor growth and failure to thrive are more common in children with sleep-disordered breathing [11]. Decreased production of growth hormone during fragmented sleep may contribute further to poor growth. Increased urine production results from hormonal disregulation. These alterations are accompanied by increased levels of catecholamines and frequent arousal that further contribute to enuresis.

Diagnosis

Clinical practice guideline on the diagnosis and management of OSA in children released by the American Academy of Pediatrics has recommended that all children be screened for snoring as part of routine health care maintenance [12]. A thorough physical examination of a child suspected of having OSA must include evaluation of the child's general appearance, with careful attention to craniofacial characteristics such as midface hypoplasia, micrognathia, adenoid facies and occlusal relationships.

Evaluation for nasal obstruction depends on the child's age. Septal deviation, choanal atresia, naso-lacrimal cysts, and nasal aperture stenosis can be seen in infants. In older children, nasal polyps and turbinate hypertrophy has to be ruled out. The intra oral examination should include evaluation of soft palate, tongue and tonsils. Detection of tonsillar hypertrophy on routine examination should prompt physicians to question parents about snoring

and other symptoms of OSA in their children. The lungs are usually clear to auscultation. Cardiac examination may reveal signs of pulmonary hypertension. Muscle tone and developmental status should be assessed.The physical examination must include a neurologic survey for hypotonia and an assessment for obesity [11].

The gold standard test for the diagnosis of obstructive Sleep Disordered Breathing is an overnight, attended, in laboratory Polysomnography (PSG) study. PSGs are highly recommended because along with the diagnosis various respiratory parameters can be assessed appropriately. Thus, enables clinicians to plan for a clinical management accordingly [12]. The AHI is the most commonly used PSG parameter for the quantification of SDB severity. OSA is diagnosed by a patient's apnea-hypopnea index (AHI). The AHI represents the average number of apneas and hypopneas per hour of sleep. In pediatric OSA, which has the same prevalence in boys and girls, more than one obstructive apnea event of any length per hour of sleep is considered abnormal [11].

Polysomnography can exclude other causes of sleep-related symptoms, such as nocturnal seizures or narcolepsy. It provides objective measures of severity and provides a baseline for those children whose condition does not resolve postoperatively. Full PSGs are labour and resource intensive, requiring in hospital monitoring of the patient by skilled staff overnight, and subsequent scoring and analysis. When PSGs are not available, possible alternatives should be considered [13].

Other diagnostic aids

Ambulatory PSG is the term used for unattended sleep studies conducted in the home. The home test performed included measurement of oxygen saturations and ventilation, along with video recordings [7]. Paediatric sleep questionnaires are filled by parents to assess the symptoms of Sleep Disordered breathing, such as snoring, excessive daytime sleepiness, attention problems, and hyperactive behaviour in children aged 2 - 18 years. The sensitivity and specificity of questionnaires for diagnosing OSA in otherwise healthy children are 78% and 72%, respectively, but it can be used for predicting OSA-related neurobehavioral morbidity and the progress after adenotonsillectomy [14].

Daytime nap studies also have been used to screen patients for OSA. Videotapes, audiotapes, and continuous pulse oximeter have been helpful in predicting OSA when positive, but unfortunately do not rule out the diagnosis of OSAS when negative [15]. Airway radiographs, cephalometric studies, and CT scans may assist in the treatment plan for high-risk children with OSA caused by unusual anatomic abnormalities. Airway fluoroscopy provides information on the degree of obstruction and the dynamics of the child's airway. Sleep fluoroscopy has been shown to assist in the evaluation of children at risk for multilevel upper airway obstruction.

Lateral neck radiography provides useful information about the size of the adenoids and their relationship to the upper airway. Concurrent cervical computed tomography and magnetic resonance imaging also demonstrate pharyngeal lymphoid hyperplasia.

Consequences of untreated obstructive sleep apnea

It mainly affects the neurocognitive development, school performance, and behaviour if persistent for a prolonged period. Other consequences include cardiovascular morbidity, metabolic disorders and nocturnal enuresis. Children with OSA will have to avail healthcare services more frequently in comparison to their peers, with more hospital visits and more medication prescriptions, mainly for respiratory infections [16].

- **Growth**: Esteller., *et al*. reported that the impaired growth in children with even mild OSA appeared to be related to increased work of breathing during sleep [17]. Nocturnal growth hormone secretion also appears to be decreased in children with sleep-related upper airway obstruction [1].
- **Behavioral and learning problems:** Studies have shown behavioral problems and mild deficits in executive functioning, attention, and motor skills in a small group of children with mild to moderate OSA that improved modestly post treatment. Attention deficit/hyperactivity disorder (ADHD) or ADHD symptoms, hypersomnolence, somatization, depression, atypicality, aggression, and abnormal social behaviors were the other most frequently reported behavioral abnormalities associated with SDB in children [18]. Learning problems also have been found in children with OSA. Obese children with OSA have shown deficits in memory, vocabulary, and learning as assessed by standardized tests when compared with obese children without OSA [19].
- **Excessive daytime sleepiness:** Studies have shown a relationship between polysomnographic measures and objective measurement of daytime sleepiness on multiple sleep latency testing [20]. Sleep fragmentation in children will lead to behavioral and learning problems.

- **Cardiopulmonary effects:** A large number of studies found that OSA can affect both the right and left ventricles subsequently affecting the systolic and diastolic blood pressure. In addition, childhood OSA can affect autonomic regulation, brain oxygenation, and cerebral blood flow. Presence of nocturnal cardiac strain in children who have moderate to severe OSA have been reported [21]. Severe untreated OSA can lead to pulmonary hypertension and cor pulmonale. With increased awareness and early diagnosis of OSA, these serious complications are seen rarely.
- **Inflammation**: It has been postulated that OSA results in intermittent hypoxemia, leading to production of reactive oxygen species which may trigger a persistent low grade inflammation or exacerbate obesity-related inflammation. However, the data on OSA and markers of systemic inflammation in children are scarce and contradictory [22]. The level of pro-inflammatory cytokines, TNF-α, IL-6 and IL-8 have been found to be increased in the serum of OSA patients. Other cytokines like IL-10 which are regulatory in nature are reduced according to the disease severity. [23].
- **Nocturnal enuresis:** A higher prevalence of nocturnal enuresis has been reported in children with OSA [24]. It can be due to the inhibitory effects of OSA on arousal responses to bladder pressure changes, or effects of increased production of Brain Natriuretic Peptide which will influence the renin-angiotensin pathway, vasopressin, and excretion of sodium and water [25].

Treatment of OSA

Usually treatment should be commenced when the patient has AHI ≥ 5/hr. Management of pediatric patients with OSA depends upon individual risk factors, the presence of OSA-related co-morbidities, and the severity of symptoms associated with each child patient [26]. The European Respiratory Society taskforce put forward a stepwise management approach, until the disease get completely resolved. This may include a combination of different treatment modalities depending on severity and related to the etiology of upper airway obstruction. The steps are:

- **Weight loss if the child is overweight or obese**: Reduction in weight has shown to be effective in obese adolescents [27]. But evidence is lacking in the case of obese younger children.
- **Intranasal corticosteroids**: Newer agents such as fluticasone propionate and mometasone appear to have higher topical potency than older agents such as beclomethasone-17-monopropionate and budesonide [28].
- **Adenotonsillectomy**: It remains the treatment of choice for most children with a strong clinical history of OSA or with OSA documented by polysomnography [11]. Although adenotonsillectomy is the mainstay of treatment for obstructive sleep apnea, recent studies have identified that non-obese patients with moderate OSA and small tonsils have comparable benefits with adenoidectomy alone with less complications [29]. Adenotonsillectomy should be the initial treatment of OSAS in children with other predisposing factors (e.g. obesity, Down syndrome), although further treatment may be necessary.
- **Oral appliance therapy**: It is a useful alternative therapy for patients with sleep-disordered breathing. There are a number of orthodontic treatment modalities that have been suggested to reduce the symptoms of paediatric OSA and at the same time, improve the associated craniofacial abnormalities. These include rapid maxillary expansion, mandibular advancement appliances, and orthopaedic maxillary protraction. The success of orthodontic appliances in improving symptoms of OSA has been attributed to enlarging the airway. Mandibular advancement appliances can increase the lateral dimension of the velopharyngeal airway. This is accomplished as a result of forward positioning of the mandible and reduced collapsibility of the airway. Stimulation of upper airway dilator muscles (genioglossus) with advancement appliances has also been suggested to improve upper airway stabilization. From an orthodontic perspective, mandibular advancement appliances alter the neuromuscular forces on the craniofacial skeleton and dentition, promoting a combination of dentoalveolar changes, and skeletal growth [30].
- **Continuous positive airway pressure (CPAP) and bilevel positive airway pressure (BiPAP):** These are the most commonly used nonsurgical treatments for children with OSAS. CPAP is the treatment of choice when adenotonsillectomy is contraindicated or has failed. Although effective, it is difficult for approximately 20 percent of children to tolerate [9]. Because children grow rapidly, frequent follow-up visits are necessary, and the mask must be adjusted at least every six months. Bilevel Positive Airway Pressure may be indicated in children if nocturnal carbon dioxide is considerably increased which is more common in patients with other conditions like neuromuscular or craniofacial pathologies. Bilevel Positive Airway Pressure has shown better patient compliance when compared to CPAP. The adverse effects of these interventions include nasal congestion, rhinorrhoea, epistaxis, facial erythema, discomfort from air leak, abdominal distension, and

midface hypoplasia. The patient has to be followed up on a long term basis as pressure requirements will change and the interface will have to be adjusted according to the growth and development of the child [13].

- **Tracheostomy:** Tracheostomy, the definitive surgery for upper airway obstruction, is reserved for use in children with severe OSA who have failed to improve with other medical and surgical treatments and in special cases in which these modalities are contraindicated or not tolerated. Tracheostomy often is necessary in children with midfacial hypoplasia wherein craniofacial advancement may be indicated. Tracheostomy must be considered in children for whom traditional surgery is unlikely to be of benefit, such as those with Pierre Robin syndrome [11].

Role of paedodontist in the management of OSA

Dentists see their patients more frequently than their primary care givers and so have a greater opportunity to observe the signs and symptoms of OSA [31]. Dentists who practice sedation dentistry should exercise extra precautions when treating patients with risk of sleep apnea. Minimal and moderate oral conscious sedation and general anaesthesia are commonly used in pediatric dentistry [32]. During sedation, children with OSA have an increased vulnerability of their airway undergoing pharyngeal collapse and of having upper airway obstruction [33].Thus pediatric dentists have an acute responsibility to be able to identify patients who may have OSA.A multidisciplinary approach including the paediatrician, pedodontist and otolaryngologist is always necessary for a treatment plan tailored for each individual patient. A paedontist can prescribe various oral appliance therapies according to the patient requirements who are diagnosed with OSA.A standardized approach in screening is mandatory for pediatric dentists to become more aware and better trained to help accurately and confidently screen for OSA [34].

Prognosis of OSA

Periodic evaluation after every intervention should be done and post-operative symptoms should be evaluated for any further systemic complications. PSG is recommended 6 weeks after adenotonsillectomy, after 12 weeks of montelukast/nasal steroid treatment,12 months after rapid maxillary expansion and after 6 months with orthodontic appliances. Children on Continuous Positive Airway Pressure and Bilevel Positive Airway should be re-evaluated at least every 12 months after initial titration [35]. In children with persistent SDB after intervention, signs and symptoms like laryngomalacia or adenoid regrowth should be suspected. Children with a fast rise in Body Mass Index postoperatively are at increased risk of having recurrent OSA [36]. So, it is very important to monitor weight, dietary modifications and regular physical activity to prevent the recurrence of OSA.

Conclusion

In summary, in developing children early diagnosis and treatment of pediatric OSA may improve a child's long-term cognitive and social potential and overall performance. Polysomnography remains the key to diagnosis, and helps to assess the need for treatment, the risk for perioperative respiratory compromise, and the likelihood of persistent OSA after treatment. Adenotonsillectomy is the mainstay of treatment, although children with complex medical conditions that affect upper airway anatomy and tone may require additional treatment [7]. As a part of the multidisciplinary team, the paedodontist plays a key role in the management of OSA right from the initial diagnosis to treatment modalities including the fabrication of oral appliances for the improvement of oropharyngeal airway.

The earlier a child is treated for OSA, the higher the trajectory for academic and, therefore, economic success [22]. There is proven improvement in terms of behavior, attention and social interactions, as well as improvement in cognitive abilities with academic and social achievements. Therefore, the benefit of treating childhood OSA outweighs the risk where treatment is feasible.

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