Australian Society of Orthodontists

University of Sydney

Obstructive Sleep Apnoea (OSA) in Children

Creating Brighter Futures

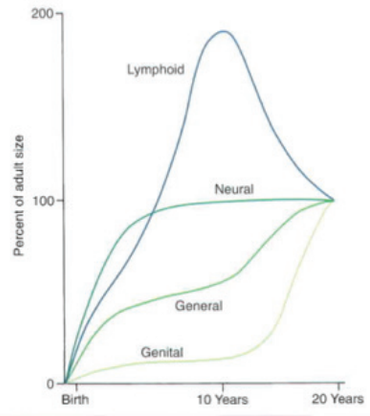
Introduction

Obstructive Sleep Apnoea (OSA) was first recognised as a clinical entity in 1976 and is characterised by episodes of partial or complete upper airway obstruction that occur during sleep, usually with episodic hypoxia, hypercarbia, and sleep fragmentation.[1-3]

Epidemiology

It is estimated that about 10% of children snore and that the prevalence of snoring in pre-adolescent children is equal between boys and girls.[2-4] OSA occurs in 1-3% of snoring children and peaks around the age of 2-4 years.[2-4] The peak in pre-school children is due to the fast growth of the lymphoid tissues as demonstrated by Scammon's Curves in Fig 1.[3-5] OSA may already be present at birth especially in premature infants and those with craniofacial deformities.[4]

Fig 1: Scammon's cuves for growth of the four major tissue systems of the body.[6]



Aetiology & Predisposing Factors

OSA has a multi-factorial aetiology and involves a combination of reduced pharyngeal muscle tone and unfavourable pharyngeal anatomy.[2]

In children adenotonsillar hypertrophy is the biggest predisposing factor for OSA.[3-5] Other risk factors include obesity which is seen with an increasing prevalence amongst children and children with neuromuscular disease and reduced upper airway tone.[2-5]

Dentofacial anomalies such as maxillary transverse deficiency and craniofacial anomalies such as mandibular retrognathia are potential causes of OSA in the paediatric population. Other commonly identified anomalies include an inferiorly placed hyoid bone relative to the mandibular plane, a long soft palate and an increased cranial base flexure.[2,5,7]

Several syndromes with associated craniofacial anomalies can also contribute to OSA. These include maxillary deficiency disorders such as cleft lip and palate, Crouzon and Alpert syndromes; mandibular deficiency such as Pierre Robin sequence; and vertical facial anomalies such as Marfans syndrome.[5]

Pathophysiology

OSA arises when there is a disturbance of the balance between factors maintaining airway patency and those promoting airway collapse.[2,4]

Adenotonsillar hypertophy, obesity and chronic nasal rhinitis can lead to the direct narrowing of the upper airway whereas craniofacial anomalies can reduce the volume of the oronasopharyngeal cavity indirectly from the tongue being lowered and falling into the oropahrynx.[2,4,7]

Signs & Symptoms

OSA in children has been associated with a number of signs that should not be overlooked by the general dentist.[5] Typical signs include behavioural problems such as aggressiveness, hyperactivity and poor impulse control. Unlike in adults, poor sleep for children does not necessarily lead to daytime sleepiness, but is often expressed as behavioural problems such as outlined above. Neurocognitve deficits may also be evident and studies have shown that OSA children display attention span problems, learning difficulties and academic demise. It may also lead to shyness and social withdrawal.[1-4]

Dentists tend to see their patients more regularly than a medical practitioner would, therefore patients that show signs of OSA should be questioned regarding symptoms that might warrant further investigation.[5] A simple sleep questionnaire can indicate whether a child may have OSA. Parents may report symptoms like heavy breathing to loud snoring, pauses in breath, snorts or gasps, restless sleep, sleeping in unusual positions especially with the neck extended backwards, unwillingness to go to sleep, night waking at 1 to 4am, difficulty in waking; parasomnia (nightmares), bruxism, and enuresis (bed wetting).[2-4] Medical conditions such as asthma, chronic nasal obstruction, growth impairment, gastroesophageal reflux disease, cor pulmonale and stertor may also be associated with OSA.[3,5]

Dentists should also be alerted to clinical findings such as periorbital venous pooling, enlarged tonsils and adenoid facies. Enlarged lingual and pharyngeal tonsils will be visible intra-orally, but the adenoids are not. The tonsillar size can be described using the Malampati score where Type 0 indicates the absence of tonsils, and Type IV indicates lateral tonsils that touch each other in the midline. (Fig 2)[1,3]

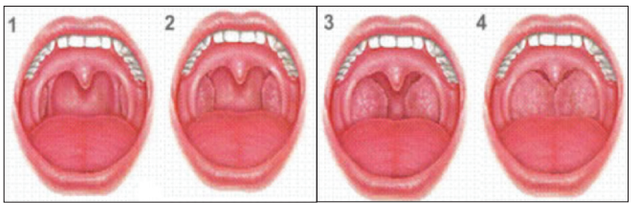


Fig. 2: Malampati classification[8]

You may wish to share this issue of Brighter Futures with your hygienists and other staff members.

A cephalometric radiograh will show both the adenoids and the tonsils as a radio-opaque mass as outlined in Fig 3.[3,5]

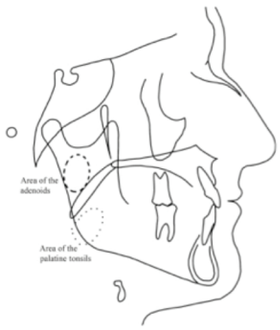


Fig.3: Lateral cephalometric radiograph tracing that illustrates the adenoids as a dashed line and the tonsils as a dotted line.[5]

Any of these findings should prompt the dentist to refer the patient to a sleep physician for further evaluation and tests.[9]

Diagnosis

To determine whether a patient has OSA requires a clinical history and a physical examination together with the gold standard test which remains overnight Polysomnography (PSG). PSG quantifies specific events during sleep.[10] This requires the patient to stay overnight at a sleep laboratory and provides information on the number and duration of complete (obstructive apnoea) or partial (obstructive hypopnoea) obstructions per hour of sleep known as the mixed and obstructive apnoea-hypopnoea index (MOAHI). In children a MOAHI higher than 1 or an apnoea-hypopnoea index (AHI - includes central apnoeas or pauses in breathing as well as obstructive apnoeas/hypopnoeas) of more than five is considered abnormal. A MOAHI greater than 5 is considered moderate, greater than 15 severe. Other parameters that are measured by PSG include oxygen saturation, carbon dioxide level, cardiac arrhythmias, respiratory disturbances, arousals and muscle movement.[2-4]

Risks

OSA has a very high morbidity if left untreated and has been linked with growth disturbances, cardiovascular disease, cor pulmonale, academic difficulties and behaviour issues.[1,2,4] General anaesthesia or sedation of children with OSA may lead to sudden respiratory decompensation and should be carried out with caution.[3]

There has also been speculation that OSA can cause dental malocclusions. Obstruction of the airway as seen in OSA will cause an altered breathing pattern, such as mouth breathing. This leads to a change in head posture, jaw position and tongue position. Classically the head is tipped back to open the airway, this causes the mandible to drop and rotate backwards and the tongue to be lowered. If these postural changes are maintained long term, it will cause over-eruption of the posterior teeth, which in turn will lead to the development of an anterior open bite, proclination of the incisors and an increased overjet. The pressure from the stretched cheeks may also lead to a narrowing of the palate and maxillary dental arch. These characteristics are often referred to as adenoid facies or long-face syndrome.[6,11]

Unfortunately it is not as clear-cut as Proffit's equilibrium theory[6,11] described above and the relevance of nasorespiratory obstruction and its effect on facial growth continues to be widely debated. Animal research studies done by Harvold[12] showed that obstructing monkeys nasal passages will lead to mouth breathing with resulting changes in their malocclusion. In most monkeys the mandible became more prognathic though, but the variation in their response may be an indication that the method of adaptation will lead to a specific type of malocclusion. However, humans and primates do not have the same breathing patterns and total nasal obstruction in humans is very rare. Linder-Arendson[13] reported craniofacial growth changes in patients after the removal of their adenoids and tonsils due to a return to nasal breathing, but the same hypothesis has not been supported by other studies.[14]

Furthermore, it was found that even though a greater percentage of long-faced children were mouth-breathers, the majority were nasal breathers. Thus, facial appearance is not diagnostic of the respiratory mode.[3,6]

Treatment

It is important to realize that each case of OSA in children needs to be assessed individually and that different types of treatment will be appropriate for different aetiological contributors. The treatment should be based on the cause of airway obstruction, severity of OSA and the desires of the family. A mild case of OSA in an overweight child can be simply managed with weight loss and monitoring.[3]

Adenotonsillectomy

By far the most common aetiological factor is enlarged adenoids and tonsils. Thus the standard treatment for OSA in children is an adenotonsillectomy. It has been found though that it may persist or return in 10-20% of patients with mild OSA.In patients with moderate to severe OSA persistence of OSA can be as high as 75%. These patients may have other contributing factors that should be addressed.[2,4,15,16] Additional tests such as an oral examination for regrowth of the tonsils and a nasal exam for septal deviation or turbinate hypertrophy should be done to determine the reason for the persistence of their OSA.[1,3]

Nasal sprays

Nasal obstruction due to allergic rhinitis can be treated with nasal steroids and leukotriene antagonists which are highly effective in increasing the airways in young children with very mild OSA. Nasendoscopy or nasal respiratory tests may indicate that the obstruction is in the nasal passages rather than the oropharynx and that this may be the appropriate line of treatment.[3,4]

Continuous Positive Airway Pressure (CPAP)

80% of children with Down syndrome have OSA and their chance of cure by adenotonsillectomy is lower due to a midface deficiency accompanied by a narrow maxilla, hypotonia and an enlarged tongue. These patients often need Continuous Positive Airway Pressure (CPAP) treatment. The success of the treatment is dependant on patient acceptance and parent cooperation.[3,4] Concern has been expressed in the past on the growth modification effects of CPAP as it will have the same mechanical effect as a head gear appliance, restricting maxillary growth with a resultant midface deficiency.[1,5] Therefore close monitoring of treatment by a paediatric sleep physician and from a dentofacial perspective is mandatory, with timely adjustment of mask and pressure parameters proving effective in preventing abnormal facial growth.

Rapid Maxillary Expander (RME)

Craniofacial deformities should not be overlooked and these patients should be sent to an orthodontist for evaluation so that multi-disciplinary treatment may be performed. The severity may differ and along with it the mode of treatment.[1]
Less severe dentofacial and craniofacial deformities such as a deficient maxilla in the transverse aspect or a retrognathic mandible can be treated orthodontically. When examining the patient, transverse deficiencies may be expressed as a high narrow palatal vault with buccally tipped molars or with the molars in cross bite. For these patients a rapid maxillary expander (RME) can be very beneficial, not only to treat their underlying orthodontic problem, but also to improve their OSA.[5,7,14,15,17]

BRIGHTER FUTURES

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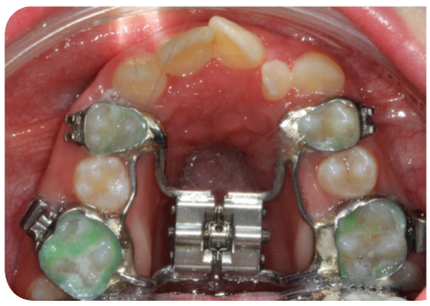

Fig 4. Rapid maxillary expander (RME)

The exact mechanism for decreasing OSA by means of maxillary expansion is not fully understood, but it is known to improve nasal airway ventilation. Cephalometric studies report an increase in the width of the nasal floor and palate as the maxilla expands with a resultant decrease in the airflow resistance due to an increase in the volume of the nasal cavity.[3,19] Cone-beam computed tomography (CBCT) has shown a decrease in the intraoral airway volume after RME, which allows for the tongue to rise and sit in closer proximity to the palate. This improves the position of the tongue and as a result increases the pharyngeal airway volume.[19] For the small percentage with residual OSA, and adenotonsillectomy following expansion may be indicated.[3]

Orthopaedic Corrections

In Class III patients, the RME may be combined with a face mask (reverse pull head gear) to protract the maxilla and correct a midface deficiency and subsequently enlarge the posterior airway.[5]

Mandibular advancement by means of a splint is well documented in adult patients and success rates vary between 50-80%.[20] Class II patients may benefit from sagittal growth modification to reposition the mandible more anteriorly and thereby opening the airway.[15] A Cochrane review found insufficient evidence for the use of functional appliances in the treatment of OSA in children, but acknowledges that it may be helpful in children with craniofacial anomalies that are risk factors for OSA.[2]

Functional appliance treatment is generally reserved for patients on the brink of adolescence, as there is a high risk for relapse if it is carried out prior to the growth spurt. In severe cases functional appliance therapy may be carried out at a younger age to improve the mandibular position. It is advised that a PSG test is carried out with the appliance in the mouth to determine the effectiveness. If it is indeed effective in treating the child's OSA, the oral appliance can be used to cure the condition by promoting dento-alveolar and skeletal growth with the mandible in a more forward position.[2, 21] In young children the retention phase (eg night time wear) will have to continue past the pubertal growth spurt to ensure a lasting outcome.

Surgical Treatment

Surgery is usually the last resort for persistent OSA and may include: uvulopalatopharyngoplasty (UPPP) when a long and thick palate and uvula are the likely site of obstruction; tongue base surgery for children with hypotonia, micrognathia or macroglossia; and maxillofacial surgery for severe craniofacial deformities. Growth considerations in children make the decision for surgery more complex, as early surgery may inhibit further growth. In children with severe craniofacial deformities such as Pierre-Robin syndrome with micrognathia of the mandible, a tracheotomy is often indicated due to the severe restriction of the airways. Mandibular advancement may be an alternative to tracheotomy or long-term CPAP.[3,6]

Treatment Outcome

Children with moderate to severe OSA (MOAHI>5) should have PSG three months after adenotonsillectomy as the persistence of OSA is not uncommon in this group. Waiting three months will allow surgical oedema to resolve before repeat PSG[3]. In addition, any child with residual snoring or apnoea following adenotonsillectomy and patients with an increased risk of surgical failure due to obesity, a syndromes such as Down syndrome, and craniofacial or dentofacial deformities should be referred for a PSG post-operatively. Similarly, PSG is recommended for all children with OSA after treatment with RME or other oral appliance to assess the efficacy of the appliance and the level of residual disease.[9]

Relationship with Adult OSA

Most children with OSA do not present with a positive family history and to date there is no conclusive evidence suggesting that childhood OSA predisposes a patient to recurrence of OSA as an adult. It can be speculated though that untreated OSA in children can continue into adulthood with severe morbidity and complications.[4]

Conclusion

Dental professionals are well equipped to provide treatment as part of a multi-disciplinary team for patients with OSA due to their education in facial growth and development as well as knowledge on craniofacial and dentofacial anomalies. For this reason it is important that dentists are familiar with OSA in children so that they can implement appropriate referrals for definitive diagnosis as it can be a severely debilitating disorder.[2,5]

References available upon request.