Paediatric Respiratory Reviews 16 (2015) 174–181

Contents lists available at ScienceDirect

Paediatric Respiratory Reviews

Review

Dental Treatment for Paediatric Obstructive Sleep Apnea

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EDUCATIONAL AIMS

The reader will be able to:

- Appreciate the complex interplay between normal respiration, craniofacial growth and development and its contribution to paediatric obstructive sleep apnea (OSA).
- Discuss the current evidence supporting the use of rapid maxillary expansion, oral appliances and distraction osteogenesis in the treatment of paediatric OSA.
- Define the indications and limitations for dental treatment for paediatric OSA

ARTICLE INFO

Keywords:
Obstructive sleep apnea
Rapid maxillary expansion
Oral appliances
Distraction Osteogenesis

SUMMARY

Paediatric obstructive sleep apnea (OSA) is common and its prevalence is expected to increase due to the rise in childhood obesity. Recent research has shown that many children, both syndromic and non-syndromic, who exhibit mouth breathing as a result of upper airway obstruction, may also exhibit dentofacial anomalies. Although adenotonsillectomy and continuous positive airway pressure have been classically proposed as the primary treatment modalities for paediatric OSA, there are significant limitations to both therapies. Therefore newer treatment modalities are needed. Current research has focused on emerging dental treatment options for paediatric OSA, such as rapid maxillary expansion, oral appliances and distraction osteogenesis. However, there are few randomized trials assessing the effectiveness of these novel dental therapies for paediatric OSA, and hence further research is required to advance the field.

INTRODUCTION

The obstructive sleep apnea syndrome (OSAS) in children is defined as a disorder of breathing during sleep characterized by prolonged partial upper airway obstruction and/or intermittent complete obstruction (obstructive apnea) that disrupts ventilation during sleep and fragments sleep patterns [1]. It is estimated that 3–26% of young children are habitual snorers [2–5] with 1.2% to 5.7% of the general paediatric population exhibiting OSAS [6–8]. The peak incidence has been reported to occur between the ages of 2 and 8 years old and is generally thought to be due to a discrepancy between the size of lymphoid tissue and airway calibre. OSAS is characterised by upper airway collapse during sleep due to an imbalance between upper airway structure contributed by factors such as adenotonsillar hypertrophy, craniofacial anomalies, upper airway neuromuscular tone and obesity. The sequelae of OSAS include neuropsychological and cognitive impairment, systemic [9,10] and pulmonary hypertension [11] and endothelial dysfunction [12].

Adenotonsillectomy (AT) has been generally proposed as the treatment of choice for children with paediatric OSAS. However, several studies have highlighted the multi-factorial nature of this condition with craniofacial anomalies, syndromic conditions such as Downs syndrome, obesity and OSA severity playing key factors in residual OSAS after AT intervention [13–16]. Complete resolution of OSA defined as AHI $<$ 1 event/hour has been reported to range between 25% - 40% [15,17,18]. The recent Childhood

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http://dx.doi.org/10.1016/j.prrv.2014.11.002
1526-0542/

Adenotonsillectomy Trial (CHAT) has provided new evidence evaluating the efficacy of early AT versus a conservative "watchful waiting" approach in children (age range 5-9 years) with moderate OSA (AHI range 2-30 event/hr) [19]. Marcus and investigators found beneficial effects of early AT improvements in certain domains including polysomnographic outcomes and quality of life, but no significant change in attention or executive function. Nevertheless, the role of AT in milder OSAS is unclear and warrants further investigation.

Nasal continuous positive airway (nCPAP) pressure remains a non-surgical treatment alternative for paediatric OSAS. However, limited compliance to this mode of therapy remains a realistic limitation in children [20–22]. Moreover, the long term implications of nCPAP therapy with mask delivered systems in growing children is poorly understood. Several studies have documented adverse dentofacial side effects including mid-facial hypoplasia following prolonged nCPAP therapy [23–25]. However, a recent small cephalometric study in children (mean age 9.0 years) undergoing PAP for a minimum of 6 months for at least 6 hours of use showed negligible change [26]. Nevertheless there is a need for treatment alternatives that are equally effective, and preferably targeting the individual pathophysiology in each child.

The current review will discuss the relationship between craniofacial development and paediatric OSAS and focus on emerging dental treatment modalities, including rapid maxillary expansion, oral appliance therapy, and maxillo-mandibular surgical interventions such as distraction osteogenesis in the co-management of paediatric sleep disordered breathing (SDB).

CRANIOFACIAL GROWTH AND FUNCTIONAL IMPLICATIONS ON CRANIOFACIAL AND DENTAL FORM

The influence of the mode of breathing on craniofacial and dentofacial growth is widely debated and still steeped in much controversy [27,28]. It is generally accepted that cartilage is the primary determinant of craniofacial growth at the cranial base synchondroses. According to the functional matrix theory proposed by Moss and Salentijn [29], growth in the craniofacial and dentofacial complex occurs in response to functional needs and possibly in response to growth of the nasal cartilage [29]. This theory is based on the principle that normal nasal breathing promotes harmonious growth and exerts influence on the development of craniofacial structures by stimulating the associated structures of the head and neck region during mastication, swallowing and breathing [30,31]. Linder-Aronson proposed the cause and effect relationship between increased airway resistance and craniofacial disharmony or malocclusion [32]. Chronic nasal obstruction leads to mouth breathing, resulting in an anterior and lowered posture of the tongue, open-mouth posture, a lowered mandibular posture and reduced orofacial muscle tonicity. This is thought to be a compensatory mechanism in response to the decreased nasal airflow in an attempt to maintain respiration. The imbalance results in the disharmonious growth and development of the orofacial structures and may manifest as discrepancies in craniofacial and dentofacial form [32,33]. These may include maxillary constriction and retrusion, under-development of the mandible, altered head and neck posture and excessive proclination of maxillary teeth. Animal studies in Rhesus monkeys with induced nasal obstruction have documented a combination of these features including an increase in the facial height and reduction in maxillary length and width [31,34]. Solow and Kreiborg proposed the soft tissue stretch theory and postulated that mouth breathing leads to altered head posture and an altered pattern of muscle recruitment, this in turn presenting as an adverse contributory factor in craniofacial morphogenesis [35].

Mouth breathing has a multifactorial aetiology and may result from anatomical obstructions due to enlarged palatine and pharyngeal tonsils, enlarged turbinates, nasal septal deviation, nasal polyps or allergic rhinitis. Children who mouth breathe due to adenotonsillar hypertrophy commonly exhibit a forward head posture, a retrognathic mandible, an increased anterior facial height, a steep mandibular plane, and lowered position of the tongue and hyoid bone [36]. Adenoidectomy promotes a change to nasal breathing and appears to facilitate maxillary and mandibular growth [36] and normalization in incisor position [37] after 5 years. Hence, the phenomenon of mouth breathing is important as this chronic habit may adversely influence growth and development of the craniofacial and dentofacial complex.

CRANIOFACIAL AND DENTAL MORPHOLOGY IN OSA

Numerous studies have identified a range of craniofacial and dental morphological characteristics associated with OSA. These are summarised in Tables 1 and 2.

Children with obstructed breathing may exhibit craniofacial abnormalities. Lofstrand et al. [38] compared 48 obstructed children with a control group of 4-year-old children with ideal occlusion. Children who snored every night or had apnoeic episodes showed a higher rate of disturbed sleep, mouth breathing, and a history of throat infections. A smaller cranial base angle and a lower ratio of posterior/anterior total face height were also seen. The obstructed children had a narrower maxilla, a deeper palatal height, a shorter lower dental arch with a higher prevalence of lateral crossbite [38]. In a recent study, children with chronic snoring were also documented to have a dolichofacial growth pattern with high mandibular plane angle, narrow palate, and severe crowding in the maxilla and the mandible, allergies, frequent colds, and habitual mouth breathing [39]. The negative impact of respiratory obstruction is not isolated to sleep disordered breathing alone. Children with asthma also exhibit increased malocclusion and mouth breathing [40,41] with significant deviations in dento-alveolar morphology such as maxillary constriction [42].

In support of these studies, Lindsay Gray in 1975 reported similar observations in his cohort of 310 patients [43]. He proposed the use of rapid maxillary expansion (RME) for medical conditions (poor nasal airway, septal deformity, recurrent ear or nasal infection, allergic rhinitis and asthma) and dental indications (crossbite, class III malocclusion, maxillary constriction, and cleft palate). Considerable improvement in colds and respiratory

Table 1
Craniofacial morphological abnormalities in OSA

• Maxillo-mandibular retrusion in relation to anterior cranial base
• Increased mandibular plane angle
• Increased anterior facial heights
• Lowered hyoid bone
• Reduced mandibular length
• Reduced pharyngeal airway space
• Elongated soft palate
• Increased tongue size

Table 2
Dental morphological abnormalities in OSA

• Maxillary constriction
• High and narrow palate
• Open Bite
• Anterior and Posterior Crossbite
• Maxillary/mandibular dental crowding
• Decreased intermolar width