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Adenoid Hypertrophy in Pediatric Sleep Disordered Breathing and Craniofacial Growth: The Emerging Role of Dentistry

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Study Objectives: Summarize and synthesize the most recent evidence about adenoid hypertrophy, impact on craniofacial growth, role in sleep disordered breathing, and effects of treatment.

Methods: Literature review of relevant manuscripts from dentistry, orthodontics, otolaryngology, and sleep medicine.

Results: Adenoid hypertrophy is the most common cause of nasopharyngeal obstruction in children; the most common cause of pediatric sleep disordered breathing (SDB); and can be an etiologic cause of altered craniofacial growth characterized by long face, retrusive chin, and narrow maxilla. Early detection and treatment may mitigate or resolve negative effects of adenoid hypertrophy. Adenoidectomy remains a front line treatment for the majority of cases, although alternative treatments must be considered when different SDB etiologies and co-morbidities are present. Best available evidence suggests that rapid maxillary expansion and adenoidectomy work synergistically to resolve SDB symptoms, and often both treatments are necessary for full treatment effect.

Conclusions: Primary care dentists, pediatric dentists, and orthodontists have an important role in early detection of adenoid hypertrophy. Emerging evidence continues to demonstrate dental treatments as playing an increasingly important role in multidisciplinary management of pediatric SDB.

Keywords: adenoids, adenoidectomy, craniofacial growth, diagnosis, palatal expansion, obstructive sleep apnea, orthodontics, sleep disordered breathing

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The adenoids are a collection of lymphatic tissue located in the most superior-posterior aspect of the nasopharynx. They are situated at the inflection point between the horizontally oriented nasal passage and the vertically oriented oropharynx. Being a lymphoid tissue, the adenoids play a role in immunity housing large numbers of immunocompetent cells such as B cells, T cells, lymphocytes, and macrophages.[1] As a result, the adenoids are highly prone to inflammation when an immune response is elicited against foreign antigens.[1]

Even in healthy children, a physiologic amount of adenoid enlargement is a part of normal craniofacial growth and development. The adenoid lymphoid tissue naturally increases to its largest size sometime between age 5-10 years, then continually decreases in size until adulthood.[2,3] Since children of this age range naturally have some element of relative lymph enlargement, additional inflammation—actual inflammatory hypertrophy beyond physiologic adenoid enlargement—can introduce partial or complete nasopharyngeal obstruction.[4]

Epidemiologic studies have reported a high prevalence of adenoid hypertrophy in children. One large study of 1,132 subjects observed a frequency of 27% for children between 5 and 7 years, and 19% to 20% for children between the age of 8 and 14 years.[5] Other smaller studies have observed frequencies of 37.9% among 370 children between 3 and 9 years[6] and 57.7% among 213 children between 6 months and 15 years.[7]

When adenoid hypertrophy occurs in a chronic state, there can be long periods of partial or complete impairment of nasal function,[8] which may lead to mouth breathing to overcome the limited passage of air through the nasopharynx.[6] Chronic nasopharyngeal obstruction is believed to increase the risk for altered craniofacial growth and increase the risk of pediatric sleep disordered breathing.

THE EFFECT OF ADENOID HYPERTROPHY

Adenoid Hypertrophy and Altered Craniofacial Growth

Although previous research studied the link between nasal function and facial pattern, it was Linder-Aronson's seminal work that helped solidify the association between adenoid hypertrophy and altered human craniofacial growth. He noted that adenoid obstruction occurred in all facial types, but children with adenoid hypertrophy presented more frequently with a recurrent craniofacial phenotype. This phenotype was characterized by a narrow maxillary dental arch, posterior dental crossbite, steep mandibular plane, and long anterior face height.[9] Such a craniofacial phenotype was often termed "adenoid facies."

Linder-Aronson acknowledged that, in theory, a genetically driven facial pattern could also cause the nasopharyngeal obstruction. However, he favored a hypothesis that nasopharyngeal obstruction—whether by adenoid hypertrophy or other etiology—increased resistance to nasal airflow such that children were obligated to mouth breathe. The resulting open mouth posture became the driving force behind altered craniofacial growth. He theorized that during mouth breathing, the tongue assumed a lower posture to facilitate oral airflow and therefore no longer rested in the palate. Without the tongue providing internal muscular force, transverse maxillary

development would be hindered, as no expansive force would be present to overcome the external constrictive pressure of the cheek muscles. Put simply: Linder-Aronson viewed maxillary transverse constriction and vertical growth pattern as a result of mouth breathing when there was concomitant evidence of nasopharyngeal obstruction.

While the pathophysiologic mechanism postulated by Linder-Aronson has been debated, the association between facial growth and nasal function has been repeatedly demonstrated.[10-13] Recent studies have demonstrated chronic nasal obstruction by mechanisms other than adenoids—such as deviated septum[14] or chronic rhinitis[15]—also cause the same altered craniofacial growth pattern as adenoid hypertrophy. Two recent systematic reviews and meta-analyses concluded that nasopharyngeal obstruction can be a primary etiologic factor causing "adenoid facies."[16,17]

Adenoid Hypertrophy and Sleep Disordered Breathing

Sleep disordered breathing is a spectrum of disorders unified by respiratory disturbance or inadequate ventilation during sleep.[18] In this context, sleep disordered breathing can range from primary snoring to upper airway resistance syndrome to severe obstructive sleep apnea.[19] In the pediatric population, the epidemiology of sleep disordered breathing is poorly described, as its presence and consequences on overall health and wellness have been widely underappreciated. Only recently has pediatric sleep disordered breathing become more widely acknowledged as a public health problem. Best available estimates suggest the frequency of obstructive sleep apnea is approximately at 1% to 5%,[20] while the frequency of sleep disordered breathing (i.e., snoring) is estimated much higher, ranging from 3% to 27%.[20] The consequences of sleep disordered breathing to overall health can be severe. Neurocognitive dysfunction including attention deficit, hyperactivity, reduced grades in school, and aggression, and cardiovascular dysfunction including hypertension, ventricular hypertrophy, valvular damage, and cor pulmonale and delayed growth have all been reported.[21-27]

Factors such as obesity, asthma, ethnicity, preterm birth, and environmental irritants are all etiologic contributors and comorbidities of pediatric sleep disordered breathing.[1,28-33] However, in the pediatric population, adenoid hypertrophy is the most pervasive primary etiology.[1-3,18,34]

THE EFFECT OF ADENOIDECTOMY

Effect of Adenoidectomy on Craniofacial Growth

Linder-Aronson's work solidified the connection between adenoid hypertrophy and altered craniofacial growth. The next logical step was to investigate whether treating adenoid hypertrophy could normalize craniofacial growth.

Subsequent studies suggested a return to normal growth after adenoidectomy was possible. Multiple prospective, non-randomized clinical trials[4,35,36] demonstrated a tendency to a normalization of the mandibular plane angle (i.e., decrease in long face morphology) over 5-year follow-up, but no noticeable change during the first year. Such a finding is not unexpected, as noticeable growth changes take time to occur. Though both studies demonstrated statistical significance, some trends were questionable and the clinical significance was not profound. These findings suggest that some normalization does occur, but growth pattern may not be fully restored to normal.

Current investigations have also produced mixed results. A recent prospective, non-randomized trial[5,37] evaluated growth in a pediatric population (n = 34, mean age 5.6 years) with OSA for 5 years after adenotonsillectomy (A&T). Initially, the treatment group subjects had distinct facial morphology consistent with "adenoid facies," while control subjects did not. Five years after A&T, there was no discernable difference between the treatment and control groups. Conversely, a non-randomized prospective trial evaluated growth differences between treated and untreated controls (n = 80) after 1 year and found no difference.[38] If the conclusions of Linder-Aronson's original intervention study[35] are valid, one year may not be sufficient time to observe a growth change, and Souki et al.[38] may have incorrectly accepted the null hypothesis.

Unfortunately, none of the cited studies had strong methodological features; therefore, inconsistencies between studies may be due to study biases (methodological flaws). Yet the study that reported the strongest results[37] also treated the youngest subjects (mean age 5.6 years). The other studies[35,36] reported subjects mean age 7.5 years and 8.2 years, respectively. A recent cross-sectional study[13] suggested that children with obstructive adenoid hypertrophy should be treated before the age of 6 to achieve total normalization of craniofacial growth. Therefore the spectrum of results across studies may be confounded by an unaccounted covariate—age. We hypothesized that the clinical implication might be that children with nasopharyngeal obstruction should be treated before age 6 for the best prognosis of normalized craniofacial growth.

Effect of Adenoidectomy on Pediatric Sleep Disordered Breathing

At the present time, A&T is the evidence-based, first-line surgical treatment of pediatric obstructive sleep apnea.[18,33] One meta-analysis[39] described an average reduction of 13.9 AHI events following A&T and success rate of 82.9%, while another more recent systematic review estimated a success rate of only 66%.[40] However, the level of evidence generally is low, primarily coming from case series and cohort studies.[39,41]

Even though A&T is the current first surgical step, there are significant questions regarding its universal efficacy. Recent publications have reported failure rates of 49% to 75%.[42,43] Continuous positive airway pressure (CPAP) has become the standard of care treatment for children with failed A&T. A growing body of research suggests that certain populations have a particularly poor prognosis following A&T. The presence of midface deficiency, obesity, family history of SBD, certain ethnicity, asthma, gastroesophageal reflux disease (GERD), septum deviation, and chronic rhinitis all have various degrees of evidence to suggest a more guarded prognosis to A&T treatment.[29,44-47]

Concurrent evidence is growing that alternative treatments are essential for SDB management, such as anti-inflammatory medication,[48,49] proton-pump inhibitors,[50] and orthodontics.[47] Unsurprisingly, each of these treatment alternatives addresses specific comorbidities that may compromise the prognosis of A&T therapy.

In conclusion, recent evidence demonstrating a more guarded prognosis of A&T treatment for pediatric SDB suggests

significant gaps in knowledge in current diagnostic standards. Further research is needed before clinicians can provide consistently accurate, patient-specific prognosis for A&T. Even though the role of A&T requires tailoring, its importance cannot be underestimated in SBD management. Because of the high prevalence of adenoid hypertrophy as a primary etiology in children with SDB, adenotonsillectomy will always remain an important front-line surgical treatment option. Simply put, A&T should be seen as an initial, simple, and important treatment, but no longer viewed as a universal or ultimate surgical treatment for pediatric SDB.

DIAGNOSIS OF ADENOID HYPERTROPHY

Numerous tools are available to evaluate the nasal and nasopharyngeal airway. Clinical exam alone, acoustic rhinometry, lateral cephalometry, multi-row detector CT imaging, video fluoroscopy, and cone beam computed tomography (CBCT) have all been described as methods for evaluating nasopharyngeal patency.[8,51-55] However, each of these methods has significant drawbacks. Clinical exam alone lacks the sensitivity to be useful.[51] Lateral cephalograms provide fair diagnostic value but tend to overestimate adenoid size.[55] Multi-row detector CT scans and video fluoroscopy are both very accurate but require specialized equipment and expose patients to unjustifiably high levels of radiation.[53,54]

Beyond all other diagnostic methods, nasoendoscopy using a standardized grading system is the gold standard for diagnosis of adenoid hypertrophy.[55-57] Nasoendoscopy is minimally invasive, highly reliable, and easy for an otolaryngologist to perform. However, performing nasoendoscopy is outside the scope of practice for other health-care providers concerned with adenoid size, such as orthodontists or sleep medicine specialists. While nasoendoscopy is an excellent diagnostic procedure, gaining access to an otolaryngologist is the most difficult step to getting a reliable diagnosis of adenoid hypertrophy.

A recent study evaluating CBCT has shown promising results.[55] Sensitivity and specificity of 88% and 93%, respectively, were reported. However, there were also challenges. Strong intra-observer and inter-observer repeatability among trained evaluators was observed, but concerns were raised about the diagnostic ability of casual clinicians' interpretation of CBCT images. In addition, there were concerns about unacceptably high ionizing radiation exposure when not used for more medically serious purposes. While CBCT can be very accurate and reliable for diagnosing adenoid size, the results cannot be taken as justification for liberal use of CBCT imaging. Evaluation of adenoid size alone provides insufficient grounds to acquire a CBCT image. However, when CBCT images are acquired for other valid reasons—such as orthodontic records—the images can be secondarily evaluated for adenoid hypertrophy with a high degree of certainty.

ROLE OF THE DENTIST AND ORTHODONTIST IN AIRWAY MANAGEMENT

The dentist has several important roles in airway management. First, for patients with a history of nasopharyngeal obstruction and altered facial growth, orthodontic manipulation of the teeth and skeleton may help normalize the dentofacial appearance, thus improving the patient's esthetics and correcting the associated malocclusion.

Second, a dentist is well situated to play an important role in early detection and screening of certain children with airway dysfunction. Through timely diagnosis, an orthodontist may altogether prevent, or at least limit, the development of malocclusion and altered craniofacial growth. Using the same diagnostic skills, an orthodontist can screen for children with sleep disordered breathing. By facilitating timely referral to an otolaryngologist and/or sleep physician, and orthodontist can substantially improve a patient's overall health and quality of life.

Third, through orthopedic manipulation of the facial skeleton, an orthodontist may be able to contribute to the treatment of specific forms of sleep disordered breathing. Early research in rapid palatal expansion[47] and mandibular repositioning appliances[58] are promising and may in the future become cornerstone treatments for select pediatric sleep disordered breathing patients. However, significant research is still required before an orthodontist can reliably treat sleep disordered breathing.

At the present time dentist's most important role in airway management is to act as an early detector of airway dysfunction, and coordinate timely referral to appropriate health professionals. However, if recent research is any indication of future clinical practice, dentists are likely to gain increasing prominence also in managing specific sleep related problems.

CONCLUSION

In summary, chronic adenoid hypertrophy is the most common etiology of pediatric sleep disordered breathing. It has been strongly implicated in the altered craniofacial growth pattern termed "adenoid facies"—that is, long face, maxillary constriction with an associated dental crossbite, increased overjet, and weak chin projection. Currently adenotonsillectomy is the front-line treatment for pediatric sleep disordered breathing.

A new paradigm is emerging that recognizes a multitude of additional causes for nasal obstruction and pediatric sleep disordered breathing. Recognition of the comorbidities and collaborative disease contributors can be very important for evaluating individual patient risk profiles and prognosis of treatment. Unsurprisingly, new treatment options are emerging as alternative or collaborative therapy modalities that specifically address these alternative disease etiologies.

Dentists and orthodontists have an increasingly important role in the early detection of children with sleep disordered breathing and adenoid hypertrophy. Dentists' ability to recognize altered craniofacial growth patterns and access to alternative diagnostic techniques enables dentists to screen children with sleep disordered breathing with high accuracy. Furthermore, new research suggests that dentists and orthodontists may have a critical role in treating select subgroups of children with sleep disordered breathing.

The understanding of pediatric sleep disordered breathing is evolving to recognize the important diagnostic and unique treatment roles dentists may contribute. Therefore it is important that dentists learn to recognize the signs, symptoms, risk factors, and comorbidities of pediatric sleep disordered

breathing in daily practice. In doing so, dentists can have a positive impact on their patients' overall health and quality of life.

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