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REVIEW

Craniofacial anatomical determinants of pediatric sleep-disordered breathing: A comprehensive review

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Abstract

Purpose: This narrative review aims to elucidate the anatomical features of sleep-disordered breathing (SDB) in children. By identifying key structures and intervening proactively, we seek to alter craniofacial growth patterns and improve functional outcomes for SDB children.

Methods: The literature on pediatric sleep-disordered breathing (PSDB), pediatric obstructive sleep apnea (OSA), anatomical predispositions, and the relationship between skeletal deformity and PSDB was examined using PubMed and Google Scholar databases, covering studies from 2006 to 2024.

Results: Forty relevant articles were reviewed, focusing on craniofacial characteristics associated with PSDB. The etiology of PSDB is multifactorial, with adenoid and palatal tonsil enlargement being the most common cause. While adenotonsillectomy is recommended as the primary treatment, residual SDB may result in craniofacial skeletal deformities contributing to upper airway constriction. Typical craniofacial phenotypes of SDB include excessive vertical growth and constriction of the maxilla, a retruded mandible, and posterior rotation, known as a Class II hyperdivergent pattern. Conversely, Class III with an underdeveloped maxilla shows a relatively lower risk for SDB due to reduced nasal cavity and nasopharyngeal airway volumes. Transverse maxillary constriction with a high, deep palatal vault is a significant risk factor. Additionally, nasal obstruction and low tongue posture, with or without a short lingual frenulum, are identified as craniofacial risk factors for SDB development in children.

Conclusion: Early diagnosis and intervention are critical in managing PSDB. Dentists, through screening and early treatment, can significantly influence craniofacial growth and health outcomes. A multidisciplinary approach is essential for effective management, improving the quality of life and long-term health of affected children.

KEYWORDS
craniofacial traits, growth modification, mouth breathing, pediatric sleep disordered breathing, pediatric obstructive sleep apnea

Sleep-disordered breathing (SDB) is defined as a disorder of breathing during sleep characterized by snoring, increased upper airway resistance, prolonged and repetitive partial upper airway obstruction, and/ or intermittent complete obstruction essentially disrupting normal ventilation, oxygenation, and sleep quality.[1,2] SDB includes a spectrum of conditions from habitual snoring to obstructive sleep apnea (OSA), defined by recurrent episodes of upper airway obstruction with often associated reductions in oxygen saturation, occurring during sleep and terminating with arousal.[3,4]

The prevalence of habitual snoring is 18%–81% in adults and 7%–27% in children and adolescents, whereas that of OSA is 9%–38% in adults and 2%–5% in children and adolescents.[3,5,6] Ikävalk et al.[7] reported that 10% of children aged 6−8 years from the general population have SDB. Although SDB is now recognized more sensitively

than before, it remains a highly under-diagnosed condition. Pediatric SDB is more challenging to diagnose due to the limited availability of polysomnography (PSG) findings, less sensitive results from home sleep tests (HST), unreliable questionnaires administered to children, and limited information obtained from imaging analysis.[8,9] For pediatric SDB, it is necessary to consider different diagnostic criteria and subjective symptoms.

Pediatric OSA was first described in 1976,[10] with Guilleminault et al.[11] highlighting that pediatric OSA differs from adult OSA. Children with OSA experience more disturbed nocturnal sleep rather than excessive daytime sleepiness and exhibit more behavioral problems, particularly school-related issues such as attention deficit, poor academic performance, and hyperactivity, often classified as attention-deficit-hyperactivity syndrome. Other symptoms include nocturnal enuresis, sleep terrors, sleepwalking, confusional arousals, NREM parasomnias, depression, insomnia, and psychiatric problems. Although cardiology-related symptoms are infrequent, tachybradycardia is regularly noted.[11]

Dentists should be aware of these awake and sleep symptoms to identify children at increased risk of SDB. Early recognition can help prevent its health consequences, such as cardiovascular disease, metabolic disturbances, delayed somatic growth, abnormal craniofacial growth, and depression.[11] By identifying these signs early, dentists can help interrupt the progression of childhood SDB into more complicated OSA in adulthood by breaking the vicious cycle between craniofacial deformation, respiratory function aggravation, and improving the quality of life in childhood.

There is a putative association between SDB and OSA with craniofacial disharmony resulting from abnormal growth. Previous studies have established strong evidence associating craniofacial morphology and SDB in children. This article reviews the anatomical features of SDB in children to better understand the patterns of craniofacial growth. By identifying specific structures and intervening proactively when appropriate, we can alter the trajectory of growth and improve function for children with SDB (Figures 1 and 2)

PATHOPHYSIOLOGIC PHENOTYPES OF PEDIATRIC OBSTRUCTIVE SLEEP APNEA (POSA)

The etiology of POSA is multifactorial, arising when the balance between airway patency and airway collapse is disrupted. In children, the main phenotypic causes that impact upper airway collapsibility can be classified into non-anatomical phenotypes such as neuromotor dysfunction and inflammation and anatomical phenotypes including skeletal deformities and soft tissue abnormalities.[1,12–15]

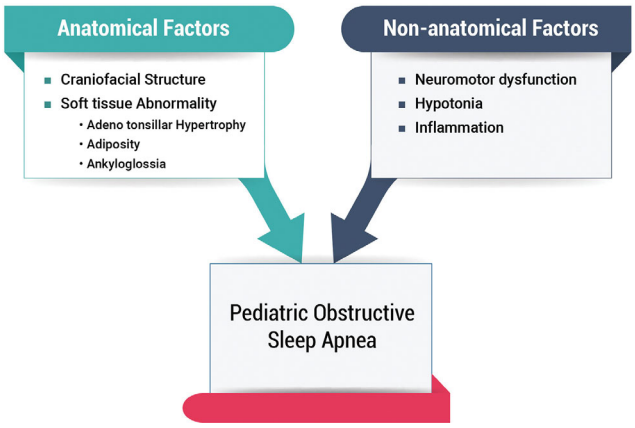

FIGURE 1 Pathophysiologic factors of pediatric OSA.

The most common cause of upper airway obstruction is the enlargement of the adenoids and palatal tonsils, which are part of Waldeyer's lymphoid ring. Recent studies have suggested that upper airway obstruction may also be related to the size and structure of the upper airway, exacerbated by nasal obstruction.[14–16] Abnormalities in craniofacial structures, such as a narrow and/or retruded maxilla, steep mandible, and increased lower facial height, may contribute to this condition.[1,12–17] These anatomical factors are important risk factors for pediatric OSA.

Neuromotor dysfunction plays a significant role in causing upper airway obstruction during sleep, characterized by changes in pharyngeal muscle tone and reflex response. Studies suggest that children's upper airways are more resistant to collapse compared to adults, as evaluated through mechanical properties assessments, including responses to resistive loading.[14] Conversely, dysfunctional soft palate due to relative hypotonia in young children, even without accompanying disorders, could contribute to OSA.[14] This particular phenotype might not respond effectively to physical treatments, making CPAP the primary intervention. However, concerns have been raised regarding long-term CPAP use potentially leading to midfacial flattening in children.[18]

Inflammatory conditions, such as bronchitis, can induce swelling and inflammation in the upper airway, narrowing the passage for airflow. This obstruction disrupts normal breathing patterns, leading to episodes of apnea during sleep. In children, airway obstruction during sleep can result in decreased oxygen supply, potentially causing severe oxygen deprivation to the brain, and exacerbating the airway obstruction. Treatment response may be reduced in children with inflammation, as conditions like bronchitis can interfere with effective management. Consequently, it is essential to consider the presence of upper airway inflammation and its impact when managing pediatric OSA. Treatment often involves steroids or other anti-inflammatory medications to alleviate airway obstruction and enhance sleep quality.[16]

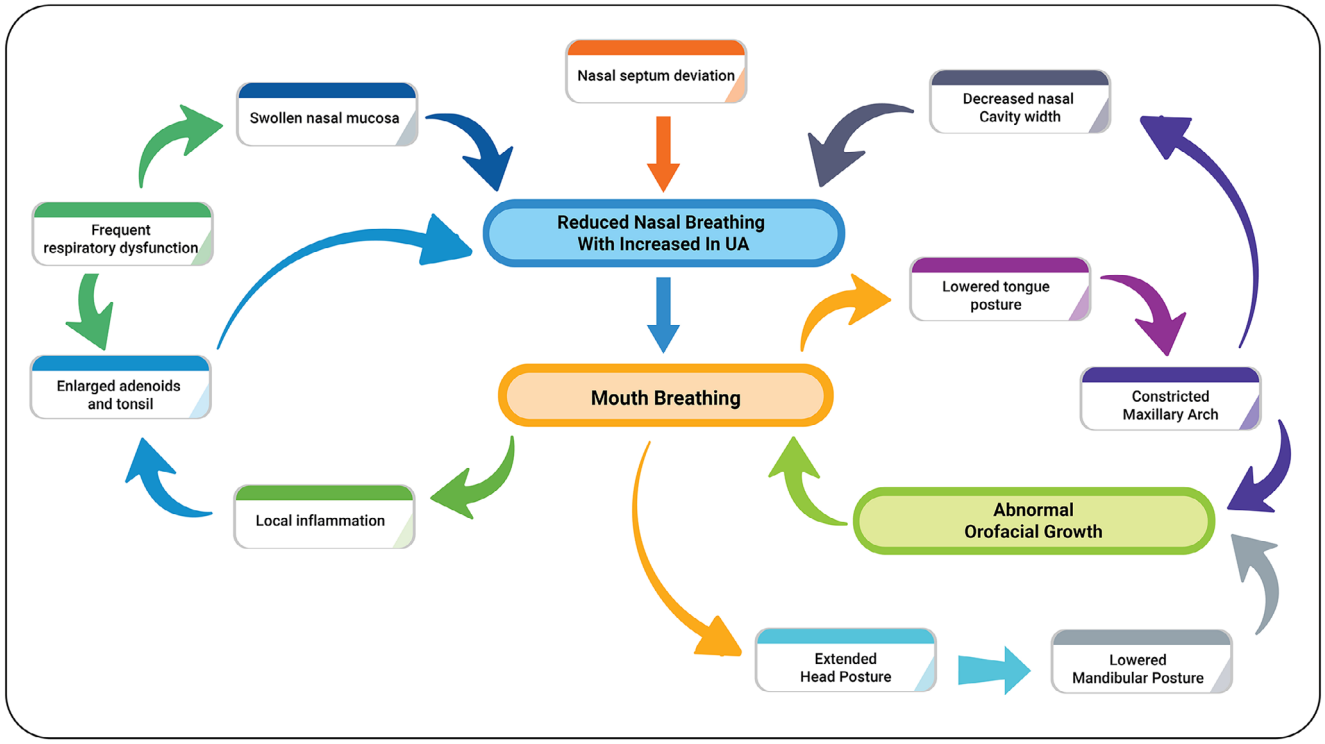

FIGURE 2 Influence of sleep-disordered breathing on orofacial growth.

CRANIOFACIAL ANATOMICAL PREDISPOSITIONS TO POSA

Skeletal deformity

Craniofacial morphology is closely linked to the dimensions of the upper airway. Changes in the size, position, and interrelationships of craniofacial structures including the cranium, nasomaxillary complex, mandible, and hyoid bone can significantly impact the dimensions of the upper airway. As the upper airway is situated beneath the skull and behind the facial structures, any developmental alterations in these craniofacial structures can have a profound effect on the upper airway dimensions. Notably, the nasomaxillary complex and mandible play pivotal roles in determining the size of the upper airway.

A recent study[19] identified several craniofacial measurements as potential risk factors for OSA. These include sagittal and transverse mandibular-maxillary relationship, vertical mandibular rotation with upper anterior face height, anterior cranial base length with inclination. In summary, a thicker and longer soft palate, along with an increased hyoid-mandibular distance, a posterior rotation of the mandible, an elongated upper anterior facial height, a skeletal Class II relationship, maxillary constriction and a shorter and more posteriorly inclined anterior cranial base are suggestive of a higher risk of OSA. These craniofacial features may manifest as a convex facial profile, which could be noticeable to pediatricians even without the aid of X-ray imaging, prompting a referral to a sleep laboratory if concerns arise.[19]

Children with craniofacial syndromes are at risk of SDB.[20] The most common sleep disorder in these children is OSA, though central apneas during sleep have also been reported. The etiology of OSA is multifactorial including anatomical craniofacial and upper airway factors such as midface hypoplasia and glossoptosis, as well as factors resulting in increased multi-level airway collapsibility such as oropharyngeal dysfunction.[16,21]

Children with Down syndrome often experience OSA due to factors such as adenotonsillar hypertrophy, airway size reduction from midface hypoplasia, and a small mandible.[18,21] While adenoid and tonsil volumes are smaller in Down syndrome patients, other soft tissues like the tongue and soft palate remain similar in size to healthy children, leading to a proportionate decrease in the upper airway size and soft tissue crowding. Similarly, children with craniosynostosis are at a higher risk of OSA, primarily due to midface hypoplasia. Central apneas in craniosynostosis may be caused by pressure on respiratory centers from underlying Chiari malformation or narrowing of the craniocervical junction.

Children with cleft lip and palate also exhibit a heightened prevalence of OSA. Studies have demonstrated smaller pharyngeal airways and differing craniofacial dimensions compared to healthy controls.[20,21] Additionally, the disruption of oropharyngeal musculature due to the cleft affects speech, swallowing, and compromises airway patency, particularly during sleep. The classic triad of micrognathia, glossoptosis, and resultant airway obstruction, initially described by Pierre Robin in 1923, is commonly associated with cleft palate. Mandibular hypoplasia leads to the superior and posterior

displacement of the tongue between the palatal shelves.[20,21] This is unsurprising, considering oropharyngeal dysfunction has been observed in patients with cleft lip and palate, affecting speech and swallowing.[20,21]

SOFT TISSUE ABNORMALITY

Adenotonsillar hypertrophy

Hypertrophy of the adenoids and tonsils is a significant risk factor in pediatric SDB, as it affects the size and patency of the upper airway. The overall craniofacial and upper airway features play a crucial role in determining the available space for air exchange, and adenoids and tonsils impact function not only due to their absolute size but also in relation to the available anatomical space.[12,17] The lymphoid tissue of the Waldeyer ring tends to be more developed between the ages of 3 and 6 years, coinciding with the peak incidence of OSA in children. Adenoid and/or tonsil hypertrophy represent the most common causes of upper airway lumen reduction in children, as evaluated by the Friedman Grading Scale.[22] This hypertrophy notably contributes to the narrowing of the retropalatal area, which frequently becomes obstructed due to its small cross-sectional area. Children with Grade IV tonsils are particularly prone to developing SDB due to oropharyngeal narrowing and lateral collapse.

Symptoms of adenotonsillar hypertrophy include mouth breathing, nasal congestion, hyponasal speech, snoring, chronic sinusitis, recurrent otitis media, as well as potential impacts on brain development, and emotional well-being. Some authors emphasize the role of inflammation in adenotonsillar hypertrophy and OSA development, citing increased expression of inflammatory response mediators in tonsils and improvements with anti-inflammatory treatments like corticosteroids, suggesting a multi-disciplinary treatment approach. Linder-Aronson et al.[1] proposed that adenotonsillar hypertrophy induces mouth breathing, disrupting the balance of labial, lingual, and cheek muscles, potentially resulting in facial anomalies. Adenotonsillectomy represents the gold standard surgical treatment, with studies demonstrating resolution of polysomnographic findings in a significant percentage of children with OSA. However, long-term follow-up is recommended, especially in children with concurrent comorbidities, to prevent residual OSA.[23] A previous study found that a Mallampati scale score of 3 or 4 was significantly related to the persistence of SDB post-adenotonsillectomy. In a retrospective review of clinical and polysomnographic data from children aged 2−17 years with SDB, 98.2% of the children with incomplete improvement had a high Mallampati score at study entry, compared to 24.6% of those that responded well to adenotonsillectomy. The Mallampati score correlates with the narrowness of the upper airway which usually involves both maxilla and mandible. Initially described to clinically predict a difficult intubation, it is not surprising that Mallampati scores of 3 and 4 are more commonly noted in subjects also recognized to have a narrow maxilla and small and/or retroplaced mandible. Of the 211 children who responded to adenotonsillectomy, 74.8% were found to have a narrow maxilla and 67.8% had a small or retroplaced mandible. This score reflects the narrowness of the upper airway, typically involving both the maxilla and mandible.[3]

The presence of one facial feature alone does not guarantee a poor response to adenotonsillectomy. A combination of craniofacial and soft tissue findings is often necessary to assess the persistence of abnormal breathing during sleep. Further refinement of clinical anatomical scoring may be warranted to improve diagnosis and treatment outcomes.

Adiposity

Adiposity has been identified as a significant risk factor for the development of SDB in children.[3,17] A recent study indicated that increased visceral fat is particularly associated with more severe manifestations of SDB.[24] Another study found that a higher body fat percentage in children aged 6–8 years was linked to an elevated risk of developing SDB in subsequent years. Additionally, the presence of adipose tissue under the chin was associated with an increased risk of SDB during follow-up.[24]

The mechanism underlying the association between adiposity and SDB may involve several factors. The added mass load on the upper airway and respiratory muscles can lead to structural and functional alterations, reduced chest wall compliance, changes in respiratory drive, and impaired functional residual capacity. These factors collectively contribute to an increased risk of upper airway obstruction.[25] In Finland, the prevalence of overweight is reported to be 10% among children and 26% among adolescents. Notably, obese children have been observed to have a higher risk of persistent SDB even after undergoing adenotonsillectomy.[25] It is important to note that the relationship between overweight/obesity and SDB in children is complex and may be influenced by other factors such as age, ethnicity, and craniofacial morphology.[3,25]

Understanding these associations is crucial for developing effective treatment strategies for SDB in pediatric populations. Addressing adiposity through lifestyle and behavioral interventions may help reduce the risk and severity of SDB in children.

Ankyloglossia

Ankyloglossia, commonly known as a short lingual frenulum, has been found to be linked to the development of a small upper airway and SDB.[19] This condition can cause difficulties in sucking, swallowing, and speech and can contribute to oral-facial dysmorphism, further reducing upper airway support.[26,27]

During embryogenesis, the lingual frenulum, a vestigial embryological element, becomes mostly fibrous in consistency due to adhesion between the tongue and the floor of the mouth. Genetically controlled apoptosis separates the

tongue from the primitive pharynx during this process. Early intervention through the clipping of the short lingual frenulum is suggested when difficulties are identified in infancy. However, the long-term outcomes of such clipping performed after the first few months of life are reported to be unpredictable. A short lingual frenulum alters the position of the tongue, which can have a structural impact on the craniofacial region.[11] This abnormal tongue position may result in an anterior and posterior crossbite, disproportionate mandibular growth, and abnormal maxillary growth, potentially serving as a craniofacial risk factor for OSA.[13,19,26,27]

CRANIOFACIAL SKELETAL CHARACTERISTICS IN PEDIATRIC SLEEP-DISORDERED BREATHING (PSDB)

Cranium

The cranial base plays a crucial role in craniofacial growth by integrating the anatomically and functionally distinct patterns of growth in various adjacent regions of the skull, including the brain, nasal cavity, oral cavity, and pharynx. The synchondroses formed via endochondral ossification in the cranial base are important growth centers for the neurocranium. Within the nasomaxillary complex, there is development of the anterior cranial base and nasal cavity. The growth of the nasomaxillary complex is influenced not only by the spheno-occipital synchondrosis but also by the activity of the synchondroses of the cranial base, particularly at the clefts of the following sutures: inter-malar, inter-maxillary, inter-palatine, maxillo-malar, and temporo-malar.

The intermaxillary suture remains active postnatally and is influenced by specific functions such as suction, mastication, swallowing, and nasal breathing. By the end of the preschool stage, up to 80% of cranial development is completed. Although cranial base lengthening and flexing are primarily controlled by genetics, they can also be influenced by respiratory patterns and head posture.

Cranial base flexion (basicranial flexion) occurs early in life and helps determine the facial phenotype. For example, an obtuse cranial base angle will displace the mandible more distally, leading to a tendency for a class II (retrognathic mandible) sagittal jaw relationship Conversely, cranial base angle closure displaces the mandible anteriorly, resulting in a tendency for a class III (prognathic mandible) sagittal jaw relationship. Impaired cranial base flexion tends to maintain the cranial base narrow and long (dolichofacial) and to displace the mandible distally.[10,12,14,28,29]

The systematic review by Finke et al.[14] with cephalometric analysis showed that male patients with OSA showed a decreased nasion-sella-basion angle (relation of the anterior cranial base relative to the clivus) indicating either posteriorly rotated anterior cranial base with a more prognathic face type or a more anteriorly positioned temporomandibular complex. And they showed a relevantly shorter anterior cranial base compared to the controls.[14] In systematic reviews, previous studies have shown a higher frequency of OSA in skeletal Class II, while conflicting and inconsistent results are observed regarding the cranial base angle due to sample heterogeneity.[14] It is considered important to recognize that the angulation and size of the cranial base influence the position of the maxilla and the mandible relative to the cranial base.[14]

Nasomaxillary complex

In lateral cephalometric analysis, maxillary retrusion, expressed by the sella-nasion-A-point angle, was significantly decreased in children aged 5–9 with OSA.[30] However, other meta-analyses have shown no significant differences in the SNA angle between children with OSA and those without OSA, indicating controversy in the sagittal relationship of nasomaxillary complex. The lack of untreated controls, long-term data, and high interindividual variability contribute to ongoing controversy regarding craniofacial morphology and the anteroposterior (AP) relationship between the maxilla and mandible.[14,30–32]

Most studies on craniofacial morphology have focused on the maxillary transverse dimension indicating that nasal breathers generally have larger maxillary transverse dimensions than mouth breathers. Conversely, children with SDB tend to have higher palatal height, suggesting a constricted maxillary arch and decreased palate surface.[12,15,23,33] Habumugisha et al.[15] have found that maxillary width and palatal area parameters play significant roles in explaining the nasopharyngeal airway volume. As the maxilla makes up most of the lateral walls of the nasal cavity, the size of the nasopharyngeal airway may be connected to maxillary width. This highlights the importance of maxillary width size, palatal area and volume, and maxillary position relative to cranial base as potential contributors to OSA.

Mandible

Many studies have examined the mandibular morphology of children with SDB compared to healthy children, highlighting the mandible's crucial role in determining the upper airway's size. A meta-analysis supports the argument that children with SDB typically exhibit a higher ANB angle and a slightly decreased SNB angle, along with a remarkably more obtuse articular angle and an increased mandibular plane angle. These findings indicate a retrognathic mandible and increased vertical dimension with a hyperdivergent pattern.[1–3,14,32,34] A meta-analysis has shown the strongest correlation between mandibular plane hyperdivergency and the severity of OSA.[34]

Hyoid

Cephalometric analysis has identified a lowered hyoid position as a characteristic associated with the severity of OSA, potentially related to a low tongue position.[13] Iwasaki et al.

emphasized the role of hyoid bone placement in relation to skeletal maxilla-mandibular relationships. In healthy people, the hyoid bone is located at the level of the C3-C4 cervical vertebrae, whereas in patients with OSA, it is usually found lower at the level of C4-C6. Shintani et al. compared children aged 1–9 years old with OSA to a control group by cephalometric analysis, revealing that the GoGn-H angle and MP-H distance of the hyoid bone were significantly lower in the OSA group for children aged 3 and 6 years.[30]

Upper airway

Children with OSA often, but not always, exhibit smaller upper airway volume, area, and size compared to healthy children. Additionally, in the OSA group, structures such as the adenoids, tonsils, retropharyngeal nodes, deep cervical nodes, and parapharyngeal fat pads tend to be larger. A meta-analysis by Katyal et al. using two-dimensional lateral cephalometric evaluation documented a significant association between pediatric OSA and reduced PNS-AD1 and PNS-AD2 distance.[32]

Regarding the volumetric evaluation, Van Holsbeke et al. investigated whether anatomical properties of the airway were correlated with OSA severity in children at 6 years of age. They concluded that children with OSA had a lower volume of the upper airway, especially in the region between the choanae and uvula, including the overlap region, and a lower mean cross-sectional area of the upper airway.[30] In addition, the soft palate and the total soft tissues were found to be larger in OSA.[16] Habumugisha et al.[15] also showed in all skeletal classes, nasal breathers had higher pharyngeal airway volumes than mouth breathers. Oropharyngeal airway volume and total pharyngeal airway volume revealed substantial increases in nasal breathing individuals compared to mouth breathing subjects in all skeletal groups.[15,35]

In summary, a lot of anatomical insight underscores the importance of early evaluation in children with suspected SDB to identify those at higher risk and to develop targeted therapeutic strategies that address these specific anatomical features. However, while many studies highlight significant craniofacial differences in children with OSA, the evidence is still mixed due to methodological variations and the complexity of craniofacial growth patterns. Further research with more rigorous control and long-term follow-up is needed to clarify the close relationship between craniofacial morphology and SDB in children.

RELATIONSHIP BETWEEN SKELETAL DEFORMITY AND PSDB

Effects of PSDB on skeletal deformation

Guilleminault et al. illustrated how mouth breathing induces negative feedback on orofacial development and worsens SDB.[36] Proper nasal breathing is essential for the maturity of the nasomaxillary complex, as obstruction of the nasal airway can significantly reduce nasal airflow and disrupt the harmonious development of the craniofacial complex. According to Moss's functional matrix theory, the development of the nasomaxillary complex and subsequent changes in size, shape, and location are secondary compensatory responses to nasal airflow. As a functional need, nasal airflow continuously stimulates for vertical growth as well as transverse expansion of nasomaxillary complex.[15,37] For example, chronic mouth breathing with extended head postures and lowered tongue postures in children under the age of 5–6, can disrupt cranial base flexion, leading to a dolichocephalic pattern characterized by narrow facial width and mandibular retrusion.[29] This, along with maxillary constriction and downward displacement, alters the growth location of the condyle, shifting cartilaginous production more posteriorly. Consequently, this change results in the downward and backward rotation of the mandible increasing anterior face height during youth development.[17] This, in turn, leads to narrowing of the upper airway, often presenting as adenoid faces. Studies have shown higher face height in children with SDB[1] and reduced nose prominence in children with SDB compared to asymptomatic children.[38]

Shirke et al.[2] indicated that children with SDB have differences in dental arch dimensions, such as increased overjet, buccal crossbite, and openbite, potentially associated with lowered tongue position caused by SDB. A recent systematic review[38] concluded that no firm conclusion can be drawn regarding the effect of SDB on specific malocclusion traits. However, the odds of having SDB were found to be much higher in children with malocclusion, particularly class II and class III malocclusions, and those with higher grades of the Index of Orthodontic Treatment Need (IOTN), which suggests a strong association between SDB and malocclusion.[2]

Problems associated with abnormal nasal breathing were recognized prior to recognition of incomplete treatment with T&A for SDB. The understanding of the relationships between function and craniofacial growth led to experiments on how honing these functions would impact craniofacial development. Myofunctional therapy combined with orthopedic treatment and craniofacial surgery has been shown to correct the deficits caused by abnormal growth patterns.[17]

Effect of progressive skeletal deformity on PSDB development

Anatomical abnormalities of the craniofacial structure may play key roles in increased upper airway resistance and the development of pediatric OSA. Mutual and reciprocal interactions have been reported between the upper airways and craniofacial complex.

The nasal cartilage provides crucial structural support and contributes to the growth of the nasal cavity. The increased incidence of nasal cavity changes with age is compatible with the idea of an underlying cartilage anomaly that becomes

evident only later during childhood. This could reflect abnormal nasal septum cartilage development, which could also be associated with abnormal midfacial growth, such as midfacial hypoplasia. A smaller nasal cavity may result in a deviated nasal septum leading to SDB. As turbinates are not implicated in midfacial growth, this might have been anticipated. The positive correlation between a deviated nasal septum and turbinate hypertrophy might not be surprising, as co-presentation of a deviated nasal septum and turbinate hypertrophy is common in the field of otolaryngology. Identification of nasal abnormalities does not necessarily translate to an increased risk for SDB. However, assessing children for nasal cavity abnormalities in addition to the PSQ score before referring them for further sleep assessment is important.[33]

In children at cervical vertebral maturation stages 2−3, growth of the nasomaxillary complex continues downward and forward with nasal cavity enlargement, the maxillary growth pattern is primarily in the vertical direction as the width of the maxilla is determined. At the adolescence stage, the nasomaxillary complex will continue to grow downward and forward, although at this stage the nasomaxillary complex will grow more vertically than horizontally.[12] At the growing stage, excessive vertical growth and constriction of the maxilla, along with a retruded mandible and posterior rotation such as Class II hyperdivergent pattern, can impact the dimensions and minimum cross-sectional area of the upper airway, leading to obstruction of the upper airways and mouth breathing as the craniofacial phenotype.

Effect of orthopedic treatment on PSDB

Children with craniofacial abnormalities are at a high risk of persistent OSA even after undergoing adenotonsillectomy. Based on craniofacial phenotyping, target approaches for growth modification can be considered.

A recent umbrella review[39] demonstrated that functional appliances positively affect the oropharyngeal airway by promoting forward growth of the mandible, provided the treatment is initiated before the pubertal peak and maintained for at least 6 months. This subsequent adaptive advancement of the tongue base, hyoid, and soft palate is facilitated by the genioglossus and palatoglossus muscles. Interestingly, oropharyngeal enlargement in three dimensions leads to a minimum cross-sectional increment, resulting in the recovery of suppressed growth hormone levels, increased oxygen saturation, and improvement of AHI. The release of growth hormone stimulated by these changes may further promote mandibular growth, enhancing both facial aesthetics and breathing function.[40]

Maxillary protraction has the potential to improve respiratory function in children with maxillary hypoplasia.[27,41] The application of a facemask stimulates forward maxillary growth, resulting in the expansion of the nasopharyngeal airway through anterior and inferior displacement of the posterior nasal spine. This process promotes the advancement of the soft palate, increasing the velopharyngeal dimension and compensating for the transient reduction in pharyngeal airway space due to hypertrophic adenoids during the pre-adolescent period. However, changes in the oropharyngeal airway dimensions following maxillary protraction are limited, attributed to the interplay between increased tongue volume facilitated by maxillary advancement and the potential posterior positioning of the tongue due to mandibular clockwise rotation.[12,27,41]

For children with a typical craniofacial phenotype characterized by a constricted nasal cavity, narrow nasal floor, and maxillary constriction, skeletal expansion becomes necessary. Rapid maxillary expansion is commonly applied to address nasal obstruction by increasing the volume of the narrow nasal cavity in sagittal, vertical, and transverse dimensions. This expansion is achieved through the forward and downward displacement of the nasomaxillary complex, as well as the expansion of the maxillary basal area. Additionally, the expanded oral cavity allows for the forward and upward repositioning of the tongue, indirectly opening the oropharyngeal airway.[27,41]

Huang and Guilleminault emphasized that orthodontists should consider nasal expansion in young children with SDB as early as possible for early establishment of nasal breathing to prevent irreversible changes in craniofacial growth.[31]

CONCLUSION

Dentists play a crucial role in identifying patients with SDB. During routine dental exams, dentists can assess a small upper airway and other anatomical risk factors for SDB. Early recognition of SDB in children is vital as it can negatively impact health, cognitive development, quality of life, and future potential. It is essential for dentists to differentiate the craniofacial characteristics associated with various pathophysiologic phenotypes of OSA. The most prevalent underlying cause of SDB is commonly attributed to adenoid and palatal tonsil enlargement, peaking in children aged 3–7 years. While adenotonsillectomy is generally recommended as the primary treatment, residual SDB following adenotonsillectomy may result in craniofacial skeletal deformities that contribute to upper airway constriction. One typical craniofacial phenotype of SDB is excessive vertical growth and constriction of the maxilla, along with a retruded mandible and posterior rotation, known as a Class II hyperdivergent pattern. However, Class III with an underdeveloped maxilla has shown a relatively lower risk for SDB than skeletal Class II hyperdivergent patterns which is also a craniofacial phenotype due to the reduced volumes of the nasal cavity and nasopharyngeal airway.[41,42] Regardless of sagittal and vertical skeletal patterns, transverse maxillary constriction with a high and deep palatal vault is a significant risk factor for SDB. Additionally, nasal obstruction, and low tongue posture with or without short lingual frenulum, have been identified as a craniofacial risk factors for the development of SDB in children.

By recognizing the craniofacial risk factor for SDB and understanding the differential growth patterns of the cranium, maxilla, mandible, and lymphoid tissues, dentists can consider orthopedic interventions for growth modification. Through timely targeted interventions to enhance the skeletal framework and upper airway volumes in growing patients, dentists can help prevent or mitigate the progression of OSA.

CONFLICT OF INTEREST STATEMENT

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