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REVIEW

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Craniofacial phenotyping of pediatric sleep-disordered breathing: orthodontic management, timing, and treatment approaches

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Abstract

Pediatric obstructive sleep apnea (OSA) presents a multifactorial etiology distinct from adult OSA, necessitating orthodontists to recognize specific craniofacial features that influence upper airway collapsibility. Accurate diagnosis and effective management of pediatric OSA require understanding these phenotypes, categorized into subtypes such as Class II with retrognathic mandible, vertical maxillary excess, constricted nasomaxillary complex, Class III with retrognathic maxilla, and bimaxillary deficiency. Growth modification treatments targeting the nasomaxillary complex and mandible's dimensions can enhance skeletal growth and airway patency, creating an optimal environment for normal respiratory function and craniofacial development. Tailored treatment strategies based on craniofacial phenotyping can significantly improve outcomes. Orthodontists play a crucial role in guiding craniofacial development at various stages, emphasizing the importance of a collaborative, team-based approach to managing sleep-disordered breathing. According to the differential growth theory, intervention timing and appliance type should be chosen based on the specific bony target and area of respiratory obstruction. The craniofacial complex and dentition undergo significant changes from infancy to adulthood, presenting critical windows for targeted intervention. This article proposes a clinical framework for multidisciplinary care, emphasizing refined phenotyping to determine the most appropriate management strategies. In the future, personalized care will identify children who would benefit most from targeted craniofacial management within an interdisciplinary team setting.

Keywords

Pediatric sleep disordered breathing; Pediatric obstructive sleep apnea; Upper airway; Craniofacial abnormalities; Clinical phenotype

1. Introduction

Pediatric sleep-disordered breathing (SDB) refers to a spectrum of respiratory issues in children that occur during sleep. It ranges from primary snoring to obstructive sleep apnea (OSA), characterized by partial or complete upper airway obstruction leading to disrupted sleep patterns, frequent arousals, and potential daytime behavioral and cognitive problems [1, 2]. It is important to identify children at increased risk of SDB to prevent its health consequences, such as cardiovascular disease, metabolic disturbances, delayed somatic growth, cognitive deficit, depression, and decreased quality of life [3, 4].

The etiology of pediatric OSA is multifactorial and arises when the equilibrium between factors that maintain airway patency and those that promote airway collapse is disrupted. In children, the primary phenotypic causes affecting upper airway collapsibility can be broadly classified into non-anatomic and anatomic phenotypes [5, 6]. Non-anatomic phenotypes include neuromotor dysfunction and inflammation. Inefficient neuromuscular control can lead to inadequate tone and coordination of the upper airway muscles during sleep, making the airway more susceptible to collapse. This includes conditions where the reflexes that normally maintain airway patency are impaired, and reduced muscle tone, particularly during rapid eye movement sleep, can exacerbate airway collapsibility due to various central nervous system disorders or developmental delays. Chronic inflammatory conditions such as chronic rhinitis or asthma can contribute to persistent upper airway inflammation, leading to swelling and increased airway resistance. Allergic reactions can also cause inflammation and congestion in the nasal passages and upper airway, promoting collapsibility [7].

Anatomic phenotypes include soft tissue abnormalities and skeletal deformities. Among soft tissue abnormalities, adenotonsillar hypertrophy is the most common anatomical cause of pediatric OSA. 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. These

enlarged lymphoid tissues can obstruct the airway, influencing airway function not only due to their size, but also in relation to the available space [8]. Obesity can lead to excess fatty tissue around the neck and upper airway, compressing the airway and making it more prone to collapse. This results in structural and functional alterations in respiratory muscles, changes in respiratory drive, and impaired functional residual capacity [9]. The large tongue and ankyloglossia can cause difficulties in sucking, swallowing, and speech, and also contribute to oral-facial dysmorphism, which further reduces upper airway support [10].

Structural abnormalities such as retrognathic mandible, constricted maxilla, and midface hypoplasia are recognized as predominant factors of pediatric OSA. Alterations in the dimensions, positioning, and interrelations of craniofacial structures, such as the cranium, nasomaxillary complex, mandible, and hyoid bone, can markedly influence the upper airway dimensions. Since the upper airway is located inferior to the skull and posterior to the facial structures, any developmental modifications in these craniofacial components can substantially affect the dimensions of the upper airway [11, 12].

Pediatric OSA poses unique diagnostic challenges compared to adult OSA, largely due to limited polysomnography (PSG) findings and less reliable questionnaires. However, dental visits provide a valuable opportunity for effective screening through lateral cephalometry, cone-beam computed tomography (CBCT), and hand wrist imaging, particularly for patients with craniofacial phenotypes. Lateral cephalometric analysis, despite being a static, two-dimensional tool with variable image acquisition, is crucial in orthodontic clinics for identifying pharyngeal narrowing and predicting treatment outcomes. It offers essential insights into the pharyngeal airway and craniofacial structures, demonstrating good diagnostic accuracy for conditions like adenoid hypertrophy. Conversely, CBCT provides a three-dimensional view, enhancing the assessment of airway volume, shape, and site-specific details, despite concerns about radiation exposure and cost. Hand-wrist imaging serves as a valuable tool for evaluating a patient's skeletal maturity and forecasting the onset of pubertal growth spurt, thereby facilitating the identification of the optimal timing for implementing growth modification strategies.

Many pediatric patients visit orthodontists during their growth, presenting an ideal opportunity to identify OSA risk factors and implement growth modification strategies. Recognizing these phenotypic factors is vital for the accurate diagnosis, effective management, and appropriate treatment of pediatric OSA. When craniofacial risk factors contributing to the pathogenesis of OSA are identified, treatment selection should be meticulously tailored to craniofacial phenotyping, enhancing the overall clinical applicability and precision in managing pediatric OSA cases (Fig. 1, Ref. [5, 11, 12]).

This narrative review underscores the significance of identifying pediatric OSA with craniofacial risk factors that contribute to upper airway collapsibility, and of customized treatment strategies based on craniofacial phenotyping. We examined the literature on pediatric sleep-disordered breathing, pediatric OSA, phenotypes, and growth modification, along with orthodontic treatments using PubMed and Google Scholar databases, spanning studies from 1999 to 2024. Seventy relevant articles were identified and reviewed by the primary reviewer. This investigation focused on the clinical phenotyping of pediatric OSA and corresponding treatment strategies.

2. Craniofacial phenotype benefiting from orthodontic management

There have been many attempts at anatomical phenotyping of OSA, aimed at identifying specific structural characteristics that contribute to SDB [13–15]. Even though there is no clear phenotyping of which structure contributes to certain SDB, based on the origin of skeletal deformity, a craniofacial phenotype of OSA can be divided into several main subtypes: Class II with retrognathic mandible, Class II with vertical maxillary excess, constricted nasomaxillary complex, Class III with retrognathic maxilla, and bimaxillary deficiency as shown in Fig. 2 (Ref. [12, 16]).

2.1 Class II with retrognathic mandible phenotype

2.1.1 Mechanism of functional appliance to open the upper airway with mandibular advancement

Previous studies have shown that skeletal Class II subjects, both adults and children, have significantly smaller pharyngeal airway dimensions in the oropharynx compared to skeletal Class I subjects [17]. This reduction in airway dimensions is likely due to the posterior positioning of the tongue and soft palate in individuals with retrognathic mandibles [18]. Functional appliance (FA) treatment has been observed to increase pharyngeal airway dimensions in skeletal Class II subjects, bringing them to levels comparable to skeletal Class I subjects. This suggests that FA treatment can induce greater airway expansion than what is normally observed with growth alone, and these improvements can be maintained until growth cessation [19]. The increase in pharyngeal airway dimensions during FA treatment is attributed to the stretching of the tongue and suprahyoid muscles associated with mandibular advancement, leading to enhanced respiration. Functional appliances can increase oropharyngeal and nasopharyngeal airway volumes and the minimum cross-sectional area (MCA) in growing patients. However, one systematic review found no significant evidence that the antero-posterior position of the hyoid bone is affected during FA treatment [20]. Nonetheless, orthopedic treatment for skeletal Class II mandibular retrusion with FAs may help enlarge airway dimensions and reduce the potential risk of OSA in the future.

While changes in the naso- and oropharyngeal volumes and MCAs with FAs, particularly in the upper pharynx, are typically small to moderate, greater effects are observed in the lower part of the pharynx. This suggests that clinically relevant benefits in airway dimensions or breathing might be attributed to changes in the lower pharyngeal compartment. Significant differences in pharyngeal airway dimensions and pharyngeal wall thickness have been noted between normal patients and those with OSA, with the lower retropalatal/retroglossal areas being the most affected [21]. The exact mechanism behind these changes in the upper airway remains unclear. However,

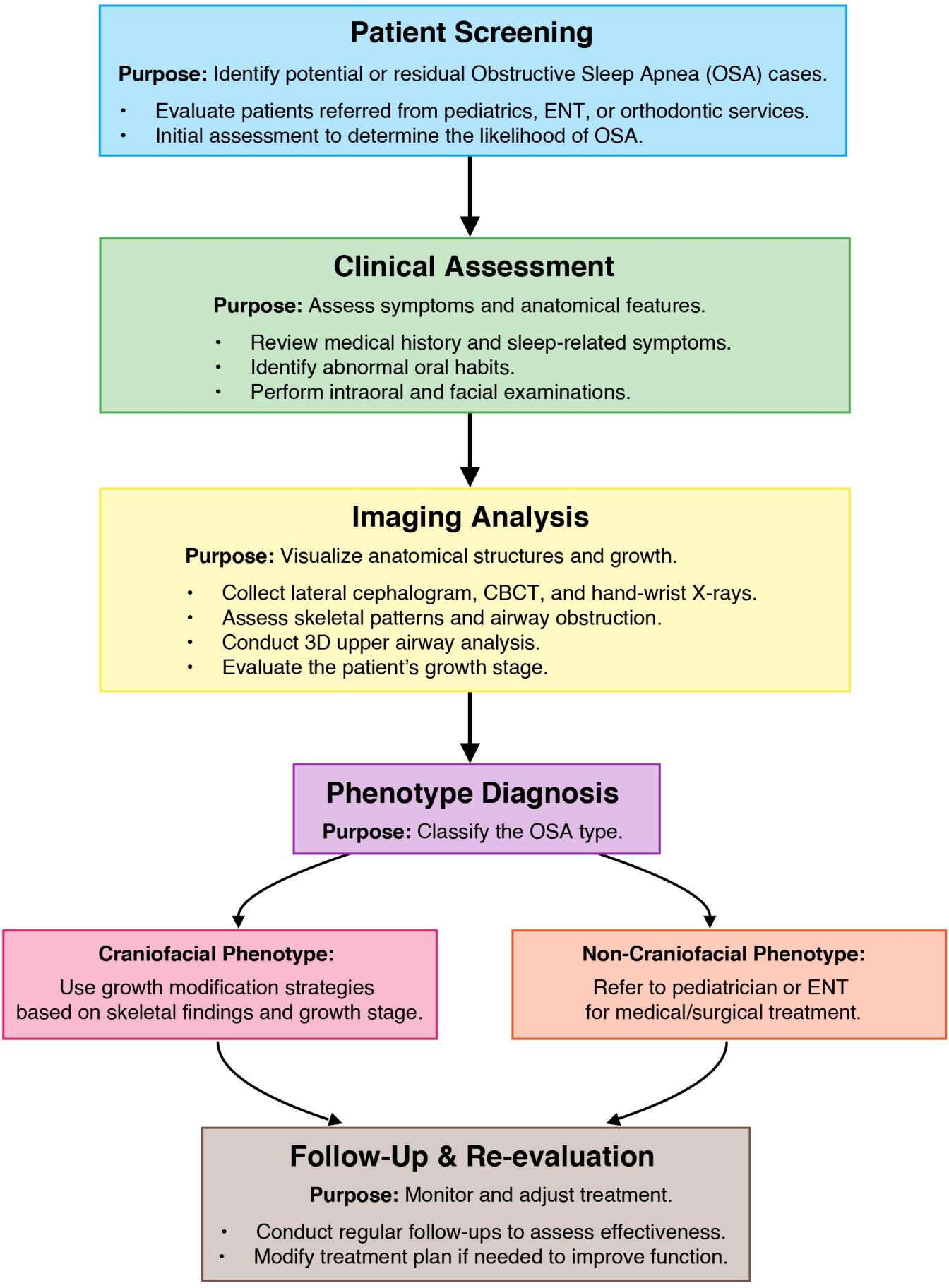

FIGURE 1. Comprehensive assessment and management protocol for pediatric OSA [5, 11, 12]. ENT: Ear, Nose, and Throat; CBCT: Cone-Beam Computed Tomography.

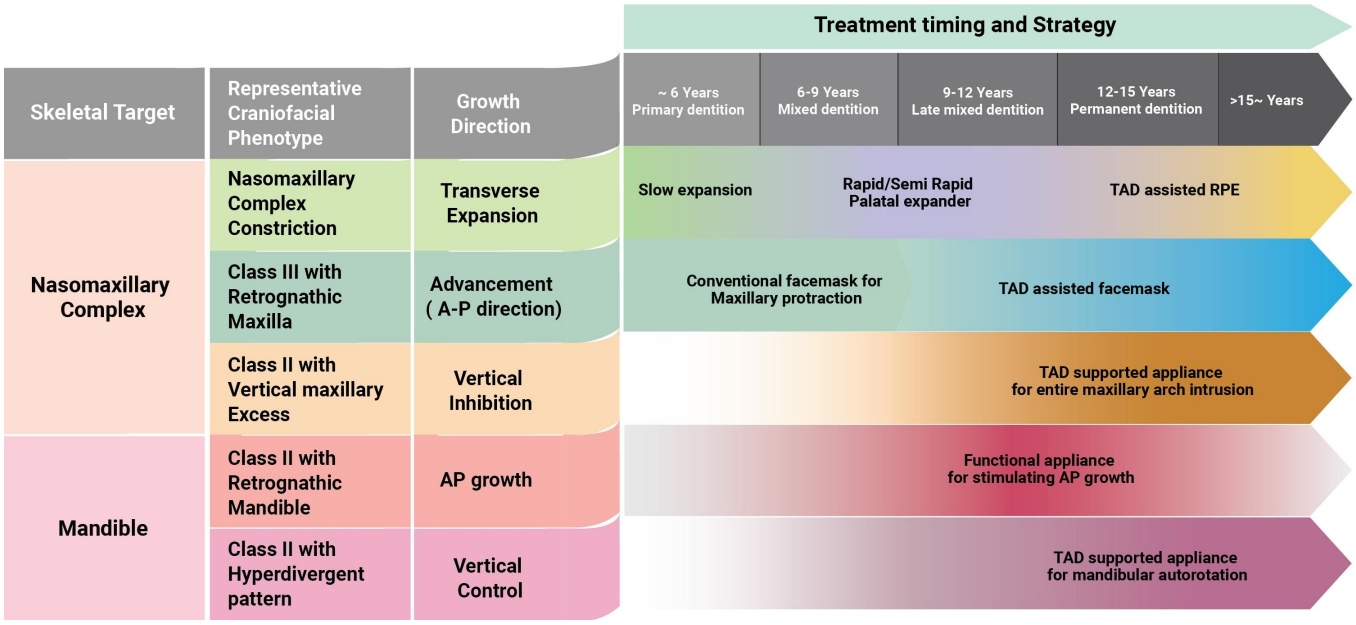

FIGURE 2. Orthodontic strategy for SDB children based on craniofacial phenotype and differential growth theory [12, 16]. This illustrates the timely-targeted orthodontic interventions for SDB children based on craniofacial phenotype. It highlights the importance of appropriately targeted and patient-specific orthodontic treatment as a therapeutic option for SDB children. TAD: temporary anchorage device; RPE: rapid palatal expansion; AP: anteroposterior.

it is hypothesized that the anterior displacement of mandible may cause anterior traction on the tongue and hyoid bone, leading to adaptive changes in the soft palate and increased pharyngeal airway dimensions. This hypothesis is supported by observations of anterior repositioning of the soft palate following FA treatment for Class II mandibular retrusion, as the tongue moves away from the palate [22–25].

2.1.2 Functional improvement from the functional appliance

In a study examining the impact of a FA on the apnea/hypopnea index (AHI) and minimum oxygen saturation (SaO_2), treatment with FA led to a marked reduction in AHI and an increase in minimum SaO_2, suggesting its efficacy in mitigating sleep-disordered breathing symptoms [26, 27]. These improvements were observed when comparing pre-treatment and post-treatment results within the treated group, without comparison to a control group.

Another systematic review focused on evaluating the changes in the AHI following treatment with FA reported contrasting findings. This review incorporated findings from twelve different studies, each reporting a reduction in AHI levels after the use of FA. This suggests a potential positive impact of the appliance on improving airway function and reducing the severity of sleep-disordered breathing symptoms in the short term. However, despite these promising results, the review underscored a critical issue: the substantial heterogeneity present in the pooled data. This variability, stemming from differences in study design, patient demographics, and treatment protocols, poses a significant challenge in drawing conclusive judgement about the overall effectiveness of the appliance across diverse patient groups. In other words, the inconsistency in findings prevents a definitive assessment of the appliance's efficacy. Additionally, the review highlighted a crucial gap in the current literature concerning the long-term stability of treatment outcomes. The enduring effects of FA treatment remain unvalidated, which signifies a pressing need for further longitudinal studies. These studies should aim to evaluate the persistence of the observed benefits over an extended period; exploring not only the immediate improvements in AHI but also the durability of these effects and the potential risk of relapse. Such comprehensive research is essential to provide a more robust understanding of the appliance's long-term impact and to guide clinical decision-making effectively [28, 29].

2.1.3 Optimal timing for the functional appliance

It has previously been demonstrated that the effectiveness of FA of mandibular growth deficiencies strongly depends on the biological responsiveness of the condylar cartilage, which is influenced by the growth rate of the mandible [30, 31]. Timely intervention, ideally 1–2 years before pubertal growth peak, and including the pubertal growth spurt in the treatment period, is considered as a key factor in achieving clinically significant supplementary mandibular growth with FA treatment [32, 33].

2.2 Class II with vertical maxillary excess phenotype

2.2.1 Mechanism of headgears for maxillary vertical growth inhibition

Originally, headgear treatment aims to restrict the forward growth of the maxilla, leading to concerns among some clinicians about its potential negative impact on the airway. Despite

ongoing controversies, when headgear therapy was used in combination with activator which is one of Class II functional appliances, it demonstrated the potential to increase oropharyngeal airway dimensions, specifically in patients with skeletal Class II malocclusion characterized by maxillary excess and mandibular deficiency. This treatment can enhance the smallest distance between the tongue and the posterior pharyngeal wall, as well as the overall pharyngeal area [34].

While there is limited research on the headgear treatment and OSA symptom, considering the adverse effects of vertical maxillary excess, which can result in clockwise mandibular rotation and subsequent narrowing of the oropharyngeal airway, the use of vertical pull headgear may offer superior benefits. By inhibiting vertical maxillary excess, vertical pull headgear can help prevent increased facial divergence and improve oropharyngeal patency. Targeted headgear therapies can play a crucial role in managing airway dimensions and improving breathing function in patients with specific craniofacial abnormalities. Further research is, however, needed to corroborate these benefits and establish standardized treatment protocols.

2.2.2 Optimal timing to control vertical overgrowth of the maxilla using high-pull headgears

The vertical growth pattern of the maxilla exhibits two growth spurts. The first period is from ages 10 to 12, during which the vertical growth of the maxilla occurs through sutural growth at the circummaxillary suture. The second period primarily involves the vertical growth of the alveolar bone. Therefore, the use of high-pull headgear to control vertical overgrowth of the maxilla should target the period between ages 10 and 12. Vertical growth of the maxilla is most pronounced in the posterior height of the face between the ages of 7 and 15, with an average increase of 29.2% in boys and 22.4% in girls. This vertical growth is relatively greater than the sagittal and transverse growth. Additionally, because the vertical growth period is prolonged compared to other dimensions, determining the proper timing for intervention is challenging. Nonetheless, headgear for suppressing vertical growth must be worn for a longer duration, including the growth spurt period of 10 to 12 years, compared to other orthopedic treatments for growth modification [35].

2.2.3 Entire maxillary arch intrusion using TAD supported appliances for vertical control

Total arch intrusion using temporary anchorage devices (TADs) could be an effective strategy for managing Class II with vertical maxillary excess. By intruding the entire maxillary arch, this technique reduces vertical facial height and brings the mandible forward in a counterclockwise rotation [36, 37], which not only enhances facial harmony but also increases pharyngeal airway space, thereby improving breathing function. Given that children have more malleable bones, the use of TADs in infrazygomatic region or in palatal locations, along with connecting orthodontic appliances, is often recommended to achieve effective total arch intrusion. Achieving proper nasal breathing and tongue function is also crucial in preventing further vertical growth. Mouth breathing and abnormal tongue habit can exacerbate vertical facial growth by altering the posture of the tongue and the position of the mandible. Encouraging nasal breathing helps to maintain a more favorable growth pattern, supports proper jaw positioning, and contributes to overall airway health.

2.3 Nasomaxillary complex constriction phenotype

2.3.1 Mechanism of maxillary expansion to open the upper airway

The maxillary bones contribute to approximately 50% of the anatomic structure of the nasal cavity. Consequently, treatment methods that modify the morphology of the maxillary dental arch can significantly impact the shape and function of the nasal cavity [38]. In cases characterized by a craniofacial phenotype with a constricted nasal cavity, narrow nasal floor and maxillary constriction are one of the most common craniofacial phenotypes, and a treatment approach involving skeletal expansion becomes necessary. The nasal cavity accounts for nearly two-thirds of airway resistance due to its upper airway dimensions. Research indicates that rapid palatal expansion (RPE) laterally widens the external walls of the nasal cavity, decreases the height of the palatal vault, and straightens the nasal septum. This restructuring increases nasal volume, reduces nasal resistance, enhances nasal airflow, and promotes better breathing [39]. By lowering airway resistance, RPE diminishes negative pressure during ventilation, thus showing promising outcomes in managing pediatric OSA. Additionally, the expanded palatal space may support improved forward and upward tongue positioning, potentially enhancing airway dimension in the oropharynx indirectly [40]. The multifaceted benefits of nasomaxillary expansion include addressing nasal cavity geometry, improving breathing, and managing sleep-disordered breathing in pediatric patients, while also positively affecting related anatomical structures.

2.3.2 Functional improvement from the nasomaxillary expansion

RPE appears to be an effective treatment for pediatric OSA in children. A study involving 215 non-syndromic children aged 0 to 12 years, diagnosed with OSA by polysomnography AHI before and after the intervention, who underwent rapid maxillary expansion (RME), indicated a decrease in AHI after maxillary expansion [41]. Additionally, this and other studies suggest that the decline in AHI is maintained, as indicated by follow-up tests ranging from 3 months to 14 years [41–43]. Yoon *et al.* [42] demonstrated improvements in both nasal obstruction symptom scores and objective measures of the internal nasal valve after RPE treatment. This suggests the potential for considering RPE in managing resistant pediatric nasal airways [42]. A retrospective study by Yoon *et al.* [44] was the first clinical study to evaluate the effectiveness of RPE in reducing palatine tonsil and adenoid volume in children. Sixty children with an average age of 8 years were divided into a study group treated with RPE and a control group that received no treatment. At the end of therapy, patients treated with RPE showed a statistically significant reduction in the volume of tonsils and adenoids compared to the control group,

which showed no improvement [44].

Research showed that TAD-supported maxillary skeletal expansion (MARPE) is more effective than traditional RPE, both subjectively and objectively, for improving nasal breathing and SDB. Veli *et al*. [38] found a significant mean difference in nasal airflow and reduced nasal resistance favoring the mini-implant assisted RPE (a form of MARPE) group. In this study, 30 patients, approximately 10 years old, were assessed using rhinomanometric measurements at the Otolaryngology to evaluate nasal airflow and nasal airway resistance. Rhinomanometry, the gold-standard test for assessing nasal airway patency, estimates nasal resistance through a variation of Ohm's law based on airflow and pressure differences [39]. The results indicated that the MARPE group exhibited significantly higher nasal mean flow and lower nasal mean airway resistance after maxillary expansion compared to the RPE group. Another study of 26 non-obese OSA patients (mean age 13.6 years) treated with nasomaxillary skeletal expansion showed significant increases in all upper airway compartments, except the glossopharyngeal airway, as measured by CBCT images [45]. In the same study, a home sleep test and modified pediatric sleep questionnaire revealed notable improvements in AHI, oxygen desaturation index, lowest oxygen saturation, flow limitation, snoring, and modified pediatric sleep questionnaires scores [45]. These findings underscore the effectiveness of nasomaxillary skeletal expansion in improving airway dimensions and sleep-related symptoms in pediatric OSA patients.

2.3.3 Optimal timing for nasomaxillary expansion

The success of nasomaxillary skeletal expansion depends on the stage of midpalatal sutural obliteration. To achieve maximum functional improvement with nasomaxillary expansion, conventional RPE is considered effective below the age of 15. Beyond this age, the midpalatal suture and adjacent articulations begin to fuse and become more rigid, leading to higher resistance to expansion forces [46, 47]. Recently, earlier application of RPE has been recommended for children with OSA under the age of 5–6 years when serious nasal obstruction is associated with OSA symptoms. According to Gorikapudi *et al*. [48], considering nasomaxillary expansion, as soon as symptoms appear, could be important for children with OSA. Huang and Guilleminault also insisted that orthodontists should consider nasal expansion in young children with OSA as early as possible to establish nasal breathing early and prevent irreversible changes in craniofacial growth [49]. For children aged 6–9 years, the nasomaxillary complex continues to develop, and the nasal cavity enlarges, making RPE a suitable orthodontic strategy. In middle childhood, aged 10–11 years, the use of a TAD with RPE or a MARPE appliance should be considered, particularly when there are severe transverse discrepancies, early skeletal maturation, severe SDB and OSA symptoms, or recurrent/residual OSA. TADs or mini-implants in the maxilla can secure an RPE appliance, allowing direct forces to be applied to the nasomaxillary complex.

2.4 Class III with retrognathic maxilla phenotypes

2.4.1 Mechanism of face mask to open the upper airway with maxillary protraction

Maxillary protraction is an effective solution for pediatric OSA, particularly in growing skeletal Class III patients with maxillary retrusion. Since maxillary retrusion is frequently associated with maxillary constriction, it is common to use a facemask in combination with RPE [50]. The skeletal modifications induced by a facemask include stimulating the forward and downward growth of the maxilla, direct counterclockwise rotation of the palatal plane, and indirect clockwise rotation of the mandible [51]. These changes are attributed to cellular activity in the circummaxillary sutures and the maxillary tubercle. Such orthopedic effects can lead to the anterior displacement of posterior nasal spine (PNS), which in turn may move the soft palate forward, increasing the post-palatal airway dimension. Additionally, the anterior repositioning of the maxilla results in significant improvements in the nasopharyngeal airway dimensions, including its thickness and area [52–55]. However, the indirect effect of the clockwise rotation of the mandible induced by facemasks might limit the position of the tongue [56]. In this context, Alhamwi *et al.* [54] found that the position of the hyoid did not significantly change during facemask therapy. Similarly, Balos *et al.* [57] concluded that there was no significant difference between the treatment and control groups regarding hyoid position. Consequently, there was less change in McNamara's lower pharynx dimension, which is highly associated with the width of the glossopharynx [57]. To minimize the indirect effect of the clockwise rotation of the mandible, bone-anchored intermaxillary traction may be more beneficial than the conventional facemask for pharyngeal airway development. This approach can modulate the mandibular shape and growth direction in a counterclockwise manner while achieving maxillary protraction [16, 53, 58, 59]. Studies pertaining to the exclusive utilization of facemasks and their functional implications were limited. Quo *et al*. [60] reported a notable decrease in AHI subsequent to early maxillary protraction treatment in patients with skeletal Class III malocclusions and OSA.

2.4.2 Optimal timing for maxillary protraction

Optimal timing for maxillary protraction has been a subject of research, with many reports comparing the sagittal skeletal response of Class III with retrognathic maxilla patients in the primary, mixed, and late mixed dentition phases [61–63]. The consensus from these studies indicates that early treatment is the most effective, therefore protraction treatment is recommended as soon as the diagnosis is made and patient cooperation allows [63]. Since Class III malocclusion tends to worsen during adolescent growth, early interception is preferable, ideally during the primary dentition stage [64]. Early treatment can harness the high biological responsiveness of the sutures and maximize skeletal outcomes, preventing the progression of the malocclusion and promoting optimal craniofacial development. This proactive approach can lead to more stable and favorable long-term results, reducing the complexity and duration of future orthodontic interventions.

The traditional treatment has been to initiate maxillary protraction using tooth borne palatal expander and a facemask during early mixed dentition stage, to achieve skeletal outcomes as the sutures' responsiveness to treatment diminishes with age. Conversely, with skeletal anchorage, treatment initiation occurs at a later age, around 10 years old, taking advantage of bone characteristics for easier placement and enhanced stability [60, 65]. For children aged 6–9, a combination of facemask and RPE therapy is typically applied to address Class III with retrognathic maxilla. However, in cases where the patient is skeletally matured early, exhibits significant skeletal discrepancy, has severe sleep-disordered breathing symptoms, or requires vertical control, a mini screw-assisted palatal expander may be used. This approach allows for more precise and effective skeletal adjustments, thereby addressing the complex needs of these patients. The use of TADs enhances the stability and effectiveness of skeletal expansion, ensuring better control over the vertical dimension and facilitating the correction of more severe discrepancies. At ages 10–11 years, the nasomaxillary complex's growth direction shifts from horizontal to vertical. MARPE combined with facemask therapy is more effective than RPE for horizontal development during this stage. Between ages 12–15 years, bone-anchored mini-implants prove more effective for maximizing orthopedic changes, as skeletal structures are mostly developed and past peak growth potential [16, 44]. Strategies may include midface advancement with MARPE and facemask therapy. Additionally, mini-plates can be used to focus force on skeletal components rather than dentition.

2.5 Bimaxillary deficiency phenotypes

Bimaxillary deficiency phenotypes are common for SDB but present significant challenges in managing the growth of both the maxilla and mandible. Some clinicians focus on maxillary transverse and anteroposterior (AP) development, using palatal expander with reverse pull headgear, then flaring the upper incisors to unlock the mandibular growth, allowing mandible to grow forward naturally. This approach emphasizes achieving functional restoration, including proper posture and nasal breathing, so that the mandible can catch up in growth. Other clinicians attempt to develop both the maxilla and mandible during early growth modifications, typically focusing first on maxillary growth before addressing the mandible. However, there is no consensus on the best approach for successfully growing both the maxilla and mandible. Children with bimaxillary deficiency are considered poor responders to orthopedic or functional appliances aimed at stimulating facial growth. In such cases, the timing of intervention and the type of appliance should be determined based on the skeletal target and the area of respiratory obstruction, according to the differential growth theory. Maxillary protraction is optimally initiated during the prepubertal period in Class III patients with retrognathic maxilla phenotypes, as soon as the diagnosis is confirmed and patient cooperation is achievable [63]. For the forward growth of the mandible, timely intervention ideally 1–2 years before the pubertal growth peak, and including the pubertal peak in the treatment period is necessary to achieve clinically significant supplementary mandibular growth with FA treatment [32]. In patients with severe bimaxillary deficiency characterized by a hyperdivergent pattern and weak musculature, maxillomandibular advancement is the sole intervention to correct skeletal patterns and manage obstructive sleep apnea in older adolescents. Maxillary advancement pulls forward the velum and velopharyngeal muscles, increases nasopharyngeal and hypopharyngeal spaces, and reduces nasal resistance by widening the alar base and mandibular advancement moves the tongue and suprahyoid muscles forward. These changes alleviate pharyngeal obstruction, enhancing upper airway dimensions both transversely and sagittally at multiple levels, and reducing airway collapsibility, thereby improving functional performance [66]. The published literature has little to say on the appropriate timing of various surgical procedures in growing patients or the effects of surgery on postoperative skeletal growth [67–69]. Normal facial growth demonstrates that maxillary growth completes earlier than mandibular growth, and after peak height velocity, the growth potential of the maxilla is minimal. In pediatric patients with refractory OSA, earlier consideration of maxillomandibular advancement surgery may be warranted, however, its application within this population remains controversial. Consequently, surgery is reserved for meticulously selected, refractory cases, and a comprehensive multidisciplinary evaluation is imperative prior to undertaking surgery [69].

2.6 Craniofacial syndromes

Children with craniofacial syndromes face a higher risk of SDB, primarily OSA, due to various anatomical and functional factors. In Down syndrome, OSA is often caused by adenotonsillar hypertrophy, midface hypoplasia, and a small mandible, resulting in airway narrowing. Craniosynostosis heightens OSA risk through midface hypoplasia and may induce central apneas from pressure on respiratory centers. Cleft lip and palate reduce pharyngeal airway size and affect oropharyngeal musculature, impacting speech and swallowing. These conditions require customized orthodontic interventions to effectively manage airway patency [70].

3. Conclusions

Given the multifactorial nature of pediatric OSA etiology, and the distinct differences from adult OSA, it is imperative for orthodontists to recognize the craniofacial features associated with various pathophysiologic OSA phenotypes that predominantly affect upper airway collapsibility. Literature reviews highlight that through growth modification treatment, controlling in the nasomaxillary complex and mandible's anteroposterior, vertical, and transverse dimensions can influence skeletal growth and promote upper airway development. This approach aims to create an environment conducive for the functional matrix to optimize normal breathing and craniofacial skeletal growth. However, the effectiveness of craniofacial growth modification is not guaranteed for all patients due to several factors. Genetic and epigenetic influences, along with environmental factors, lead to high variability, complicating precise predictions. Furthermore, the lack of robust studies, including untreated controls and prospective long-term data, limits the

evidence needed to distinguish appliance effects from natural growth. Additionally, there is scant high-quality evidence supporting functional respiratory improvements beyond airway size changes from craniofacial interventions.

To address these challenges, we categorize the craniofacial phenotype of OSA into main subtypes based on the etiology of skeletal deformity: Class II with retrognathic mandible, Class II with vertical maxillary excess, constricted naso-maxilla, Class III with retrognathic maxilla, and bimaxillary deficiency and proposed treatment options. Timely skeletal target interventions, considering different growth theories, are necessary for maximized skeletal and functional growth potential in a favorable direction, significantly enhancing the sustainability of treatment success. When structural improvement occurs through growth modification, it is essential to evaluate whether functional improvement follows. Interdisciplinary communication among pediatric sleep physicians is required to develop comprehensive, patient-centered treatments for the upper airway development. Additionally, combining growth modification with adenotonsillectomy, medication, or weight and dietary control, as appropriate, is expected to maximize the effectiveness of the treatment.

4. Key takeaways

1. Pediatric OSA is affected by the differential growth of cranial structures, necessitating the correction of skeletal restrictions to prevent airway collapsibility.

2. Skeletal modifications should be controlled three-dimensionally, tailored to craniofacial phenotype subtypes based on the etiology of skeletal deformities.

3. Interventions should be timely and aligned with the skeletal growth peaks relevant to the patient's age.

4. An interdisciplinary approach is crucial, involving collaboration with pediatric sleep specialists for non-craniofacial phenotypes, integrating treatments like adenotonsillectomy, medication, weight control, or continuous positive airway pressure.

5. The lack of robust studies and high-quality evidence calls for caution when assessing appliance effects, distinguishing their effects from natural growth, and evaluating respiratory improvements beyond mere airway size changes from craniofacial interventions.

ABBREVIATIONS

OSA, Obstructive Sleep Apnea; SDB, Sleep-Disordered Breathing; FA, Functional Appliance; MCA, Minimum Cross-sectional Area; RPE, Rapid Palatal Expansion; AHI, Apnea/Hypopnea Index; SaO_2, Minimum Oxygen Saturation; CBCT, cone-beam computed tomography; MARPE, mini-implant assisted RPE; TADs, temporary anchorage devices; PSG, polysomnography; AP, anteroposterior; RME, rapid maxillary expansion; ENT, Ear, Nose, and Throat; PNS, posterior nasal spine.

AVAILABILITY OF DATA AND MATERIALS

The data that support the findings of this study are available from the corresponding author, upon reasonable request.

AUTHOR CONTRIBUTIONS

KAK—conceptualization; writing. HJ—investigation; writing-original draft preparation. SJK and AY—writing-review and editing. All authors have read and agreed to the published version of the manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This review article does not involve any original clinical data or patient cases; therefore, ethics approval and consent to participate are not applicable.

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