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The Craniofacial–Occlusal–Airway Triad in Pediatric Sleep-Disordered Breathing: *A Narrative Review*

Amit Patel[1], Divya Singh[2*], Natasha Gambhir[3], Priyanka Sachdeva[4], Subhashree Sahoo[5], Avantika Tuli[6]

[1](Undergraduate Student, Santosh Dental College, Ghaziabad, Uttar Pradesh, India)
[2](Reader, Department of Pediatric and Preventive Dentistry, Santosh Dental College, Ghaziabad, Uttar Pradesh, India)
[3](Professor and Head of Department, Department of Pediatric and Preventive Dentistry, Santosh Dental College, Ghaziabad, Uttar Pradesh, India)
[4](Reader, Department of Pediatric and Preventive Dentistry, Santosh Dental College, Ghaziabad, Uttar Pradesh, India)
[5](Lecturer, Department of Pediatric and Preventive Dentistry, Santosh Dental College, Ghaziabad, Uttar Pradesh, India)
[6](Professor, Department of Pediatric and Preventive Dentistry, Santosh Dental College, Ghaziabad, Uttar Pradesh, India)
***Corresponding Author:** Dr. Divya Singh, Reader, Department of Pediatric and Preventive Dentistry, Santosh Dental College, Ghaziabad, Uttar Pradesh, India*

Abstract
***Background:** Sleep-disordered breathing (SDB) in children ranges from primary snoring to obstructive sleep apnea (OSA) and is increasingly understood to arise from an interconnected craniofacial-occlusal-airway triad rather than any single anatomical abnormality.*
***Objective:** This narrative review synthesizes evidence on the craniofacial, occlusal, and upper airway components of pediatric SDB, their interrelationships, and their implications for interdisciplinary screening, diagnosis, and management.*
***Methods:** A structured search of PubMed/MEDLINE, ScienceDirect, Scopus, and Google Scholar was conducted for English-language literature addressing these domains.*
***Results:** Cross-sectional and case-control evidence consistently links craniofacial cephalometric features — including mandibular retrognathia and vertical growth patterns — to pediatric OSA, though GRADE-based appraisal indicates that certainty regarding causal strength remains low to moderate. Occlusal findings, including transverse maxillary constriction and Class II malocclusion, are similarly associated with mouth breathing and SDB, with age and hereditary factors modifying their expression. Airway obstruction occurs across multiple anatomical levels, and polysomnography remains the diagnostic gold standard, though validated screening tools deployable in the dental setting identify substantial proportions of at-risk children. Evidence for craniofacial and occlusal interventions — rapid maxillary expansion, mandibular advancement appliances, orofacial myofunctional therapy, and lingual frenectomy — varies considerably in maturity, with myofunctional therapy best supported and lingual frenectomy least.*
***Conclusion:** These findings support the craniofacial-occlusal-airway triad as a clinically useful, hypothesis-informed framework for integrated screening and management, while underscoring the need for standardized diagnostic criteria and higher-quality controlled trials to clarify causal relationships and optimize interdisciplinary care.*
***Key Word:** pediatric sleep-disordered breathing; obstructive sleep apnea; craniofacial morphology; malocclusion; upper airway; rapid maxillary expansion; myofunctional therapy; orthodontics*

I. Introduction

Pediatric sleep-disordered breathing (SDB) encompasses a clinical spectrum of respiratory issues in children that occur during sleep. It extends from habitual primary snoring to frank obstructive sleep apnea (OSA), characterized by recurrent partial or complete collapse of the upper airway during sleep with sleep fragmentation. Pediatric OSA is defined as a sleep-related breathing disorder marked by recurrent partial or complete upper airway obstruction leading to disrupted ventilation and fragmented sleep, arising from anatomical and neuromuscular factors, with inflammatory processes also implicated, and is recognized as clinically and polysomnographically distinct from adult OSA (1,2).

Population-based estimates place the prevalence of pediatric OSA at roughly 1–5% among healthy children, with wide variation across populations and diagnostic thresholds, and substantially higher rates reported in children with additional risk factors such as obesity, craniofacial anomalies, or Down syndrome (1).

Pediatric OSA is multifactorial. Adenotonsillar hypertrophy is the most common proximate cause, especially in children aged roughly 2–8 years when lymphoid tissue is relatively enlarged (1,2). Craniofacially, retrognathic and hyperdivergent facial patterns, a steep mandibular plane angle, and transverse maxillary constriction are reported with varying consistency in affected children (3-5,7), with severe forms seen in craniofacial syndromes such as Treacher Collins, Pierre Robin sequence, Crouzon, Apert, and achondroplasia (8,9). Occlusally, chronic mouth breathing and altered tongue/perioral posture are associated with a narrow, high-arched palate and Class II malocclusion, with these functional abnormalities proposed as upstream contributors to craniofacial dysmorphism rather than mere consequences of dysmorphism (10-13). Upper airway factors include ankyloglossia, which restricts tongue posture and mobility and has been identified as an independent risk factor for sleep-disordered breathing (32-34).

Several observations link craniofacial, occlusal, and airway abnormality into a self-reinforcing cycle. Airway obstruction promotes compensatory mouth breathing; mouth breathing alters resting tongue posture and removes the expansile pressure normally exerted against the palate during growth; and the resulting narrow, high-arched maxilla — the occlusal signature of this cycle — further narrows the nasal floor and compromises airway patency, reinforcing the original obstruction (10,11,13). Guilleminault and Huang have described this as a specific developmental sequence running from early oral-facial dysfunction to craniofacial dysmorphism and, ultimately, to the onset of pediatric OSA (6). This cycle underlies the concept of a "triad" of craniofacial, occlusal, and airway abnormality, in which each component functions simultaneously as a cause, a consequence, and a diagnostic marker of the others — meaning a finding in any one domain should prompt clinical screening for abnormality in the remaining two.

Because orthodontists and pediatric dentists routinely examine the dentition, occlusion, and craniofacial skeleton of growing children, they are well positioned to recognize early signs of SDB risk, often before a formal sleep medicine referral would otherwise occur (14-17).

Thus, this narrative review aims to describe the craniofacial, occlusal, and upper airway components of pediatric sleep-disordered breathing individually and as an interdependent triad; summarize validated interdisciplinary screening approaches; and critically appraise the evidence base for craniofacial and occlusal interventions — including rapid maxillary expansion, mandibular advancement appliances, orofacial myofunctional therapy, and lingual frenectomy.

II. Materials and Methods

This narrative review was conducted through a structured search of PubMed/MEDLINE, ScienceDirect, Scopus, and Google Scholar for English-language articles addressing the craniofacial, occlusal, and upper airway dimensions of pediatric sleep-disordered breathing, published without a fixed lower date limit but with an emphasis on literature from the past two decades.

Search terms included combinations of "pediatric obstructive sleep apnea," "craniofacial morphology," "malocclusion," "mouth breathing," "rapid maxillary expansion," "mandibular advancement appliance," "myofunctional therapy," "ankyloglossia," "adenotonsillectomy," "cone-beam computed tomography," "polysomnography," "drug-induced sleep endoscopy," "candidate genes," and "screening."

Systematic reviews, meta-analyses, randomized and non-randomized controlled trials, and prospective and retrospective cohort studies were prioritized for inclusion. Case reports, non-peer-reviewed sources, and articles not available in English were generally excluded, except where they provided uniquely relevant mechanistic or historical context.

As a narrative rather than systematic review, no formal risk-of-bias scoring or PRISMA-based study selection protocol was applied; instead, the aim was a qualitative, integrative synthesis of the most methodologically robust and frequently cited evidence addressing each component of the triad.

III. Result

Across the literature reviewed, several consistent patterns emerge. First, cross-sectional and case-control evidence robustly demonstrates that children with OSA differ from unaffected peers on multiple cephalometric parameters — including mandibular plane angle, retrognathic mandible, and vertical growth direction — though GRADE-based appraisal indicates that certainty regarding the causal strength of these associations remains low to moderate (3-5). Second, occlusal findings including increased overjet, anterior open bite, posterior crossbite, and transverse maxillary constriction are consistently reported at higher frequency in mouth-breathing and SDB-affected children, with both age-related and hereditary factors modifying their expression (10-13,40,41). Third, airway obstruction in pediatric SDB occurs across multiple anatomical levels, and CBCT imaging confirms that craniofacial skeletal pattern correlates with pharyngeal airway dimension, though specific orthodontic

interventions differ in their measurable three-dimensional airway effect (27,35,36). Fourth, validated screening tools deployable in the dental setting identify substantial proportions of at-risk children within orthodontic populations, supporting a plausible and increasingly formalized role for dental clinicians in the diagnostic pathway (14-17,37). Fifth, intervention-level evidence varies considerably in maturity: orofacial myofunctional therapy carries the most consistent evidence base, rapid maxillary expansion and mandibular advancement appliances occupy an intermediate position supported by a growing but still limited volume of controlled data, and lingual frenectomy remains the least mature intervention reviewed (20-26,28,32-34). Taken together, these findings support the craniofacial-occlusal-airway triad as a clinically coherent framework, while underscoring that the strength of evidence differs meaningfully across its components and across the interventions used to address them.

IV. Discussion

Building on the patterns summarized above, this discussion examines each component of the craniofacial-occlusal-airway triad in turn — the craniofacial, occlusal, and airway findings associated with pediatric SDB — before addressing how these components interconnect, how they can be screened for and diagnosed in the dental setting, and what the evidence supports regarding their clinical management. The discussion closes by identifying gaps in the current literature and priorities for future research.

The Craniofacial Component: *Anatomical and Non-Anatomical Factors*

Anatomical Factors

Craniofacial growth and pharyngeal airway dimensions are anatomically and developmentally linked, since the maxilla, mandible, and hyoid bone form the bony boundaries that determine the cross-sectional space available for airflow through the nasal and oropharyngeal airway (4,7).

Systematic reviews and meta-analyses of cephalometric studies in children with polysomnography-confirmed OSA have reported associations with an increased mandibular plane angle, retrognathic mandible, and a tendency toward vertical (hyperdivergent) growth patterns relative to unaffected peers (4,5).

Katyal et al. in their systematic review and meta-analysis found significant differences between children with SDB and controls for numerous cephalometric characteristics including reduced posterior airway space and altered hyoid position (4). Likewise, Flores-Mir et al. found significant differences in mandibular plane angle, SNB, and ANB values in children with OSA syndrome compared with unaffected controls, and described a craniofacial phenotype of retrusive chin, steep mandibular plane, and vertical growth direction associated with pediatric OSA (5).

Craniofacial syndromes extend this anatomical evidence to its most severe form: conditions involving midface hypoplasia, mandibular hypoplasia, or craniosynostosis — including Treacher Collins syndrome, Pierre Robin sequence, Crouzon and Apert syndromes, and achondroplasia — carry a markedly elevated prevalence of OSA, illustrating in an extreme form the principle that skeletal deficiency in the craniofacial complex predisposes to airway compromise even in the absence of soft-tissue lymphoid hypertrophy (8,9). Population-based case-control data similarly show that children with craniofacial anomalies, even outside of defined syndromes, carry substantially elevated odds of a diagnosed sleep apnea compared with unaffected peers (9).

Non-Anatomical Factors

Skeletal morphology alone does not fully account for the craniofacial contribution to pediatric SDB. A more recent systematic review and meta-analysis by Fagundes et al., applying GRADE certainty assessment, concluded that the overall certainty of evidence linking craniofacial features to pediatric OSA remained very low to moderate, such that neither a definitive association nor its absence could be firmly established from the available literature (3).

Part of this uncertainty likely reflects confounding by adenotonsillar size, which independently influences head posture and several standard cephalometric measurements and is rarely fully controlled for across the pooled studies. Beyond this methodological confound, neuromuscular tone of the pharyngeal dilator muscles and inflammatory processes affecting the upper airway are also recognized contributors to airway patency that operate independently of static skeletal measurements (1,2), suggesting that non-skeletal factors interact with anatomical predisposition rather than acting as independent, sufficient causes.

Beyond structural anatomy and airway physiology, two further dimensions shape the craniofacial contribution to pediatric SDB. First, craniofacial growth is not static: the direction and rate of facial growth across childhood, rather than skeletal position captured at a single point in time, shapes airway dimensions as children mature, and the relative weight of individual etiologic factors shifts with age — adenotonsillar hypertrophy predominates in early childhood, whereas skeletal and metabolic factors such as obesity become more prominent later (1).

Second, a hereditary component is increasingly recognized: the craniofacial features most strongly associated with sleep-related breathing disorders, including craniosynostosis, retrognathia, micrognathia, and midface or maxillary hypoplasia, show high heritability, and a proposed panel of candidate genes involved in orofacial

development has been suggested to extend this genetic contribution beyond well-characterized craniofacial syndromes to a broader group of non-syndromic children with milder craniofacial dysmorphism (38).

The Occlusal Component: ***Dental and Skeletal Factors***

Dental Factors

At the level of the dentition itself, the findings most consistently reported in mouth-breathing and SDB-affected children are increased overjet, anterior open bite, posterior crossbite, and Class II, division 1 malocclusion, all occurring at higher frequency than in nasal breathers (10-13). A systematic review of observational studies concluded that Angle Class II, division 1 malocclusion occurs at a consistently higher prevalence in mouth-breathing children than Class I malocclusion (12).

Skeletal Factors

At the skeletal level, transverse maxillary constriction — the narrow, high-arched palate frequently noted on clinical examination — is the most consistently reported finding (10-13). A systematic review and meta-analysis by Zhao et al., pooling ten studies, found that mouth-breathing children displayed significantly reduced SNA and SNB values, an increased ANB angle suggesting maxillomandibular retrusion, and increased vertical cephalometric measurements (SN-PP, SN-OP, PP-MP, SNGoGn) relative to nasal breathers, consistent with a long-face, vertically-growing pattern (10). A more recent systematic narrative review corroborated these findings, while cautioning that considerable heterogeneity in diagnostic criteria for mouth breathing limits the strength of causal inference (11).

These occlusal findings carry clinical value beyond their own correction: a narrow palate or Class II malocclusion identified on routine dental examination can serve as an early marker prompting screening for underlying SDB.

Two further dimensions shape the occlusal component of pediatric SDB. First, the significance of occlusal findings is not fixed across childhood: a cross-sectional study of 177 children aged six to 12 years found that SDB was significantly associated with developing malocclusion, with gender and tonsillar enlargement acting as significant modifiers of this association, indicating that the occlusal signature of SDB should be interpreted alongside age, dentition stage, and concurrent adenotonsillar status rather than as a fixed, age-independent marker (40). Second, malocclusion in this population is not purely an acquired, functional finding: a narrative review of the genetics of pediatric sleep disorders reported silent PHOX2B genotype mutations in 32% of children with Class III malocclusion, compared with none of the controls, indicating that a hereditary component can also underlie the craniofacial dysmorphology seen alongside SDB, independent of functional or environmental influences (41).

The Airway Component: ***From Anatomy to Polysomnographic Confirmation***

Pediatric SDB arises from obstruction at multiple anatomical levels of the upper airway (1,2). Nasal obstruction — from allergic rhinitis, adenoid hypertrophy, or maxillary hypoplasia — increases upstream resistance and predisposes to mouth breathing. At the retropalatal level, adenotonsillar hypertrophy is the most common site of mechanical obstruction in young children (1). More posteriorly, the retroglossal airway — bounded by the tongue base, mandible, and hyoid — represents a common site of dynamic narrowing during sleep. Airway patency also depends on pharyngeal dilator muscle tone, which normally counteracts negative intraluminal pressure; the physiological drop in this tone at sleep onset is why marginally narrow airways can collapse once asleep (2).

Pediatric OSA is defined as recurrent partial or complete upper airway obstruction during sleep that disrupts ventilation and sleep architecture, arising from anatomical and neuromuscular factors, with inflammatory processes also implicated, and is clinically and polysomnographically distinct from adult OSA (1,2). Polysomnography (PSG) remains the diagnostic gold standard, with pediatric OSA defined using an obstructive apnea-hypopnea index (AHI) ≥1 event/hour, with severity graded accordingly — a threshold considerably stricter than that applied in adults (1). Because attended PSG is resource-intensive, validated screening questionnaires — notably the 22-item Pediatric Sleep Questionnaire (PSQ), developed by Chervin and colleagues — are widely used as first-line triage despite limited sensitivity and specificity (15-17,37).

CBCT permits three-dimensional volumetric assessment of the pharyngeal airway alongside craniofacial structures (35), and has been used to demonstrate reduced pharyngeal airway volume in children with retrognathic skeletal patterns relative to controls (36). While CBCT characterizes airway anatomy in a static, awake state, drug-induced sleep endoscopy (DISE) allows direct visualization of the airway under sedation approximating natural sleep, identifying the specific site or sites of dynamic collapse — commonly at the velum, oropharynx and lateral pharyngeal walls, tongue base, or epiglottis — and is used particularly in children with persistent OSA after adenotonsillectomy or small tonsils, to guide targeted surgical planning (39).

The Craniofacial–Occlusal–Airway Triad: ***An Integrated Developmental and Clinical Framework***

The evidence reviewed above indicates that craniofacial morphology, occlusal development, and airway function are not independent domains in children with sleep-disordered breathing (SDB), but interact dynamically during growth, with the relative contribution of each component varying by age, phenotype, and additional risk factors. Rather than a linear sequence, the triad is better conceptualized as a network of bidirectional relationships, elaborated below at the level of each pairwise connection.

The craniofacial-airway connection operates through direct anatomical mechanisms. A retrognathic mandible and hyperdivergent (vertical) growth pattern displace the tongue base and hyoid bone posteriorly and inferiorly, narrowing the retroglossal airway space and increasing its collapsibility during sleep, while maxillary hypoplasia reduces nasal cavity volume and raises nasal airway resistance (7) — providing a plausible anatomical basis for the craniofacial-OSA associations described in the Craniofacial Component above.

The occlusal-airway relationship is thought to run in both directions. A transversely deficient maxilla may itself narrow the nasal floor and increase nasal airway resistance, promoting compensatory mouth breathing; conversely, chronic mouth breathing alters the resting posture of the tongue and perioral musculature, removing the tongue's normal expansile pressure against the palate during growth — the proposed mechanism underlying the frequently cited but incompletely proven "mouth-breathing malocclusion" cycle (11,13). The anterior open bite frequently observed in this phenotype follows a related pathway: mouth breathing rotates the mandible posteriorly with excessive eruption of the posterior teeth, while a low, forward tongue posture prevents normal incisor overlap, so that both the dental and skeletal changes described in the Occlusal Component share a common muscular origin rather than arising independently (13). Adenotonsillar hypertrophy adds a further layer to this relationship: tonsillar hypertrophy specifically, more than adenoid hypertrophy, correlates with increased overjet and Class II malocclusion in young children, linking airway-obstructing lymphoid tissue directly to occlusal development (13).

Imaging evidence further illustrates this interconnection at the level of specific interventions: a systematic review found that functional appliances correcting Class II malocclusion measurably increase pharyngeal airway volume, whereas rapid maxillary expansion, despite reliably achieving transverse skeletal correction, is not consistently associated with significant three-dimensional airway change (27) — a dissociation illustrating that correcting one component of the triad does not automatically or proportionately correct another.

Genetic susceptibility may additionally influence the expression of these interacting phenotypes (38).

The strength of evidence, however, is not equivalent across the triad. Associations between craniofacial characteristics and pediatric OSA have been reported repeatedly, but certainty regarding causality remains limited (3). The triad should therefore be regarded as a hypothesis-informed clinical framework that encourages integrated assessment, not as proof that abnormality in one domain inevitably produces abnormality in the other two.

Clinically, this framework supports bidirectional screening: children presenting with occlusal or craniofacial findings suggestive of the triad may warrant targeted questioning about snoring and sleep symptoms, while children diagnosed with SDB may benefit from craniofacial and occlusal assessment — recognizing contributing factors without assuming any single anatomical finding fully explains the disorder.

Interdisciplinary Screening and Diagnosis in the Dental Setting

Because orthodontic and pediatric dental visits are frequent, low-threshold points of contact throughout childhood, several professional bodies have positioned dentistry as a natural setting for SDB screening — a role consistent with the bidirectional assessment the Craniofacial–Occlusal–Airway Triad framework supports (14,17). Recommended screening tools include the 22-item Pediatric Sleep Questionnaire (PSQ), the OSA-18 quality-of-life questionnaire, the Sleep Clinical Record, the Pediatric-Modified STOP-Bang, the Modified Pediatric Epworth Sleepiness Scale (ESS-CHAD) for children aged 6–16 years, and the more recently described Functional Airway Evaluation Screening Tool (FAIREST-6), alongside structured clinical examination for mouth breathing, tonsillar size, retrognathia, and palatal morphology — findings already detailed in the Craniofacial and Occlusal Components (16,17,37).

Cross-sectional studies conducted within orthodontic populations report SDB screening-positive rates considerably higher than general pediatric population estimates, ranging from roughly 7% to nearly 48% depending on the screening instrument and population studied, and have found significant associations between SDB screening status and parent-reported behavioral symptom domains including inattention and hyperactivity (14,15). These findings support incorporating brief, validated airway- and sleep-oriented screening into routine orthodontic assessment, particularly for children presenting with mouth breathing, snoring, or behavioral concerns.

Screening, however, identifies risk rather than confirming diagnosis. A positive screen — whether from questionnaire, clinical examination, or the Craniofacial and Occlusal Component findings — should prompt referral for definitive evaluation, since diagnostic confirmation in children draws on clinical assessment, history, and validated questionnaires, with polysomnography — detailed in the Airway Component — remaining the

diagnostic gold standard (1,42). Recognizing this distinction allows dental and orthodontic clinicians to function as an effective screening gateway into the SDB diagnostic pathway without overstating what screening tools alone can establish.

Management and Therapeutic Interventions in the Triad

Rapid Maxillary Expansion

Rapid maxillary expansion (RME) is an orthopedic-orthodontic procedure that opens the midpalatal suture to achieve skeletal widening of a transversely deficient maxilla, and has been proposed as a treatment for pediatric OSA associated with maxillary constriction on the rationale that widening the maxillary base secondarily enlarges the nasal cavity and reduces nasal airway resistance (7,18). Pirelli et al.'s early prospective study reported apnea-hypopnea index (AHI) normalization following RME in children with narrow maxillae and OSA, a finding frequently cited as foundational evidence for the procedure (18). A systematic review and meta-analysis by Camacho et al. pooling seventeen studies and 314 children found consistent improvement in AHI and lowest oxygen saturation following RME, particularly at follow-up under three years, though the authors emphasized the paucity of randomized controlled data and long-term follow-up (20).

More recent controlled evidence has tempered earlier enthusiasm. A systematic review restricted to controlled, ideally randomized, studies identified only a single RCT directly comparing RME against watchful waiting, which found no statistically significant difference in AHI improvement between the two arms (22). A 2026 update to this review identified three additional RCTs but noted that none included a watchful-waiting or supportive-care control arm, leaving the original null comparison against observation unchanged (21). A more recent RCT directly comparing RME against adenotonsillectomy in children with concurrent adenotonsillar hypertrophy and transverse maxillary deficiency found both approaches produced measurable improvement, informing discussion of RME as a viable alternative or adjunct rather than a universal substitute for surgery (19). Taken together, the RME literature illustrates a broader pattern in this field: an accumulation of favorable uncontrolled and retrospective evidence that has not yet been matched by an equivalent volume of high-quality randomized data isolating the specific contribution of skeletal expansion from spontaneous resolution and growth.

Mandibular Advancement Appliances and Functional Orthopedic Devices

Mandibular advancement appliances (MAAs), including twin-block and monoblock designs, aim to reposture the mandible anteriorly, thereby enlarging the retroglossal and oropharyngeal airway space in children with Class II, hyperdivergent, or retrognathic skeletal patterns (23,24). Villa et al. reported that MAAs represented an alternative and valid treatment option for pediatric OSA, particularly in children without significant adenotonsillar hypertrophy or as an adjunct following incomplete resolution after adenotonsillectomy (23). An umbrella review synthesizing multiple systematic reviews found long-term appliance use (six to twelve months) more effective than short-term protocols across the variety of MAA designs examined, though studies were heterogeneous in appliance type, wear protocol, and outcome measurement (25). A subsequent systematic review and meta-analysis comparing different MAA designs, based on three RCTs and five non-randomized studies, likewise concluded that MAAs represent a promising non-invasive adjunct particularly for children with craniofacial anomalies or Class II malocclusion, while highlighting the continued absence of a standardized appliance protocol (26).

This mechanism is consistent with the CBCT evidence discussed in the Airway Component, where mandibular repositioning — unlike palatal expansion — was associated with a measurable three-dimensional airway effect (27).

Orofacial Myofunctional Therapy

Orofacial myofunctional therapy (OMT) targets the soft-tissue and neuromuscular contributors to the triad — tongue posture, lip seal, and swallowing pattern — through structured exercise protocols directed at the tongue, soft palate, and facial musculature (28). A landmark systematic review and meta-analysis by Camacho et al. found that myofunctional therapy reduced AHI by roughly 50% in adults and 62% in children, alongside improvements in snoring and oxygen saturation, and proposed that isolated OMT or OMT combined with other therapies produced clinically meaningful improvement in mild-to-moderate SDB (28). Guilleminault et al. described myofascial reeducation as playing a critical role in preventing OSA relapse following adenotonsillectomy in children, attributing residual post-surgical OSA partly to persistent abnormal tongue and orofacial muscle function (29). Subsequent controlled cohort work has shown that OMT can measurably increase tongue tone and peak tongue pressure alongside improvement in nocturnal pulse oximetry parameters (30), and a systematic review specific to craniomaxillofacial outcomes reported favorable, if heterogeneous, effects of OMT on craniofacial growth patterns in children with OSA-related conditions (31). Despite this accumulating evidence, Camacho et al. cautioned that the absence of standardized protocols and inconsistent adherence reporting continue to limit clinical translation of OMT as a pediatric-specific therapy (28).

Ankyloglossia and Lingual Frenectomy

Restricted lingual frenulum (ankyloglossia) has been proposed as an underrecognized contributor to the triad, on the premise that a tethered tongue cannot achieve or maintain the palatal resting posture required for optimal maxillary development and cannot be adequately repositioned anteriorly during sleep, thereby predisposing to airway collapse (32,34). Guilleminault et al. described a distinct pediatric SDB phenotype associated with short lingual frenulum, characterized by a higher, narrower palate and greater maxillary hypoplasia compared with children with normal frenulum anatomy (32). Villa et al. subsequently identified short lingual frenulum as an independent risk factor for SDB in school-age children (33). A more recent integrative review examining the ankyloglossia-OSA relationship identified only four studies meeting inclusion criteria, of which two retrospective cohorts supported an association between untreated shortened lingual frenulum at birth and later obstructive sleep apnea, while explicitly calling for prospective polysomnographic data before and after frenectomy in larger cohorts (34). This remains among the least mature evidence bases within the triad and should be interpreted with appropriate caution.

Current Gaps in the Literature

Several gaps limit the strength of conclusions that can currently be drawn about the craniofacial-occlusal-airway triad. Randomized controlled trial evidence remains sparse for the two interventions most directly framed around the triad concept: rapid maxillary expansion has been assessed against watchful waiting in only a single RCT, with no subsequent trial including a supportive-care or observation control arm, and the ankyloglossia-OSA literature comprises only four studies meeting basic inclusion criteria, two of which are retrospective cohorts (21,22,34). Across the intervention literature more broadly, inconsistent AHI thresholds, appliance protocols, and outcome measures prevent direct comparison between studies and preclude meaningful pooled effect estimates (20-22,25,26,28).

Mechanistic gaps are equally notable. Although candidate-gene and heritability data suggest a genetic contribution to craniofacial dysmorphism independent of the functional mouth-breathing cycle, no study has yet examined how genetic susceptibility interacts with environmental and functional risk factors to determine which children with a given craniofacial phenotype go on to develop clinically significant SDB (38,41). Similarly, no identified study has evaluated combined or sequential multimodal management — for example, orofacial myofunctional therapy alongside rapid maxillary expansion, or frenectomy followed by structured myofunctional rehabilitation — despite this reflecting how children with multiple triad risk factors are frequently managed in practice. Finally, the evidence supporting a dental and orthodontic screening role rests on cross-sectional prevalence and association data; no study has yet evaluated whether dental-setting screening measurably changes downstream diagnostic or treatment rates, or improves clinical outcomes, relative to usual care.

V. Future Directions

Addressing these gaps will require several concurrent lines of research. Adequately powered, randomized, and ideally sham- or watchful-waiting-controlled trials are needed for rapid maxillary expansion and lingual frenectomy specifically, given how thin the controlled evidence base remains for both relative to the volume of uncontrolled and retrospective data already available. Standardizing diagnostic and outcome criteria across the craniofacial, occlusal, and airway literatures — common AHI thresholds, consistent cephalometric landmarks, and validated definitions of mouth breathing — would allow future studies to be pooled meaningfully in a way the current literature does not support.

Longer-term follow-up extending into adolescence and adulthood would help distinguish genuine treatment effects from the natural resolution of pediatric SDB with growth, a confound that affects interpretation of nearly every intervention reviewed here. Prospective studies examining the interaction between genetic susceptibility and environmental or functional risk factors would help clarify why children with similar craniofacial phenotypes do not uniformly develop SDB. Trials of combined or staged multimodal management, reflecting actual interdisciplinary practice rather than single-intervention protocols, would better inform real-world treatment sequencing. Finally, pragmatic studies conducted in general pediatric and primary dental care settings — rather than tertiary orthodontic, dental sleep medicine, or otolaryngology centers — are needed to establish whether dental-setting screening translates into measurable improvements in diagnostic timeliness and clinical outcomes.

VI. Limitations

This narrative review carries several limitations inherent to its design and to the underlying literature. First, as a narrative rather than systematic review, study selection was not governed by a formal PRISMA-based protocol or independent risk-of-bias scoring, so the synthesis presented here reflects an integrative reading of the most frequently cited and methodologically robust evidence rather than an exhaustive, reproducible search. Second, the literature addressing the craniofacial, occlusal, and airway components is characterized by substantial

heterogeneity in diagnostic criteria for mouth breathing and SDB, in cephalometric landmark definitions, and in the AHI thresholds used to define treatment success (10-13) — differences that limit direct comparison across the studies reviewed and constrain the strength of any pooled conclusions drawn from them.

Third, the intervention literature reviewed — spanning rapid maxillary expansion, mandibular advancement appliances, orofacial myofunctional therapy, and lingual frenectomy — remains limited by small sample sizes, short follow-up durations, and a continued scarcity of randomized, sham-, or watchful-waiting-controlled trials (20-22,25,26,28). Where such controlled data do exist, effect sizes are often attenuated relative to those reported in uncontrolled case series, raising the possibility that some of the apparent benefit attributed to these interventions reflects the natural tendency of pediatric SDB to improve with growth rather than a specific treatment effect. Fourth, much of the cited evidence originates from tertiary orthodontic, dental sleep medicine, and otolaryngology centers, which may not fully represent presentation, screening uptake, or treatment access in general pediatric or primary dental care populations. Finally, because this review synthesizes cross-sectional and short-term prospective evidence for most of its mechanistic claims, it can describe consistent associations across the craniofacial, occlusal, and airway domains but cannot establish the direction of causality between them (3,11) — a distinction addressed throughout the review but worth restating explicitly as a boundary on what this evidence base can currently support.

VII. Conclusion

The craniofacial, occlusal, and airway components of pediatric sleep-disordered breathing are best understood as an interacting system rather than three independent findings: craniofacial skeletal pattern shapes the anatomical environment of the airway, occlusal development both reflects and modifies that environment through functional adaptation, and the resulting airway physiology in turn influences craniofacial and dental growth across childhood. This triad framework does not imply that any single anatomical finding proves or excludes SDB; rather, it supports using craniofacial and occlusal findings as clinical markers that should prompt targeted screening, and using confirmed SDB status as a prompt for craniofacial and occlusal assessment in turn.

Because orthodontic and pediatric dental visits are frequent and low-threshold relative to sleep medicine referral, dental and orthodontic clinicians are well positioned to contribute to early recognition of at-risk children using validated screening tools alongside routine clinical examination, while referring appropriately for polysomnographic confirmation rather than substituting screening for diagnosis. Among the interventions reviewed, the evidence base is most mature for orofacial myofunctional therapy and least mature for lingual frenectomy, with rapid maxillary expansion and mandibular advancement appliances occupying an intermediate position supported by a growing but still limited volume of controlled trial data. Future research prioritizing randomized, adequately-powered, and longer-follow-up designs — alongside standardized diagnostic and outcome criteria across the craniofacial, occlusal, and airway literatures — will be needed to move this triad framework from a well-supported clinical heuristic toward a validated basis for interdisciplinary management.

Author Contributions

Amit Patel conceived the review topic and structure; Divya Singh performed the literature search and drafted the manuscript; Natasha Gambhir, Priyanka Sachdeva, Subhashree Sahoo, and Avantika Tuli critically revised the manuscript for intellectual content; all authors read, critically reviewed, and approved the final version of the manuscript.

Ethical Statement

This narrative review did not involve human participants, human data, or animal subjects, and did not require institutional ethical approval.

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Conflict of Interest

The authors declare no conflict of interest.

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