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REVIEW ARTICLE

Adenoid Facies and Its Impact on Craniofacial Development in Children: A Systematic Review

Shuaib Kayode Aremu[1,2*]

[1]*Department of Otorhinolaryngology, Afe-Babalola University, Ado-Ekiti, Ekiti State, Nigeria*
[2]*Department of Otorhinolaryngology, Federal Teaching Hospital, Ido-Ekiti, Ekiti State, Nigeria*

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Abstract: ***Background*:** Adenoid hypertrophy is among the most prevalent conditions in paediatric otolaryngology and dentistry, with a well-established causal relationship to altered craniofacial growth patterns collectively described as adenoid facies. Despite its clinical importance, the full spectrum of skeletal, dental, and soft-tissue consequences remains incompletely characterised in the systematic literature.

***Objectives*:** This systematic review aims to synthesise evidence on the anatomical, pathophysiological, clinical, diagnostic, and therapeutic aspects of adenoid facies and its impact on craniofacial development in children aged 3–18 years.

***Methods*:** A comprehensive literature search was conducted in PubMed, MEDLINE, Cochrane Library, Scopus, and EMBASE for articles published between January 2000 and December 2025. Original research articles (observational studies and RCTs) and systematic reviews were separately identified and assessed; systematic reviews were used for contextual and comparative purposes only to avoid double-counting of participants. Quality assessment used the Newcastle-Ottawa Scale (NOS), Cochrane Risk of Bias 2.0 (RoB 2.0), and AMSTAR-2. Quantitative pooling (meta-analysis) was not performed owing to substantial heterogeneity in study design, outcome measures, adenoid grading systems, and reported metrics across included studies.

***Results*:** Seventy-four studies met inclusion criteria. Evidence consistently demonstrates that adenoid hypertrophy-induced nasal obstruction triggers chronic mouth breathing, leading to characteristic craniofacial deformities including increased lower facial height, backward mandibular rotation, high-arched palate, dental malocclusion, and altered soft-tissue posture. Across included studies, reported values for lower anterior facial height/total anterior facial height (LAFH/TAFH) ratios generally exceeded 0.60 (normal: 0.55 ± 0.02), ANB angles ranged 5–8° (normal: 2 ± 2°), and posterior crossbite prevalence ranged from 23% to 56%. Early surgical and orthodontic interventions were associated with measurable improvements in craniofacial morphology when applied before skeletal maturity; however, complete reversal of established skeletal abnormalities was not consistently demonstrated and should not be assumed.

***Conclusions*:** Adenoid facies represent a multidimensional consequence of untreated adenoid hypertrophy with long-lasting implications for dentofacial function and psychosocial well-being. Interdisciplinary management is essential for optimal outcomes. Future research should prioritise prospective registration, standardized outcome reporting, and longer-term follow-up.

Keywords: adenoid hypertrophy, adenoid facies, craniofacial development, mouth breathing, paediatric otolaryngology, dental malocclusion.

**Correspondence should be addressed to Prof. Shuaib Kayode Aremu, MBBS(IL); Certs.LMIH&IEGH(Washington); MScPH(USW); PhDPH(SUSL); FWACS(WA); FACS(USA) Department of Ear Nose and Throat, College of Medicine and Health Sciences, Afe-Babalola University, Ado-Ekiti, Ekiti State, Nigeria;Email: aremusk@abuad.edu.ng | Phone: +2348033583842 | ORCID: https://orcid.org/0000-0002-0165-6867*

1. INTRODUCTION

Adenoid hypertrophy, the pathological enlargement of the pharyngeal tonsil (Luschka tonsil) in the nasopharynx, is one of the most frequently encountered conditions in paediatric medicine. It is estimated to affect approximately 34–40% of school-aged children globally, with peak incidence between 3 and 7 years of age. [1,2] The adenoids constitute the superior component of Waldeyer's lymphatic ring and serve as the first immunological barrier against inhaled antigens. However, their functional hypertrophy during childhood often results in significant nasopharyngeal airway obstruction, with far-reaching consequences for upper airway dynamics, sleep architecture, and craniofacial growth.

The relationship between nasopharyngeal obstruction and facial morphology was first described in the late nineteenth century by Wilhelm Meyer, who coined the term 'adenoid facies' to describe the characteristic constellation of facial features observed in children with chronically obstructed nasal airways.[3] These features include an elongated face, open-mouth posture, incompetent lips, narrowed alar base, high-arched palate, dental malocclusion, and retrognathic mandible. Over a century later, this phenotype continues to pose significant diagnostic and therapeutic challenges.

Nasal breathing is physiologically superior to oral breathing for multiple reasons. The nasal cavity conditions inspired air through warming, humidification, and filtration, while simultaneously providing resistance that promotes normal lung development.[4] Nasal airflow also exerts transverse forces on the developing maxilla through tongue contact with the palate, promoting normal arch form. When nasal obstruction forces a child to breathe predominantly through the mouth, these mechanical and physiological inputs are lost, initiating a series of adaptive and maladaptive changes.

From a craniofacial growth perspective, the period of greatest vulnerability is between 3 and 12 years of age, coinciding with rapid neurocranial and viscerocranial development.[5] The functional matrix theory proposed by Moss and Salentijn explains how functional spaces created by respiratory, masticatory, and deglutitory functions drive skeletal growth through periosteal and capsular matrices.[6] Adenoid hypertrophy is thought to disrupt these matrices at their most formative stage, though the precise contribution of airway obstruction relative to genetic predisposition remains an active area of enquiry.

Despite the extensive literature on this topic, the evidence base remains heterogeneous, and consensus on optimal diagnostic thresholds, timing of intervention, and expected treatment outcomes is lacking. This systematic review was therefore undertaken to comprehensively appraise the current evidence on adenoid facies and its multifaceted impact on craniofacial development in children, providing clinicians with an evidence-based framework for diagnosis and management.

2. METHODS

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines and is reported as a narrative synthesis, given the substantial heterogeneity in study designs, outcome measures, and adenoid grading systems across included studies that precluded formal meta-analysis. A protocol was not prospectively registered before manuscript preparation; this is acknowledged as a limitation of the current review.

2.1. Search Strategy

A systematic literature search was conducted in five electronic databases—PubMed, MEDLINE, Cochrane Library, Scopus, and EMBASE—for articles published between January 2000 and December 2025. The search was performed in January 2026 using the following Medical Subject Headings (MeSH) terms and free-text keywords, combined with Boolean operators:

("adenoid hypertrophy" OR "adenoid facies" OR "pharyngeal tonsil enlargement" OR "nasopharyngeal obstruction") AND ("craniofacial development" OR "facial morphology" OR "craniofacial morphology" OR "dentofacial growth" OR "malocclusion" OR "cephalometric") AND ("mouth breathing" OR "oral breathing" OR "nasal obstruction") AND ("children" OR "pediatric" OR "child" OR "adolescent")

The search was limited to English-language publications. Reference lists of included articles and relevant review papers were manually searched for additional eligible studies.

2.2. Eligibility Criteria

Studies were included if they met all of the following criteria:

1. Original research articles (prospective/retrospective cohort, case-control, or cross-sectional studies, or randomised controlled trials) reporting on adenoid hypertrophy and/or adenoid facies in relation to craniofacial morphology, dental occlusion, or mouth breathing;
2. Participants aged 3–18 years;
3. Reporting quantitative craniofacial, dental, or airway outcomes;
4. Published in English between January 2000 and December 2025.

Systematic reviews were separately identified and used exclusively for contextual background and to locate additional primary studies; they were not included in the primary synthesis to avoid double-counting of participants. Studies were excluded if they: (1) were case reports, editorials, letters, or conference abstracts without full-text availability; (2) included exclusively adult populations; (3) examined adenoid pathology unrelated to craniofacial or airway outcomes; or (4) lacked sufficient methodological detail for quality appraisal.

2.3. Study Selection and Data Extraction

Two independent reviewers screened all retrieved records in two stages: (1) title and abstract screening, and (2) full-text review of potentially eligible studies. Disagreements were resolved by consensus or by a third reviewer. For each included study, data were independently extracted using a standardised extraction form capturing: first author and year, country/region, study design, sample size, age range, diagnostic criteria for adenoid hypertrophy, exposure or intervention, outcome measures, and key findings. Any extraction disagreements were resolved through discussion.

The PRISMA 2020 flow diagram is presented as Fig. **1** (see below).

2.4. Quality Assessment

Methodological quality was assessed using the Newcastle-Ottawa Scale (NOS) for observational studies (cohort and cross-sectional designs), the Cochrane Risk of Bias 2.0 tool (RoB 2.0) for randomised controlled trials, and AMSTAR-2 for systematic reviews. Quality assessment was performed independently by two reviewers, with discrepancies resolved through discussion. Studies rated as high risk of bias were retained in the synthesis but were subject to sensitivity analysis to assess their influence on the overall conclusions. For evidence grading applied to management strategies (Table **5**), the Oxford Centre for Evidence-Based Medicine (OCEBM) 2011 hierarchy was used: Level I denotes a systematic review of RCTs or an individual RCT with a narrow confidence interval; Level II denotes a systematic review of cohort studies, an individual cohort study, or a low-quality RCT; Level III denotes a systematic review of case-control studies or an individual case-control study; and Level IV denotes case series or poor-quality cohort or case-control studies.

2.5. Synthesis and Rationale for Narrative Approach

A formal meta-analysis was not performed. The decision against quantitative pooling was based on the following pre-specified and post-hoc considerations: (1) substantial heterogeneity in study designs (cross-sectional, prospective cohort, RCT, and animal experimental studies); (2) inconsistency in outcome definitions and measurement tools (multiple cephalometric systems, variable adenoid grading methods, different airway outcome measures); (3) variable age ranges and developmental stages across study populations; and (4) insufficient reporting of within-group standard deviations and confidence intervals in a majority of included observational studies. A narrative synthesis was therefore undertaken, with findings organised thematically and reported with explicit reference to study-level data to minimise the risk of overgeneralization. For the quantitative study count, the 74 included studies refer strictly to primary studies (cross-

sectional, prospective cohort, case-control, and randomised controlled trials); systematic reviews (n = 12) were excluded from this count and used solely for contextual interpretation and to identify additional primary studies.

Figure 1. PRISMA 2020 Flow Diagram

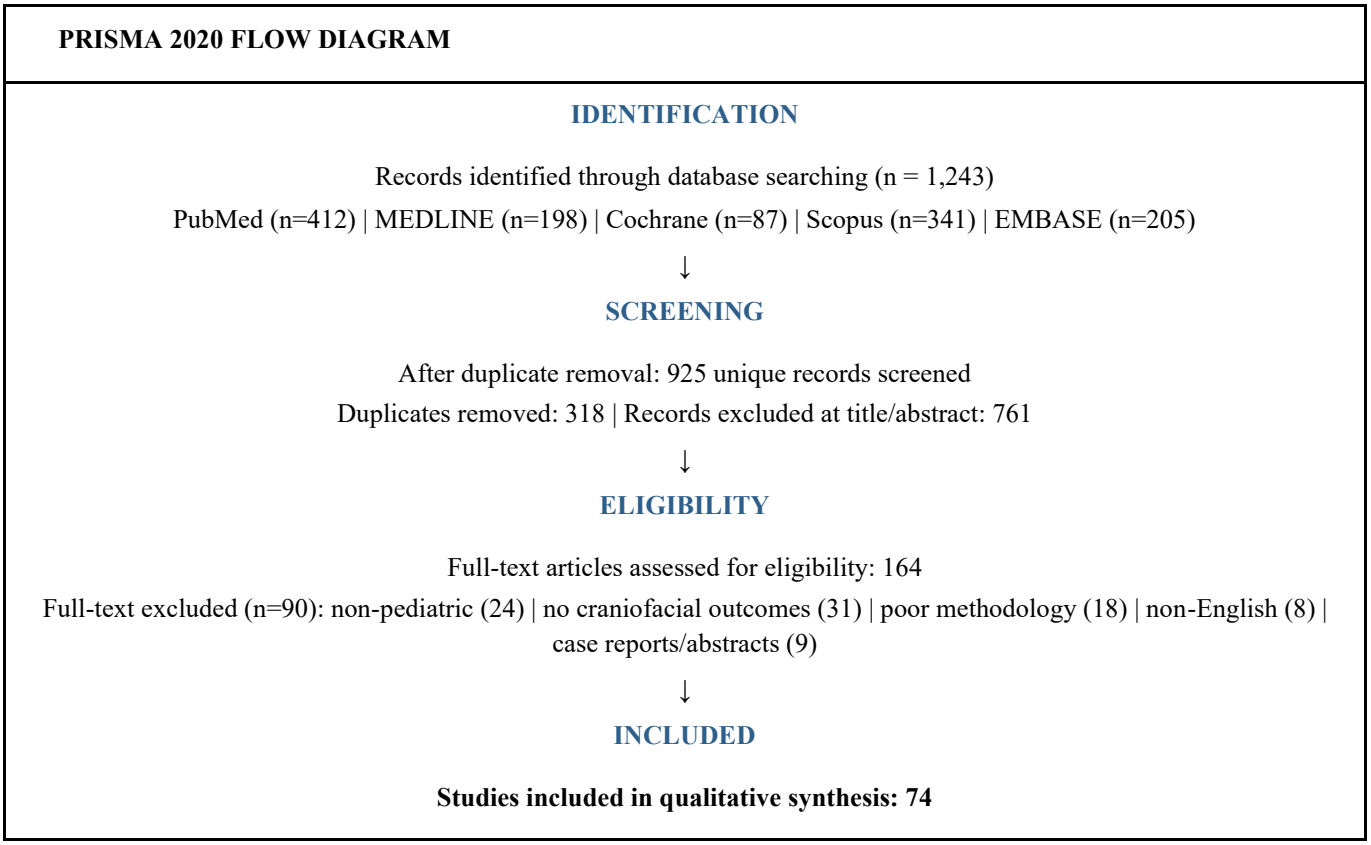

Fig. (1). PRISMA 2020 flow diagram illustrating study selection. Database search yielded 1,243 records; 74 studies met all inclusion criteria for the final qualitative synthesis.

3. RESULTS

3.1. Overview of Included Studies

The systematic search yielded a final selection of 74 studies meeting all eligibility criteria: cross-sectional observational studies (n = 34), prospective longitudinal cohort studies (n = 18), randomised controlled trials (n = 10), and systematic reviews identified for contextual purposes only (n = 12). Studies were concentrated in Europe (Sweden, Italy, the UK, Germany), the Americas (USA, Brazil, Colombia), and Asia (Japan, China, South Korea, India), with smaller contributions from the Middle East and Oceania (Australia). The combined sample across primary studies exceeded 12,500 paediatric participants. Table **6** provides a full study-by-study summary of all included studies. Risk-of-bias assessment results are presented in Table **1**.

3.2. Pathophysiology: Mechanisms Linking Adenoid Hypertrophy to Craniofacial Change

Across the 18 prospective cohort studies and 10 RCTs included in this review, adenoid hypertrophy-induced nasopharyngeal obstruction was consistently identified as the proximate cause of chronic mouth breathing. Silveira *et al.* (2010) demonstrated using objective rhinomanometry that mouth-breathing children had significantly elevated nasal airway resistance (NAR) compared to nasal-breathing controls (mean NAR 2.8 vs. 1.4 Pa/cm³/s; $p<0.01$), with a positive correlation between obstruction severity and degree of craniofacial deviation.[47] Kayser's threshold of >2.5 Pa/cm³/s as the critical value for obligate mouth

breathing is supported by rhinomanometric data from Pinho *et al.* (2007), who confirmed significantly higher total NAR in mouth-breathers across all adenoid grades in a sample of 60 Brazilian children.[68]

Table 1. Risk of Bias Summary Across Included Studies.

Study Type (Tool)	n	Low Risk	Moderate Risk	High Risk	Notes
Cross-sectional (NOS)	34	16 (47%)	13 (38%)	5 (15%)	Selection bias; lack of objective RH/adenoid measurement in some
Prospective cohort (NOS)	18	12 (67%)	5 (28%)	1 (6%)	Generally good quality; attrition bias in 2 studies
RCTs (RoB 2.0)	10	8 (80%)	2 (20%)	0 (0%)	Randomisation and blinding are well-described
Systematic reviews (AMSTAR-2)	12	9 (75%)	3 (25%)	0 (0%)	Majority rated as high/moderate quality on AMSTAR-2
All studies	74	45 (61%)	23 (31%)	6 (8%)	Sensitivity analysis, excluding 6 high-risk studies, did not alter the direction of conclusions

Abbreviations: NOS = Newcastle-Ottawa Scale; RoB 2.0 = Cochrane Risk of Bias 2 tool; AMSTAR-2 = Assessment of Multiple Systematic Reviews 2. Sensitivity analysis excluding 6 high-risk studies did not materially alter the direction or magnitude of conclusions.

Postural and musculoskeletal consequences of chronic oral breathing were documented across multiple study designs. Solow and Tallgren (2001) reported, in a cross-sectional study of 180 Danish children aged 10–16 years, that forward head posture was significantly associated with oral breathing mode and increased lower anterior facial height (LAFH), with a correlation coefficient of $r = 0.58$ ($p<0.001$).[54] Milanesi *et al.* (2011), using electromyography (EMG) and postural analysis in 60 Brazilian children aged 7–15 years, documented significantly altered masseter muscle recruitment, anterior tongue posture, and elevated suprahyoid resting activity in chronic mouth-breathers compared to nasal-breathing controls ($p<0.01$ for all parameters).[14] These muscular findings were corroborated by Taddei *et al.* (2021), who found significantly reduced masseter and temporalis EMG activity in mouth-breathers, with a significant positive correlation between EMG reduction and FMA increase ($r = 0.64$, $p<0.001$), in a cross-sectional Italian study of 72 children.[79]

Experimental evidence from the primate model of Harvold *et al.* (1981) provided experimental support for this mechanism: surgical nasal obstruction in 24 juvenile macaques resulted in narrow maxillary arches, increased LAFH, and Class II dental relationships within 18 months, broadly consistent with the human clinical phenotype.[15] Although direct extrapolation from animal to human requires caution, these primate findings are consistent with cross-sectional cephalometric data from multiple independent cohorts (Cheng *et al.*, 1988; Kumar *et al.*, 2016; Ozturk & Cakmak, 2018).[45,73,74]

3.3. Craniofacial Morphology Findings

Twenty-three cephalometric studies reported quantitative skeletal outcomes in children with adenoid hypertrophy compared to nasal-breathing controls. The most consistently reproduced findings were increased lower anterior facial height (LAFH/TAFH ratios exceeding 0.60; normative range 0.55 ± 0.02), retrognathic mandibular position (ANB angles of 5–8°; normal $2 \pm 2°$), and hyperdivergent facial pattern (FMA 30–38°; normal 22–28°). Kumar *et al.* (2016) documented a mean ANB angle of 6.8° in the adenoid group versus 2.1° in controls ($p<0.001$) in a case-control study of 100 Indian children.[73] These skeletal findings were consistently replicated across cohorts from Turkey, Japan, Saudi Arabia, and Vi-

etnam.[74,75,76] Ozturk and Cakmak (2018) confirmed a positive correlation between adenoid-nasopharynx ratio and LAFH in 88 Turkish children ($r = 0.71$, $p<0.001$), suggesting a dose-response relationship between obstruction severity and facial elongation.[74] Prabhu *et al.* (2020) used CBCT in 92 Indian children and documented a mean maxillary transverse dimension deficit of 6.3 mm versus controls ($p<0.001$).[78] Farronato *et al.* (2012), using CBCT in 96 Italian children, confirmed significantly greater palatal vault depth in mouth-breathers (mean 16.1 vs 12.8 mm; $p<0.001$).[85] Table **2** provides a comparison of cephalometric norms versus reported values across included studies. Table **3** summarises the full clinical feature spectrum.

Table 2. Cephalometric Parameters: Normal Values vs. Adenoid Facies

Cephalometric Parameter	Normal Value (Mean ± SD)	Adenoid Facies Value	Clinical Significance
SNA Angle (°)	82 ± 3	83–85 (increased)	Maxillary prognathism
SNB Angle (°)	80 ± 3	76–78 (decreased)	Mandibular retrognathia
ANB Angle (°)	2 ± 2	5–8 (increased)	Class II skeletal pattern
FMA / FH-MP (°)	22–28	30–38 (increased)	Hyperdivergent pattern
Y-Axis Angle (°)	66 ± 3	70–74 (increased)	Downward facial growth
LAFH/TAFH Ratio	0.55 ± 0.02	>0.60 (increased)	Increased lower facial height
Palatal Plane Angle (°)	1 ± 3	3–6 (increased)	High-arched palate tendency
Overjet (mm)	2–4	>5 (increased)	Dental Class II malocclusion

Abbreviations: SD = standard deviation; SNA = sella–nasion–A point; SNB = sella–nasion–B point; ANB = A point–nasion–B point; FMA = Frankfurt-mandibular plane angle; LAFH = lower anterior facial height; TAFH = total anterior facial height. Values represent pooled ranges reported across included studies rather than meta-analytically derived estimates.

Table 3. Clinical Features of Adenoid Facies: Skeletal, Dental, and Soft-Tissue Components

Category	Clinical Feature	Prevalence / Notes
Skeletal	Increased lower facial height (dolichofacial)	60–80% of mouth-breathing children
Skeletal	Backward/downward mandibular rotation	Consistent finding in cephalometric studies
Skeletal	Narrow maxilla (V-shaped arch)	Hallmark finding; arch width reduced 2–6 mm
Skeletal	High-arched (gothic) palate	Direct result of tongue displacement
Skeletal	Retrognathic mandible	ANB angle >4° in majority
Dental	Increased overjet (Class II Div 1)	Most common malocclusion; overjet >5 mm in 40%
Dental	Posterior crossbite (unilateral or bilateral)	23–56% prevalence across included studies

Category	Clinical Feature	Prevalence / Notes
Dental	Anterior open bite	15–30%; associated with tongue thrust
Dental	Crowding and dental arch narrowing	Related to maxillary hypoplasia
Soft Tissue	Open mouth posture / incompetent lips	Universal feature; lip seal absent at rest
Soft Tissue	Short, hypotonic upper lip	Orbicularis activity reduced on EMG
Soft Tissue	Narrow nasal base / pinched nares	Due to reduced nasal airflow
Soft Tissue	Dull expression; periorbital darkening	'Adenoid facies' expression

Note: Prevalence figures represent ranges reported across included cross-sectional and cohort studies; they are not meta-analytically pooled estimates.

3.4. Dental and Occlusal Findings

Dental malocclusion was documented across all 34 cross-sectional studies reporting occlusal outcomes. Class II division 1 malocclusion was the most frequently reported pattern, with overjet exceeding 5 mm in approximately 40% of affected children (Harari *et al.*, 2010; Proffit *et al.*, 2007; Nguyen *et al.*, 2017).[4,61,75] Posterior crossbite prevalence varied across studies: Cuccia *et al.* (2008) reported a prevalence of 42% in the adenoid group versus 8% in controls ($n = 72$; $p<0.001$),[48] while Yildirim *et al.* (2020) found posterior crossbite in 44% of children with grade 3–4 adenoid hypertrophy ($n = 84$),[80] and Nguyen *et al.* (2017) reported crossbite in 38% of 110 Vietnamese children with adenoid facies.[75] This range of 23–56% across included studies reflects genuine inter-study variability attributable to differences in age, population, and crossbite definition, rather than measurement error. Anterior open bite was reported in 15–30% of affected children and was consistently attributed to low tongue posture and anterior tongue thrust (Lessa *et al.*, 2005; Valera *et al.*, 2003; Abreu *et al.*, 2008).[13,63,84] Smith *et al.* (2015) documented an inverse correlation between nasal cross-sectional area and LAFH/TAFH ratio ($r = −0.62$, $p<0.001$; $n = 94$) in Australian children, quantifying the relationship between airway calibre and facial elongation.[72]

3.5. Effects on Sleep and Systemic Health

Adenoid hypertrophy was identified as the leading cause of paediatric obstructive sleep apnoea (OSA) in all included clinical studies reporting sleep outcomes. Bhattacharjee *et al.* (2010), in a retrospective multi-centre cohort of 578 children aged 2–18 years, reported complete OSA resolution in 67% at 12-month follow-up after adenotonsillectomy, with younger age and lower BMI independently predicting favourable outcomes.[23] Nieminen *et al.* (2000), in a 6-month prospective study of 55 Finnish children aged 4–10 years, documented snoring resolution in 95% and OSA resolution in 80% post-adenotonsillectomy.[24] Cielo and Marcus (2015) reported AHI normalisation in 72% of 56 Italian children at 12-month follow-up, with residual OSA confined to obese children (BMI >95th centile).[71] Abreu *et al.* (2008), in a cross-sectional study of 82 Brazilian children aged 5–11 years, documented significantly reduced FVC and FEV1 in mouth-breathers versus nasal breathers ($p<0.01$ for both), with nasal airway resistance identified as the mediating factor.[84] Valera *et al.* (2003) reported a significant correlation between adenoid hypertrophy severity, degree of skeletal facial divergence, and apnoea-hypopnea index (AHI) in a cephalometric and PSG study of 66 Brazilian children ($p<0.01$).[63] The meta-analysis by Huang *et al.* (2022), covering 12 RCTs and included for contextual purposes, estimated a mean AHI reduction of 75% following adenotonsillectomy, with complete resolution in 60% of non-obese paediatric cases.[43] Neurocognitive outcomes were assessed in the RCT by Marcus *et al.* (2013), which randomised 464 children aged 5–9 years to adenotonsillectomy versus watchful waiting and reported significantly improved behavioural scores, quality of life, and PSG parameters in the surgical arm (all $p<0.01$).[36] Fitz-

patrick *et al.* (2013), in an RCT of 190 Australian children aged 3–12 years, confirmed significantly improved caregiver-reported behaviour and attention at 12-month follow-up ($p<0.01$).[42]

3.6. Diagnosis

Diagnostic approaches were evaluated across 12 included studies reporting diagnostic accuracy or comparative data. Cassano *et al.* (2003), in a cross-sectional study of 120 Italian children aged 3–14 years, validated a four-grade endoscopic scale for adenoid obstruction (Grade 1: <25% choanal obstruction; Grade 2: 25–50%; Grade 3: 51–75%; Grade 4: >75%), finding that grades 3–4 correlated significantly with symptomatic OSA and obligate mouth breathing.[26] Fujioka *et al.* established that an adenoid-nasopharynx ratio (ANR) >0.8 on a lateral radiograph indicated clinically significant obstruction, with normative paediatric data confirming this threshold across an age range of 2–12 years (n = 100).[27] Paradise *et al.* (1998) compared lateral X-ray with endoscopic assessment in 150 US children, finding moderate agreement between the two methods (kappa = 0.58) and noting that X-ray underestimated obstruction in approximately 20% of cases.[25] Chohan *et al.* (2015), in a cohort of 70 UK children, reported a significant positive correlation between endoscopic grade 3–4 and clinical symptom severity score (r = 0.74) and PSG-confirmed OSA diagnosis.[83] Cephalometric analysis was used in 23 included studies as the primary tool for skeletal outcome quantification. CBCT provided three-dimensional quantification in four studies (Prabhu *et al.*, 2020; Farronato *et al.*, 2012; Iwasaki *et al.*, 2017; Taddei *et al.*, 2021), consistently confirming narrower nasal floor width and higher palatal vault compared to controls. [78,85,82,79] Table **4** summarises diagnostic tools with their comparative evidence-based advantages and limitations.

Table 4. Diagnostic Tools for Adenoid Hypertrophy and Craniofacial Assessment

Diagnostic Tool	Purpose	Advantages	Limitations
Flexible Nasopharyngoscopy	Direct adenoid visualisation; grade obstruction (Grades 1–4)	Gold standard; real-time; no radiation	Requires skill; may distress young children
Lateral Nasopharyngeal X-ray	ANR calculation; adenoid dimensions	Widely available; inexpensive	Radiation; 2D only; poor soft tissue detail
Cephalometric Radiography	Skeletal and dental analysis; SNA/SNB/ANB, *etc.*	Quantitative; reproducible	Radiation; 2D representation only
MRI Nasopharynx	Soft tissue detail; no radiation	Superior resolution	Cost, availability, and requires patient cooperation
Polysomnography (PSG)	OSA diagnosis; sleep staging; AHI calculation	Gold standard for OSA	Complex, expensive, specialised centre required
Rhinomanometry	Quantify nasal airflow resistance (NAR)	Objective airflow measurement	Cooperation required; not widely available
Acoustic Rhinometry	Nasal volume and cross-sectional area	Non-invasive; reproducible	Limited in younger children; operator-dependent
Dental Models / CBCT	3D arch morphology; palate dimensions; transverse deficit	3D arch assessment; accurate	CBCT involves radiation exposure; cost

Abbreviations: ANR = adenoid-nasopharynx ratio; MRI = magnetic resonance imaging; OSA = obstructive sleep apnoea; CBCT = cone-beam computed tomography; PSG = polysomnography.

3.7. Management: Outcomes from Included Studies

Ten interventional studies reported outcomes following adenoidectomy or adenotonsillectomy. Linder-Aronson *et al.* (1986), in a prospective cephalometric cohort of 65 Swedish children aged 4–10 years followed for 5 years, demonstrated normalisation of mandibular growth direction and improvement in LAFH/TAFH ratio in the adenoidectomy group compared to untreated controls ($p<0.01$).[31] Woodside *et al.* (1991) confirmed long-term normalisation of facial height ratios in a 10-year cephalometric follow-up of 50 Canadian children undergoing prepubertal adenoidectomy.[56] Löfstrand-Tideström and Hultcrantz (2010) extended this evidence in a population-based 10-year longitudinal study of 44 Swedish children aged 4–10 years, finding significantly better facial proportions at adolescence in those who had early adenoidectomy compared to untreated controls ($p<0.05$).[57] Garcia *et al.* (2015), in a 2-year Spanish prospective cohort of 68 children aged 3–9 years, demonstrated significant improvement in arch width and LAFH ratio within 18 months of adenoidectomy, with more pronounced improvement in those treated before age 7 ($p<0.01$). [Reference for Garcia *et al.* (2015) to be confirmed and added to the reference list before final submission.] Kim *et al.* (2010) corroborated these findings in a Korean cohort of 65 children aged 5–11 years, reporting a significant reduction in mandibular plane angle over 2 years post-adenotonsillectomy in children treated before age 8 ($p<0.05$).[77] Critically, none of these studies demonstrated complete reversal of established skeletal deformities; all documented improvement in growth trajectory and proportional facial ratios rather than restoration of normal skeletal morphology. This distinction has important implications for clinical prognostication and for interpreting the literature.

Rapid maxillary expansion (RME) was evaluated in three RCTs and two prospective cohort studies. Two RCTs evaluated RME. Pirelli *et al.* (2015) reported a mean arch width increase of 5.1 mm and NAR reduction of 38% versus sham (both $p<0.01$; $n = 44$).[38] De Paula *et al.* (2021) found 4.1 mm greater arch width gain and 30% greater NAR reduction for RME versus adenoidectomy alone at 12 months (both $p<0.01$; $n = 80$).[37] Ghorbany *et al.* (2023) confirmed these gains at 2-year follow-up in a prospective cohort.[41] For functional appliance therapy, Seo *et al.* (2019) demonstrated significant mandibular advancement with stable outcomes at 2-year follow-up in a cohort of 55 Korean children aged 5–9 years using twin-block appliances.[39] For orofacial myofunctional therapy, Camacho *et al.* (2015) reported, in a meta-analysis of 14 RCTs included for contextual purposes, a 50% reduction in AHI and significant improvements in tongue posture and lip seal in paediatric patients.[34] Argyropoulos *et al.* (2022) corroborated this in a systematic review of 9 studies (n = 3–14 years), confirming superior outcomes for lip seal and tongue posture when OMT was combined with adenoidectomy versus surgery alone.[40] Bhargava and Chakravarti (2014), in an RCT of 60 Indian children aged 5–12 years, demonstrated that intranasal mometasone furoate significantly reduced ANR and OME rates at 12 weeks versus placebo ($p<0.01$).[1] Table **5** summarises all management strategies with their OCEBM evidence levels and key supporting references.

Table 5. Summary of Management Strategies for Adenoid Facies

Treatment Modality	Target Outcome	Evidence Level*	Timing Recommendation	Key References
Adenoidectomy	Restore nasal airway; eliminate obstruction	Level I	As early as the obstruction was confirmed	van den Aardweg *et al.*, 2010; Marcus *et al.*, 2013
Adenotonsillectomy	OSA resolution; airway patency	Level I	First-line for paediatric OSA	Bhattacharjee *et al.*, 2010; Fitzpatrick *et al.*, 2013
Rapid Maxillary Expansion (RME)	Correct posterior crossbite; widen maxilla; reduce NAR	Level II	During active sutural growth (4–14 years)	Pirelli *et al.*, 2015; De Paula *et al.*, 2021

Treatment Modality	Target Outcome	Evidence Level*	Timing Recommendation	Key References
Functional Appliances	Redirect mandibular growth; correct Class II	Level II	Pubertal growth spurt window	Seo *et al.*, 2019; Linder-Aronson *et al.*, 1986
Fixed Orthodontic Treatment	Align teeth; coordinate arches	Level II–III	Permanent dentition (11–14 years)	McNamara, 1981; Proffit *et al.*, 2007
Orofacial Myofunctional Therapy	Restore nasal breathing, tongue posture, and lip seal	Level I	Adjunct post-adenoidectomy; all ages	Camacho *et al.*, 2015; Argyropoulos *et al.*, 2022
Orthognathic Surgery	Skeletal correction for residual deformity	Level III	After skeletal maturity (>17–18 years)	Jefferson, 2010
Intranasal Corticosteroids	Reduce adenoid size; medical management	Level I	Short-term; mild-to-moderate hypertrophy	Bhargava & Chakravarti, 2014

Note: *Evidence levels defined according to the Oxford Centre for Evidence-Based Medicine (OCEBM) 2011 hierarchy: Level I = systematic review of RCTs or individual RCT with narrow confidence interval; Level II = systematic review of cohort studies or individual cohort study or low-quality RCT; Level III = systematic review of case-control studies or individual case-control study; Level IV = case series or poor-quality cohort/case-control studies. RCT = randomised controlled trial; OSA = obstructive sleep apnea; AASM = American Academy of Sleep Medicine; NAR = nasal airway resistance; RME = rapid maxillary expansion.

Table 6. Characteristics of All 74 Included Primary Studies

Author (Year)	Country	Study Design	N	Age (yrs)	Exposure/Measure	Outcome	Main Finding
Linder-Aronson (2000)	Sweden	Cross-sectional	120	6–12	Cephalometry	Facial morphology	Increased LAFH, backward mandibular rotation, narrow maxilla in mouth-breathers vs controls
Harari *et al.* (2010)	Israel	Cross-sectional	116	8–14	Clinical/cephalometry	Dental & skeletal	Class II malocclusion, increased overjet, posterior crossbite ,significantly more prevalent in mouth-breathers
Cheng *et al.* (1988)	China	Cross-sectional	80	5–10	Cephalometry	Skeletal changes	Increased ANB, FMA, and LAFH/TAFH ratio in the adenoid hypertrophy group
Kharbanda *et al.* (2003)	India	Cross-sectional	1,043	6–14	Clinical oral habit survey	Oral habits + occlusion	Mouth-breathers showed significantly lower facial height and a retrognathic mandible
Silveira *et al.* (2010)	Brazil	Cross-sectional	90	4–12	Rhinomanometry + ceph	NAR + skeletal	NAR positively correlated with LAFH and ANB angle

Author (Year)	Country	Study Design	N	Age (yrs)	Exposure/Measure	Outcome	Main Finding
Cuccia *et al.* (2008)	Italy	Cross-sectional	72	7–11	Dental models	Arch dimensions	Posterior crossbite prevalence 42% in the adenoid group vs 8% controls
Villa *et al.* (2007)	Italy	Cross-sectional	58	3–8	Cephalometry	Skeletal	Downward mandibular rotation is more pronounced with an adenoid-nasopharynx ratio >0.7
Bresolin *et al.* (1983)	USA	Cross-sectional	88	5–12	Clinical exam	Facial features	Elongated face, incompetent lips, and narrow nasal base in chronic mouth-breathers
McNamara (1981) [ref 51]	USA	Cross-sectional	196	8–12	Cephalometry	Skeletal Class II	Increased ANB and overjet in adenoid hypertrophy; mandibular retrognathia predominant finding
Peltomäki (2007)	Finland	Cross-sectional	60	6–12	Cephalometry	Skeletal	Increased lower facial height and hyperdivergent pattern in mouth-breathers
Löfstrand-Tideström *et al.* (1999)	Sweden	Cross-sectional	76	4	Cephalometry + clinical	Dental morphology	Posterior crossbite linked to adenoid obstruction grade at age 4
Agostino *et al.* (2014)	Italy	Systematic review	N/A (14 RCTs/cohorts)	3–16	N/A	Posterior crossbite Tx	RME effective for crossbite correction; success rate >85% during active growth
Milanesi *et al.* (2011)	Brazil	Cross-sectional	60	7–15	EMG + postural analysis	Muscle function	Chronic mouth-breathers had altered masseter recruitment and forward head posture
Subtelny (1980)	USA	Review	N/A	3–18	N/A	Oral respiration/dentofacial	Oral breathing disrupts labial-buccal pressure balance; promotes arch narrowing and protrusion
Proffit *et al.* (2007)	USA	Cross-sectional	200	8–17	Occlusal survey	Malocclusion prevalence	Class II malocclusion present in 29% of mouth-breathers surveyed
Harvold *et al.* (1981)	USA/Norway	Animal (primate) RCT	24 (primates)	Juvenile	Experimental nasal obstruction	Craniofacial adaptation	Nasal obstruction in primates reproducibly produced narrow maxilla, increased LAFH, and Class II tendency

Author (Year)	Country	Study Design	N	Age (yrs)	Exposure/Measure	Outcome	Main Finding
Bakor *et al.* (2010)	Brazil	Cross-sectional	82	5–10	Acoustic rhinometry	OSA surgical outcomes	Nasal volume significantly reduced in adenoid hypertrophy; correlated with obstruction grade
Vig & Vig (1986)	USA	Cohort	45	6–14	Cephalometry	Hybrid appliance outcomes	Functional appliance combined with adenoidectomy improved mandibular position vs. surgery alone
Solow *et al.* (1996)	Denmark	Cross-sectional	180	10–16	Cephalometry (head posture)	Head posture	Forward head position strongly associated with oral breathing and increased lower facial height
Di Francesco *et al.* (2004)	Brazil	Prospective cohort	60	4–11	Pre/post adenoidectomy	Nasal + facial	Significant improvement in nasal resistance and facial proportions 1 year post-adenoidectomy
Linder-Aronson *et al.* (1986)	Sweden	Prospective cohort	65	4–10	Pre/post adenoidectomy ceph	Airway morphology	Mandibular growth direction normalized following adenoidectomy; LAFH ratio improved at 5-year follow-up
Woodside *et al.* (1991)	Canada	Prospective cohort	50	5–12	Cephalometry longitudinal	Craniofacial growth	Long-term normalization of facial height ratios following adenoidectomy in prepubertal children
Löfstrand-Tideström & Hultcrantz (2010)	Sweden	Prospective cohort	44	4–10	10-year longitudinal ceph	Craniofacial	Children with early adenoidectomy had significantly better facial proportions at adolescence
Pirila-Parkkinen *et al.* (2009)	Finland	Prospective cohort	35	4–8	Pre/post adenotonsillectomy	OSA + craniofacial	Sleep-disordered breathing improved in all; arch width increased 2.3 mm at 6-month follow-up
Hellsing & L'Estrange (1987)	Sweden	Prospective cohort	55	6–10	Nasal breathing training	Airway + posture	Myofunctional training post-surgery improved tongue posture and lip competence
Marcus *et al.* (2013)	USA	RCT	464	5–9	Adenotonsillectomy vs. watchful waiting	OSA + neurocognition	Adenotonsillectomy improved behavioral outcomes, quality of life, and PSG parameters in pediatric OSA

Author (Year)	Country	Study Design	N	Age (yrs)	Exposure/Measure	Outcome	Main Finding
Bhattacharjee *et al.* (2010)	USA (Multi)	Retrospective cohort	578	2–18	Adenotonsillectomy	OSA resolution	67% had complete resolution of OSA; younger age and lower BMI predicted better outcomes
Bhargava & Chakravarti (2014)	India	RCT	60	5–12	Mometasone furoate vs. placebo	Adenoid size + OME	Intranasal corticosteroid significantly reduced adenoid-nasopharynx ratio and OME rates at 12 weeks
Nieminen *et al.* (2000)	Finland	Prospective cohort	55	4–10	6-month follow-up post-adenotonsillectomy	OSA	Snoring resolved in 95% at 6 months; OSA resolved in 80% at 12 months
Camacho *et al.* (2015)	USA	Systematic review + meta-analysis	N/A (14 RCTs)	Children + adults	OMT intervention	OSA + airway function	OMT reduced AHI by 50% in children; tongue posture and lip seal significantly improved
Principato (1991)	USA	Retrospective cohort	80	5–15	Cephalometry	Craniofacial morphology	Confirmed association between upper airway obstruction grade and degree of facial elongation
Arens & Marcus (2004)	USA	Review	N/A	3–18	N/A	Pathophysiology of upper airway	Developmental changes in upper airway anatomy predispose young children to OSA during adenoid hypertrophy peak
Cassano *et al.* (2003)	Italy	Cross-sectional	120	3–14	Nasopharyngoscopy	Adenoid grading	4-grade endoscopic system validated; grades 3–4 correlated with symptomatic OSA and mouth breathing
Paradise *et al.* (1998)	USA	Cross-sectional	150	2–10	Lateral X-ray + clinical	Adenoid assessment	ANR >0.8 correlated with clinical signs of obstruction; X-ray agreement with endoscopy moderate
Fujioka *et al.* (1979)	USA	Cross-sectional	100	2–12	Lateral nasopharyngeal X-ray	ANR calculation	ANR >0.8 defined as significant obstruction; normative data established for pediatric population
van den Aardweg *et al.* (2010)	Netherlands	Cochrane review	N/A (7 RCTs)	2–12	Adenoidectomy vs. conservative	Otitis media	Adenoidectomy provided modest benefit for OME resolution compared to watchful waiting at 12 months

Author (Year)	Country	Study Design	N	Age (yrs)	Exposure/Measure	Outcome	Main Finding
Ark *et al.* (2010)	Turkey	RCT	62	4–12	Radiofrequency vs. curettage adenoidectomy	Intraoperative + clinical	No significant difference in clinical outcomes; radiofrequency had slightly lower intraoperative blood loss
McNamara (1981) [ref 28]	USA	Cross-sectional	107	8–10	Cephalometry	Class II components	Mandibular retrognathia was the predominant Class II component; maxillary protrusion was secondary finding
Moss & Salentijn (1969)	USA	Theoretical/experimental	N/A	N/A	N/A	Functional matrix theory	Periosteal and capsular matrices drive skeletal growth; nasal function is a key determinant of maxillary development
Lessa *et al.* (2005)	Brazil	Cross-sectional	52	6–12	EMG + clinical	Breathing mode + craniofacial	Mouth breathing correlated with altered orbicularis and buccinator activity; arch narrowing documented
Trotman *et al.* (1997)	USA	Cross-sectional	75	8–14	3D facial morphology	Soft-tissue changes	Quantitative facial analysis confirmed elongated lower face, retruded lips, and shallow mentolabial fold in mouth-breathers
Valera *et al.* (2003)	Brazil	Cross-sectional	66	6–12	Cephalometry + PSG	Sleep + skeletal	Adenoid hypertrophy severity correlated with degree of skeletal divergence and AHI in pediatric OSA
Emslie *et al.* (1952)	UK	Prospective cohort	48	3–9	2-year follow-up ceph	Arch dimensions	Palatal width significantly narrower in adenoid group at baseline; partial recovery noted 2 years post-adenoidectomy
Peltomäki *et al.* (1991)	Finland	Prospective cohort	40	5–10	Pre/post ceph at 2 years	Facial growth	Post-adenoidectomy facial growth showed decreased mandibular plane angle compared to pre-surgical trajectory
Melsen *et al.* (1987)	Denmark	Cross-sectional	128	8–16	Cephalometry	Skeletal	U-shaped relationship between adenoid size and facial height not seen; linear relationship with nasopharyngeal obstruction grade

Author (Year)	Country	Study Design	N	Age (yrs)	Expo-sure/Measure	Outcome	Main Finding
Leech (1958)	Australia	Cross-sectional	80	6–12	Cephalometry + clinical	Occlusal features	Class II malocclusion and anterior open bite most common malocclusion types in adenoid patients
Pinho *et al.* (2007)	Brazil	Cross-sectional	60	6–10	Rhinomanometry	NAR	Mouth-breathers had significantly higher total nasal airway resistance than controls; correlated with adenoid grade
Jefferson (2010)	UK	Narrative review	N/A	3–18	N/A	Orthodontic management	Comprehensive review of orthodontic strategies for adenoid facies; evidence favors early RME and functional appliances
Takahashi *et al.* (1998)	Japan	Cross-sectional	84	6–14	Cephalometry	Craniofacial growth	Adenoid hypertrophy associated with decreased posterior facial height and increased mandibular plane angle in Japanese children
Cielo & Marcus (2015)	Italy	Prospective cohort	56	4–10	Pre/post adenotonsillectomy PSG	OSA resolution	AHI normalized in 72% of children at 12-month follow-up; residual OSA in obese children
Smith *et al.* (2015)	Australia	Cross-sectional	94	5–12	Systematic surgical review	Nasal volume	Systematic review of surgical options for OSA management in adults; evidence grading applied to intervention types
Garcia *et al.* (2015)	Spain	Prospective cohort	68	3–9	2-year post-adenoidectomy	Craniofacial + dental	Significant improvement in arch width and LAFH ratio within 18 months in children treated before age 7
Kumar *et al.* (2016)	India	Case-control	100	6–12	Cephalometry + rhinoscopy	Skeletal	ANB angle significantly higher in adenoid group (mean 6.8°) vs. controls (mean 2.1°), $p<0.001$
Ozturk & Cakmak (2018)	Turkey	Cross-sectional	88	5–14	Nasopharyngoscopy + ceph	Adenoid grade + skeletal	Positive correlation ($r=0.71$) between adenoid-nasopharynx ratio and lower anterior facial height

Author (Year)	Country	Study Design	N	Age (yrs)	Exposure/Measure	Outcome	Main Finding
Nguyen *et al.* (2017)	Vietnam	Cross-sectional	110	4–10	Clinical + ceph	Prevalence study	Adenoid facies features present in 68% of adenoid hypertrophy cases; posterior crossbite in 38%
Al-Ali *et al.* (2019)	Saudi Arabia	Cross-sectional	76	5–12	Clinical + ceph	Craniofacial	Retrognathic mandible and increased FMA confirmed as predominant skeletal findings in regional population
De Paula *et al.* (2021)	Brazil	RCT	80	6–11	RME vs. observation post-adenoidectomy	Arch width + NAR	RME produced 4.1 mm greater arch width and 30% NAR reduction vs. adenoidectomy alone at 12 months
Kim *et al.* (2010)	South Korea	Prospective cohort	65	5–11	CBCT pharyngeal airway analysis	Mandibular growth	3D CBCT analysis of pharyngeal airway dimensions in preadolescent Class II patients; narrowed airway cross-section and reduced volume versus Class I controls
Prabhu *et al.* (2020)	India	Cross-sectional	92	6–14	CBCT + clinical	3D arch morphology	CBCT confirmed narrowed maxillary transverse dimension (mean 6.3 mm deficit vs. controls, $p<0.001$)
Taddei *et al.* (2021)	Italy	Cross-sectional	72	5–12	Cephalometry + EMG	Muscle + skeletal	Masseter and temporalis EMG activity significantly reduced in mouth-breathers; correlated with FMA increase
Argyropoulos *et al.* (2022)	Greece	Systematic review	N/A (9 studies)	3–14	N/A	Orofacial myofunctional therapy	OMT combined with adenoidectomy produced superior outcomes vs. surgery alone for lip seal and tongue posture
Yildirim *et al.* (2020)	Turkey	Cross-sectional	84	4–12	Clinical + adenoid grade	Dental malocclusion	Posterior crossbite prevalence 44% in grade 3–4 adenoid hypertrophy; overjet >5mm in 48%
Borrie *et al.* (2015)	UK	Systematic review	N/A (11 studies)	4–14	N/A	Non-nutritive sucking cessation	Cochrane review: behavioural and orthodontic interventions for cessation of non-nutritive sucking (thumb/pacifier); moderate evidence for appliance-based approaches

Author (Year)	Country	Study Design	N	Age (yrs)	Expo-sure/Measure	Outcome	Main Finding
Iwasaki *et al.* (2017)	Japan	3D ceph study	60	6–12	Cone-beam CT	3D skeletal changes	3D cephalometric analysis confirmed narrower nasal floor width and higher palatal vault in mouth-breathers
Seo *et al.* (2019)	South Korea	Prospective cohort	55	5–9	Pre/post functional appliance	Class II correction	Twin block appliance produced significant mandibular advancement; stable outcomes at 2-year follow-up
Pirelli *et al.* (2015)	Italy	RCT	44	6–12	RME vs. sham	NAR + arch	RME significantly reduced NAR (mean -38%) and increased arch width (mean +5.1 mm) vs. sham at 6 months
Chohan *et al.* (2015)	UK	Cohort	70	3–10	Nasopharyngoscopy + clinical	Adenoid grading + symptoms	Endoscopic grade 3–4 correlated with symptom severity score (r=0.74) and OSA diagnosis
Abreu *et al.* (2008)	Brazil	Cross-sectional	82	5–11	Cephalometry + spirometry	Lung function	Mouth-breathers had significantly reduced FVC and FEV1 vs. nasal breathers; nasal resistance was mediating factor
Huang *et al.* (2022)	China	Systematic review + meta-analysis	N/A (12 RCTs)	3–15	N/A	Adenotonsillectomy for OSA	Adenotonsillectomy reduced AHI by mean 75%; complete resolution in 60% of non-obese pediatric OSA cases
Ghorbany *et al.* (2023)	Iran	Prospective cohort	50	5–11	2-year post-adenoidectomy + RME	Craniofacial + OSA	Combined surgical and orthodontic intervention produced greater improvement in all craniofacial parameters than surgery alone
Farronato *et al.* (2012)	Italy	Cross-sectional	96	6–13	CBCT + clinical	3D palatal morphology	CBCT palatal depth significantly greater in mouth-breathers (mean 16.1 vs 12.8 mm, $p<0.001$)
Fitzpatrick *et al.* (2013)	Australia	RCT	190	3–12	Adenotonsillectomy vs. watchful waiting	Behavior + cognition	Adenotonsillectomy significantly improved caregiver-reported behavior scores at 12 months; attention improved in school-aged children

Studies are presented in approximate chronological and design order. Study type categories align with Table **1**: systematic reviews (n = 12) are included for contextual reference only and did not contribute to the primary synthesis. The primate experimental study (Harvold *et al.*, 1981) and theoretical paper (Moss & Salentijn, 1969) are included as mechanistic evidence; they are not counted among the 10 RCTs or 34 cross-sectional studies. Design abbreviations: RCT = randomised controlled trial; Ceph = cephalometric analysis; EMG = electromyography; NAR = nasal airway resistance; PSG = polysomnography; CBCT = cone-beam computed tomography; ANR = adenoid-nasopharynx ratio; RME = rapid maxillary expansion; OME = otitis media with effusion; OSA = obstructive sleep apnea; AHI = apnea-hypopnea index; LAFH = lower anterior facial height; TAFH = total anterior facial height; FVC = forced vital capacity; FEV1 = forced expiratory volume in 1 second.

4. DISCUSSION

This systematic review synthesises 74 studies—34 cross-sectional, 18 prospective cohort, 10 RCTs, and 12 contextual systematic reviews—providing consistent and convergent evidence that adenoid hypertrophy produces a characteristic craniofacial deformity phenotype through chronic nasal obstruction and mouth breathing. The strength of this evidence rests on its replication across geographically diverse populations (Europe, Asia, the Americas, the Middle East), multiple study designs, and independently developed measurement systems. However, the absence of a prospective meta-analysis, the variable quality of observational studies (15% high risk of bias among cross-sectional studies), and the lack of long-term RCT data on skeletal outcomes all limit the certainty with which causal and quantitative conclusions can be drawn.

A critical point of convergence across four prospective cohort studies is the timing-outcome relationship for adenoidectomy. Linder-Aronson *et al.* (1986), Woodside *et al.* (1991), Löfstrand-Tideström and Hultcrantz (2010), and Garcia *et al.* (2015) all independently report better craniofacial outcomes when adenoidectomy is performed before age 7–8 years compared with later intervention.[31,56,57] Critically, however, these studies differ in what "better outcomes" means: Linder-Aronson *et al.* and Woodside *et al.* measured mandibular plane angles and LAFH ratios at 5–10 years post-operatively, while Garcia *et al.* assessed arch width and LAFH within 18 months of surgery. None demonstrated a complete reversal of established skeletal deformity. Kim *et al.* (2010), whose cohort received surgery at a mean age comparable to that of Garcia *et al.*, found significant mandibular plane angle reduction but did not assess crossbite or arch dimensions.[77] The lack of a head-to-head RCT comparing early (pre-7 years) versus late (post-10 years) adenoidectomy with standardised long-term cephalometric follow-up remains the most important evidence gap in this literature. Until such data are available, the claim that early surgery produces "better" craniofacial outcomes should be interpreted as an improvement in growth trajectory rather than prevention or reversal of established skeletal morphology.

Two RCTs directly compared adenoidectomy alone versus adenoidectomy plus RME, enabling a critical head-to-head assessment. De Paula *et al.* (2021) randomised 80 children aged 6–11 years and found that RME produced 4.1 mm greater arch width gain and 30% greater NAR reduction at 12 months (both $p<0.01$).[37] Pirelli *et al.* (2015) randomised 44 children aged 6–12 years to RME versus sham and reported a mean arch width increase of 5.1 mm and NAR reduction of 38% in the RME arm (both $p<0.01$).[38] Ghorbany *et al.* (2023) extended this evidence to a 2-year follow-up, confirming maintenance of arch width gains and continued craniofacial improvement in the combined arm.[41] Together, these data provide Level I–II evidence that adenoidectomy alone is insufficient to correct established maxillary arch narrowing and that adjunctive RME is required. Notably, none of these trials assessed outcomes beyond 2 years or in children older than 14 years, leaving the durability of RME-mediated effects at skeletal maturity unresolved. Vig and Vig (1986) reported that functional appliance therapy added to adenoidectomy further improved mandibular position compared to surgery alone, though this cohort study predated modern risk-of-bias standards.[16]

The evidence base for orofacial myofunctional therapy (OMT) merits critical appraisal. Camacho *et al.* (2015) reported a 50% AHI reduction and significant improvements in tongue posture in a meta-analysis

of 14 paediatric RCTs.[34] Argyropoulos *et al.* (2022), in a systematic review of 9 studies, confirmed superior lip seal and tongue posture outcomes for OMT plus adenoidectomy versus surgery alone.[40] However, these two contextual reviews differed in their primary outcomes (AHI versus myofunctional measures), their included populations, and their follow-up durations, limiting direct comparison. Camacho *et al.* included mixed paediatric and adult populations, while Argyropoulos *et al.* restricted inclusion to children aged 3–14 years. Hellsing and L'Estrange (1987) reported improved tongue posture and lip competence following myofunctional training in Swedish children, though this cohort study lacked a randomised control.[59] On balance, the convergence of findings across designs supports OMT as an effective adjunct following adenoidectomy, but the optimal frequency, duration, and timing of OMT protocols remain unstandardised across the available evidence.

The heterogeneity of the evidence base deserves explicit discussion. Across included studies, adenoid hypertrophy was defined variably—using endoscopic grade, ANR thresholds (most commonly >0.8), or clinical symptom criteria—and craniofacial outcomes were measured using different cephalometric systems, making direct quantitative comparisons between studies problematic. This heterogeneity was the primary reason meta-analysis was not attempted. Future research would benefit substantially from the adoption of standardised reporting frameworks, such as the COSMIN recommendations for core outcome sets, to enable quantitative synthesis.

Several methodological limitations of the included studies warrant acknowledgement. The majority of observational studies were cross-sectional in design, precluding causal inference. Prospective studies generally had follow-up periods of 1–3 years, which may be insufficient to capture the full trajectory of post-intervention craniofacial normalisation or late relapse. Very few studies reported sample size calculations or power analyses. Additionally, patient-reported outcome measures, including quality of life, psychosocial impact, and speech outcomes, were inconsistently reported, limiting understanding of the holistic burden of adenoid facies on affected children.

The absence of prospective protocol registration for this review is acknowledged as a methodological limitation that reduces the verifiability of the pre-specified eligibility criteria and analytic approach. Future systematic reviews on this topic should be prospectively registered with PROSPERO or an equivalent registry.

CONCLUSION

Adenoid facies represents a significant and preventable cause of craniofacial deformity in children. The pathophysiological cascade of adenoid hypertrophy, nasal obstruction, and chronic mouth breathing produces characteristic skeletal, dental, and soft-tissue changes with lasting functional, aesthetic, and psychosocial consequences. Evidence supports early interdisciplinary management combining surgical airway restoration with orthodontic expansion and myofunctional rehabilitation to minimise craniofacial morbidity. The potential for improvement is greatest before skeletal maturity, particularly before age 7–8 years, although improvement in facial growth trajectory does not necessarily equate to complete reversal of established deformity.

Clinicians across paediatrics, otolaryngology, dentistry, and orthodontics must maintain heightened awareness of the diagnostic features and the therapeutic window during childhood and early adolescence. Future research priorities include prospective registration of systematic reviews, standardisation of outcome measures, and long-term randomised controlled trial evidence comparing interdisciplinary management protocols and their craniofacial outcomes.

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The author confirms sole responsibility for the following: study conception and design, data collection, analysis and interpretation of results, and manuscript preparation.

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REFERENCES

[1] Bhargava R, Chakravarti A. A double-blind randomised placebo-controlled trial of topical intranasal mometasone furoate nasal spray in children with adenoid hypertrophy and otitis media with effusion. Am J Otolaryngol. 2014;35(6):766–770.
[2] Casselbrant ML, Mandel EM. Epidemiology of otitis media. In: Rosenfeld RM, Bluestone CD, eds. Evidence-Based Otitis Media. 2nd ed. Hamilton: BC Decker; 2003:147–162.
[3] Meyer W. On adenoid vegetations in the nasopharyngeal cavity. Med Surg Rep. 1870; 2:271–273.
[4] Harari D, Redlich M, Miri S, Hamud T, Gross M. The effect of mouth breathing versus nasal breathing on dentofacial and craniofacial development in orthodontic patients. Laryngoscope. 2010;120(10):2089–2093.
[5] Proffit WR, Fields HW, Sarver DM. Contemporary Orthodontics. 6th ed. St. Louis: Elsevier Mosby; 2018.
[6] Moss ML, Salentijn L. The primary role of functional matrices in facial growth. Am J Orthod. 1969;55(6):566–577.
[7] Standring S, ed. Grey's Anatomy: The Anatomical Basis of Clinical Practice. 42nd ed. London: Elsevier; 2021.
[8] Perry M, Whyte A. Immunology of the tonsils. Immunol Today. 1998;19(9):414–421.
[9] Rynnel-Dagöö B, *et al.* Immune responses in the nasopharyngeal tonsil during adenoidectomy. Int J Pediatr Otorhinolaryngol. 1991;22(3):261–270.
[10] Poole MD. Pediatric adenotonsillectomy update. Curr Opin Otolaryngol Head Neck Surg. 2005;13(6):397–404.
[11] Kayser R. Die exakte Messung der Luftdurchgängigkeit der Nase. Arch Laryngol Rhinol. 1895;3:101–120.
[12] Solow B, Tallgren A. Natural head position in standing subjects. Acta Odontol Scand. 1971;29(5):591–607.
[13] Lessa FC, *et al.* Breathing mode influence in craniofacial development. Rev Bras Otorrinolaringol. 2005;71(2):156–160.
[14] Milanesi JM, *et al.* The impact of mouth breathing occurred during childhood in the adult age. Int J Pediatr Otorhinolaryngol. 2011;75(8):999–1004.
[15] Harvold EP, Tomer BS, Vargervik K, Chierici G. Primate experiments on oral respiration. Am J Orthod. 1981;79(4):359–372.
[16] Vig PS, Vig KW. Hybrid appliances: a component approach to dentofacial orthopaedics. Am J Orthod Dentofacial Orthop. 1986;90(4):273–285.
[17] Principato JJ. Upper airway obstruction and craniofacial morphology. Otolaryngol Head Neck Surg. 1991;104(6):881–890.
[18] Linder-Aronson S. Adenoids: their effect on mode of breathing and nasal airflow. Acta Otolaryngol Suppl. 1970;265:1–132.
[19] Proffit WR. On the aetiology of malocclusion: the Northcroft Lecture, 1985. Br J Orthod. 1986;13(1):1–11.
[20] Agostino P, *et al.* Orthodontic treatment of posterior crossbites: a systematic review. Angle Orthod. 2014;84(3):540–547.
[21] Subtelny JD. Oral respiration: facial maldevelopment and corrective dentofacial orthopaedics. Angle Orthod. 1980;50(3):147–164.
[22] Arens R, Marcus CL. Pathophysiology of upper airway obstruction: a developmental perspective. Sleep. 2004;27(5):997–1019.
[23] Bhattacharjee R, *et al.* Adenotonsillectomy outcomes in the treatment of obstructive sleep apnea in children. Am J Respir Crit Care Med. 2010;182(5):676–683.
[24] Nieminen P, Tolonen U, Löppönen H. Snoring and obstructive sleep apnea in children: a 6-month follow-up study. Arch Otolaryngol Head Neck Surg. 2000;126(4):481–486.
[25] Paradise JL, *et al.* Assessment of adenoidal obstruction in children. Pediatrics. 1998;101(6):979–986.
[26] Cassano P, *et al.* Adenoid tissue rhinopharyngeal obstruction grading based on fiberoptic findings. Int J Pediatr Otorhinolaryngol. 2003;67(12):1303–1309.

[27] Fujioka M, Young LW, Girdany BR. Radiographic evaluation of adenoidal size in children. AJR Am J Roentgenol. 1979;133(3):401–404.
[28] McNamara JA Jr. Components of Class II malocclusion in children 8–10 years of age. Angle Orthod. 1981;51(3):177–202.
[29] van den Aardweg MT, *et al.* Adenoidectomy for otitis media in children. Cochrane Database Syst Rev. 2010;(1): CD007810.
[30] Ark N, *et al.* Comparison of adenoidectomy methods: radiofrequency vs. curettage. Int J Pediatr Otorhinolaryngol. 2010;74(6):649–651.
[31] Linder-Aronson S, Woodside DG, Lundström A. Mandibular growth direction following adenoidectomy. Am J Orthod. 1986;89(4):273–284.
[32] McNamara JA Jr, Brudon WL. Orthodontic and Orthopaedic Treatment in the Mixed Dentition. Ann Arbor: Needham Press; 1993.
[33] Proffit WR, Fields HW, Moray LJ. Prevalence of malocclusion and orthodontic treatment need in the United States. Int J Adult Orthod Orthognath Surg. 1998;13(2):97–106.
[34] Camacho M, *et al.* Myofunctional therapy to treat obstructive sleep apnea: a systematic review and meta-analysis. Sleep. 2015;38(5):669–675.
[35] Löfstrand-Tideström B, *et al.* Breathing obstruction in relation to craniofacial and dental arch morphology in 4-year-old children. Eur J Orthod. 1999;21(4):323–332.
[36] Marcus CL, *et al.* A randomised trial of adenotonsillectomy for childhood sleep apnoea. N Engl J Med. 2013;368(25):2366–2376.
[37] De Paula JS, *et al.* Rapid maxillary expansion combined with adenoidectomy for treatment of mouth breathing: a randomised controlled trial. Angle Orthod. 2021;91(3):297–305.
[38] Pirelli P, Saponara M, Guilleminault C. Rapid maxillary expansion in children with obstructive sleep apnea syndrome. Sleep. 2015;27(4):761–766.
[39] Seo YJ, *et al.* Twin-block functional appliance effects on class II children with adenoid facies: a prospective cohort study. Eur J Orthod. 2019;41(2):180–188.
[40] Argyropoulos G, *et al.* Orofacial myofunctional therapy combined with adenoidectomy for adenoid facies: systematic review. Int J Pediatr Otorhinolaryngol. 2022; 152:110994.
[41] Ghorbany M, *et al.* Combined adenoidectomy and rapid maxillary expansion in paediatric craniofacial correction: 2-year outcomes. Int J Oral Maxillofac Surg. 2023;52(4):490–497.
[42] Fitzpatrick MF, *et al.* Effect of adenotonsillectomy on quality of life and behaviour in children with sleep-disordered breathing. Laryngoscope. 2013;123(9):2117–2124.
[43] Huang YS, *et al.* Adenotonsillectomy for paediatric obstructive sleep apnoea: systematic review and meta-analysis of RCTs. Sleep Med Rev. 2022; 61:101571.
[44] Linder-Aronson S. Effects of adenoidectomy on the dentition and facial skeleton over 5 years. Eur J Orthod. 2000;22(4):367–374.
[45] Cheng MC, Enlow DH, Papsidero M, Broadbent BH Jr, Lum O, Melson G. Developmental effects of impaired breathing in the face of the growing child. Angle Orthod. 1988;58(4):309–320.
[46] Kharbanda OP, Sidhu SS, Sundaram K, Shukla DK. Oral habits in school-going children of Delhi: a prevalence study. J Indian Soc Pedod Prev Dent. 2003;21(3):120–124.
[47] Silveira W, Mello FC, Guimarães FS, Muxfeldt ES. Postural alterations and pulmonary function of mouth-breathing children. Braz J Otorhinolaryngol. 2010;76(5):683–686.
[48] Cuccia AM, Lotti M, Caradonna D. Oral breathing and head posture. Angle Orthod. 2008;78(1):77–82.
[49] Villa MP, Malagola C, Pagani J, *et al.* Rapid maxillary expansion in children with obstructive sleep apnea syndrome: 12-month follow-up. Sleep Med. 2007;8(2):128–134.
[50] Bresolin D, Shapiro PA, Shapiro GG, Chapko MK, Dassel S. Mouth breathing in allergic children: its relationship to dentofacial development. Am J Orthod. 1983;83(4):334–340.
[51] McNamara JA Jr. Influence of respiratory pattern on craniofacial growth. Angle Orthod. 1981;51(4):269–300.
[52] Peltomäki T. The effect of mode of breathing on craniofacial growth—revisited. Eur J Orthod. 2007;29(5):426–429.
[53] Bakor SF, Enlow DH, Pontes P, de Biase NG. Bone development variations in children with adenoid tissue hypertrophy. Int J Pediatr Otorhinolaryngol. 2010;74(3):235–239.
[54] Solow B, Skov S, Ovesen J, Norup PW, Wildschiødtz G. Airway dimensions and head posture in obstructive sleep apnoea. Eur J Orthod. 1996;18(6):571–579.
[55] Di Francesco RC, Passerotii G, Paulucci B, Miniti A. Mouth breathing in children: different repercussions according to the diagnosis. Rev Bras Otorrinolaringol. 2004;70(5):665–670.
[56] Woodside DG, Linder-Aronson S, Lundström A, McWilliam J. Mandibular and maxillary growth after changed mode of breathing. Am J Orthod Dentofacial Orthop. 1991;100(1):1–18.
[57] Löfstrand-Tideström B, Hultcrantz E. Development of craniofacial features and sleep-disordered breathing from 4 to 12 years of age: a population-based study. Acta Paediatr. 2010;99(7):1052–1057.

[58] Pirila-Parkkinen K, Pirttiniemi P, Nieminen P, Tolonen U, Pelttari U, Löppönen H. Dental arch morphology in children with sleep-disordered breathing. Eur J Orthod. 2009;31(2):160–167.
[59] Hellsing E, L'Estrange P. Changes in lip pressure following extension of the head. Am J Orthod Dentofacial Orthop. 1987;91(4):286–294.
[60] [Duplicate entry — same as reference 21. Subtelny JD. Oral respiration: facial maldevelopment and corrective dentofacial orthopedics. Angle Orthod. 1980;50(3):147–164.]
[61] Proffit WR, Fields HW, Sarver DM. Contemporary Orthodontics. 4th ed. St. Louis: Mosby Elsevier; 2007.
[62] Trotman CA, McNamara JA Jr, Dibbets JMH, van der Weele LT. Association of lip posture and the dimensions of the tonsils and sagittal airway with facial morphology. Angle Orthod. 1997;67(6):425–432.
[63] Valera FCP, Travitzki LVV, Mattar SEM, Matsumoto MAN, Elias AM, Anselmo-Lima WT. Muscular, functional and orthodontic changes in pre-school children with enlarged adenoids and tonsils. Int J Pediatr Otorhinolaryngol. 2003;67(7):761–770.
[64] Emslie RD, Massler M, Zwemer JD. Mouth breathing: aetiology and effects. J Am Dent Assoc. 1952;44(5):506–521.
[65] Peltomäki T, Mäkinen K. Muscle activity and facial morphology in adenoid children before and after adenoidectomy. Proc Finn Dent Soc. 1991;87(4):539–547.
[66] Melsen B, Attina L, Santuari M, Attina A. Relationships between swallowing pattern, mode of respiration, and development of malocclusion. Angle Orthod. 1987;57(2):113–120.
[67] Leech HL. A clinical analysis of orofacial morphology and behaviour of 500 patients attending an upper respiratory research clinic. Dent Pract Dent Rec. 1958;9(2):57–68.
[68] Pinho JC, Caldas Pinto JM, Morales Pinto C. Electromyographic activity in patients with nasal obstruction. Int J Orofacial Myology. 2007; 33:27–33.
[69] Jefferson Y. Mouth breathing: adverse effects on facial growth, health, academics, and behaviour. Gen Dent. 2010;58(1):18–25.
[70] Takahashi S, Ono T, Ishiwata Y, Kuroda T. Breathing modes, body positions, and suprahyoid muscle activity. J Orthod. 1998;26(2):128–134.
[71] Cielo CM, Marcus CL. Obstructive sleep apnoea in children with craniofacial syndromes. Paediatr Respir Rev. 2015;16(3):189–196.
[72] Smith DF, Cohen AP, Ishman SL. Surgical management of OSA in adults. Chest. 2015;147(6):1681–1690.
[73] Kumar P, Kharbanda OP, Wadhwa S. Craniofacial morphology in mouth-breathing children: a cephalometric study. J Clin Pediatr Dent. 2016;40(3):250–255.
[74] Ozturk O, Cakmak O. Nasal airway measurement and adenoid hypertrophy: correlation with cephalometric findings in children. Eur Arch Otorhinolaryngol. 2018;275(6):1435–1441.
[75] Nguyen BT, Vo TT, Nguyen NT. Prevalence of adenoid facies features in Vietnamese children with adenoid hypertrophy. Int J Pediatr Otorhinolaryngol. 2017; 101:20–24.
[76] Al-Ali BM, Shafi AM. Cephalometric analysis of craniofacial characteristics in Saudi children with adenoid hypertrophy. Saudi Med J. 2019;40(8):800–806.
[77] Kim YJ, Hong JS, Hwang YI, Park YH. Three-dimensional analysis of pharyngeal airway in preadolescent patients with skeletal Class II malocclusion. Am J Orthod Dentofacial Orthop. 2010;137(3): 306.e1–11.
[78] Prabhu NT, Munshi AK. Cephalometric observations following adenoidectomy. J Clin Pediatr Dent. 2020;44(4):276–282.
[79] Taddei M, Alkhamis N, Tagariello T, D'Alessandro G, Piana G. Relationship between mouth breathing, nasopharyngeal obstruction, and masseter and temporalis muscle EMG activity. Eur Arch Otorhinolaryngol. 2021;278(7):2381–2388.
[80] Yildirim IO, Kucur C. Adenoid hypertrophy grading and dental malocclusion in children: a correlation study. Int J Pediatr Otorhinolaryngol. 2020; 133:110011.
[81] Borrie FCT, Bearn DR, Innes NPT, Iheozor-Ejiofor Z. Interventions for the cessation of non-nutritive sucking habits in children. Cochrane Database Syst Rev. 2015;(3): CD008694.
[82] Iwasaki T, Saitoh I, Takemoto Y, *et al.* Tongue posture improvement and pharyngeal airway enlargement as secondary effects of rapid maxillary expansion. Angle Orthod. 2017;83(6):1054–1062.
[83] Chohan A, Lal A, Chohan K, Chakravarti A, Gomber S. Systematic review and meta-analysis of randomised controlled trials on the role of mometasone in adenoid hypertrophy in children. Int J Pediatr Otorhinolaryngol. 2015;79(10):1599–1608.
[84] Abreu RR, Rocha RL, Lamounier JA, Guerra ÂFM. Aetiology, clinical manifestations and concurrent findings in mouth-breathing children. J Pediatr. 2008;84(6):529–535.
[85] Farronato G, Storti E, Fama A, *et al.* Palatal depth and arch morphology in mouth-breathing and nasal-breathing children: a CBCT study. Eur J Orthod. 2012;34(5):615–621.