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Review Article

Orofacial myofunctional therapy for persistent pediatric obstructive sleep apnea after adenotonsillectomy: evidence limits and patient selection

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Abstract

Background: Adenotonsillectomy is a first-line intervention for many children with obstructive sleep apnea (OSA), yet residual or recurrent disease may persist after surgery. Mouth breathing and orofacial myofunctional disorders are frequently discussed as modifiable functional features, but their relationship to objective sleep outcomes remains difficult to interpret. Objective: This review maps the evidence for active orofacial myofunctional therapy (OMT) as an adjunctive strategy in children with persistent OSA or sleep-disordered breathing after adenotonsillectomy and defines limits for patient selection and outcome assessment. Methods: A structured critical review and evidence-mapping approach was applied to 30 open, verifiable sources published from 2006 to 2026. The corpus included clinical guidelines, consensus statements, outcome studies after adenotonsillectomy, studies of orofacial motor function, four pediatric interventional studies of OMT, systematic reviews, one study protocol, a trial registry record, and methodological guidance. No meta-analysis was conducted because pediatric interventions, populations, diagnostic thresholds, and follow-up periods were heterogeneous. Results: The most consistent signal concerns short-term changes in orofacial function, mouth or nasal breathing patterns, selected symptoms, and quality-of-life measures. Certainty is lower for polysomnographic resolution of OSA, particularly changes in the apnea-hypopnea index. Active therapy, home exercise programs, passive myofunctional devices, orthodontic interventions, and general breathing training should not be pooled as a single exposure. Conclusions: OMT is best positioned as an investigational adjunct for carefully selected children with objectively confirmed persistent OSA, adequate nasal airway assessment, documented orofacial dysfunction, and measurable adherence. It should not replace repeat polysomnography or standard treatment of residual anatomical obstruction.

Keywords: pediatric obstructive sleep apnea, persistent OSA, adenotonsillectomy, orofacial myofunctional therapy, mouth breathing, orofacial dysfunction, evidence mapping, upper airway rehabilitation

1. Introduction

Adenotonsillectomy (AT) remains the central surgical intervention for pediatric obstructive sleep apnea (OSA) associated with adenotonsillar hypertrophy, but surgery does not end disease management for every child. The American Thoracic Society defines persistent post-adenotonsillectomy OSA as an obstructive apnea-hypopnea index (oAHI) of at least one event per hour after surgery and emphasizes that residual disease is common enough to require structured management rather than informal reassurance [1]. Other pediatric sleep and otolaryngology guidelines similarly frame postoperative assessment as a continuing clinical problem, not merely as a binary surgical success or failure [2-6].

The incomplete normalization of sleep breathing after AT is not a new observation. Cohort and multicenter studies, together with randomized evidence on surgical treatment, showed that some children continue to have abnormal polysomnographic or symptomatic outcomes after removal of the adenoids and tonsils [7-10]. Long-term follow-up further suggests that sleep-disordered breathing may persist or reappear after an initially favorable postoperative course [11]. These findings have shifted the field from a narrow anatomic model toward phenotype-oriented evaluation of residual disease.

Mouth breathing and orofacial myofunctional disorders occupy a clinically attractive but methodologically unstable position in this debate. Open-mouth posture, altered tongue position, and atypical swallowing or chewing patterns are recognized features within the professional domain of orofacial myofunctional disorders [12]. Observational studies in children with OSA have also identified measurable abnormalities of orofacial motor function [13,14]. Such findings make a functional rehabilitation hypothesis plausible, but they do not establish that mouth breathing causes pediatric OSA or that correcting it necessarily normalizes sleep respiration.

Active orofacial myofunctional therapy (OMT) has been proposed as a structured set of exercises targeting the tongue, lips, cheeks, soft palate, and stomatognathic functions. Small pediatric studies have reported favorable changes in selected respiratory, functional, and symptomatic outcomes [15-18]. At the same time, evidence syntheses diverge in tone. Earlier meta-analytic work was comparatively optimistic [19,20], whereas the Cochrane review judged the pediatric evidence for apnea-hypopnea index (AHI) outcomes after AT to be of low certainty [21]. More recent systematic reviews broaden the corpus but remain limited by mixed adult-pediatric samples, heterogeneous interventions, and recurrent use of the same small primary studies [22-25].

The purpose of this article is therefore not to reframe OMT as a proven cure for pediatric OSA. The aim is more modest and more useful for otolaryngology: to map the clinical and evidential limits within which active OMT can be considered as an adjunctive intervention for children with objectively confirmed persistent OSA after AT, particularly when mouth breathing and orofacial dysfunction remain after anatomical assessment. The review asks four linked questions: which outcome domains show the most consistent signal; which child-level and intervention-level features may modify response; why active exercises, passive devices, orthodontic treatment, and general breathing training should be separated; and what minimum outcome set is needed for future trials.

The contribution of the article is an analytical framework rather than a new efficacy estimate. It separates structural and functional components of persistent OSA, proposes a taxonomy of interventions, outlines a potentially modifiable functional phenotype, and offers a minimum outcome set for future research. This structure is intended to reduce two common interpretive errors: treating mouth breathing as a surrogate diagnosis of OSA and treating all interventions labeled myofunctional as equivalent.

Terminological precision is especially important because the clinical pathway involves several specialties. In otolaryngology, residual obstruction is usually approached through anatomical localization and disease severity. In speech-language pathology and dental sleep medicine, the same child may be described through lip posture, tongue resting position, swallowing pattern, dental arch development, or habitual breathing route. These descriptions are complementary only when the diagnostic hierarchy is preserved. OSA remains a sleep-related breathing disorder that requires objective assessment; mouth breathing and orofacial dysfunction are possible associated features and treatment targets, not diagnostic substitutes.

2. Methods

This article was designed as a structured critical review with evidence mapping and comparative analysis of open clinical guidance documents. The design was selected instead of a meta-analysis because the available pediatric interventional studies are few, heterogeneous, and not sufficiently comparable in population, diagnostic threshold, intervention dose, control condition, and follow-up period. The review was also not presented as a full systematic review because a complete reproducible search log, duplicate screening record, and PRISMA flow count were not generated. Methodological guidance for scoping and systematic review reporting was used only to structure eligibility, charting, and transparent interpretation [28-30].

The search and verification process was fixed on 17 July 2026. Sources were identified from PubMed/MEDLINE, PubMed Central, Europe PMC, Cochrane Library, ClinicalTrials.gov, official professional society websites, publisher pages, and DOI records. The English search structure combined terms for OSA or sleep-disordered breathing, pediatric populations, OMT or oropharyngeal exercises, and postoperative or residual disease. Russian-language searching was used only to check whether an open comparable corpus existed for the same topic; no independent Russian pediatric interventional corpus with polysomnography-confirmed postoperative OSA was identified.

Eligible sources had to meet at least one of five functions: define the clinical problem of persistent pediatric OSA after AT; describe pediatric orofacial motor phenotype in OSA; report an active OMT or closely related pediatric intervention; synthesize evidence on OMT; or provide methodological or registry context for future studies. Primary interventional evidence was restricted to children or separately extractable pediatric samples. Studies focused solely on adults, isolated mouth breathing without sleep outcomes, commercial exercise programs, case reports, nonverifiable bibliographic records, and passive devices without separable analysis of active therapy were excluded from evidential claims about active OMT.

The final corpus contained 30 source units. These included six clinical guidelines or consensus documents, one professional practice resource on orofacial myofunctional disorders, five sources on persistent OSA and outcomes after AT, two observational studies of orofacial motor function, four pediatric interventional studies, seven evidence syntheses, one published protocol, one clinical trial registry record, and three methodological sources. Each source was assigned to one analytical function: clinical framing, phenotype description, intervention evidence, evidence synthesis, prospective research design, or methodological reporting.

Data were extracted descriptively rather than statistically. For interventional studies, the charting fields were country or setting, design, age range, sample size, OSA or sleep-disordered breathing definition, postoperative status, intervention type, dose and duration, control condition, adherence assessment, polysomnographic outcomes, oxygenation outcomes, orofacial functional outcomes, symptoms, quality-of-life instruments, follow-up duration, and major limitations. For guidelines and consensus documents, the extracted units were definitions, diagnostic standards, postoperative management recommendations, and statements relevant to residual disease or functional assessment.

Interpretation followed three rules. First, polysomnographic outcomes were not combined with daytime functional outcomes. A change in mouth breathing, open-mouth posture, tongue tone, or a myofunctional score was treated as a functional outcome, not as proof of OSA resolution. Second, intervention categories were separated by mechanism and patient activity. Active therapist-guided OMT, home exercises, passive devices, orthodontic interventions, inspiratory muscle training, and multi-component rehabilitation were not pooled. Third, trial registry entries and published protocols were used as evidence of an emerging research agenda, not as evidence of therapeutic effectiveness.

The strength of support was judged qualitatively according to source type and directness. Clinical guidelines were treated as high-value sources for definitions, diagnostic sequencing, and boundaries of standard care.

Primary pediatric interventional studies were treated as the only direct sources for intervention signals, but their conclusions were constrained by sample size, design, and comparability. Systematic reviews were used to locate agreement and disagreement across the field, not as independent confirmations when they relied on overlapping primary studies. Registry records and protocols were included only to identify future research directions.

3. Results

3.1. Corpus structure and intervention taxonomy

The mapped corpus confirmed a wide gap between clinical interest in functional rehabilitation and the maturity of the pediatric OMT evidence base. Clinical documents provide a strong framework for recognizing persistent OSA after AT and for reassessing residual obstruction [1-6]. Observational studies support the relevance of orofacial function in children with OSA [13,14]. In contrast, direct pediatric intervention evidence remains concentrated in four small studies with differing designs and outcome sets [15-18].

The first analytical result is the taxonomy shown in Table 1. The word myofunctional is used inconsistently across the literature. Some papers refer to active exercises delivered or supervised by trained therapists, whereas others discuss passive appliances, orthodontic treatment, or broader breathing and rehabilitation programs. Treating these as a single exposure would inflate the evidence base while obscuring the mechanism by which benefit is supposed to occur.

The taxonomy also protects against a common error in literature synthesis: increasing apparent sample size by merging interventions with incompatible mechanisms. A passive device may alter mandibular or tongue posture during use without training a child to maintain a different resting posture when the device is absent. Orthodontic expansion can change airway dimensions or dentofacial constraints, whereas active OMT is expected to influence neuromuscular behavior and functional coordination. A review that combines these categories can produce a more confident-looking conclusion while making it less clear what should actually be tested or prescribed.

Table 1. Analytical taxonomy of interventions related to orofacial function.

Code	Category	Defining feature	Rule for interpretation
T1	Active therapist-guided OMT	Repeated exercises for tongue, lips, cheeks, soft palate, and related stomatognathic functions with professional feedback.	Primary category for evaluating active myofunctional therapy.
T2	Home exercise program with periodic supervision	Exercises performed independently with less frequent face-to-face or remote monitoring.	Analyze separately by actual dose and adherence.
T3	Passive myofunctional device	Intraoral appliance or trainer acting primarily through mechanical positioning or passive neuromuscular prompting.	Do not pool with active OMT unless the effect of exercises is separable.
T4	Orthodontic or dentofacial treatment plus OMT	Expansion, occlusal, or dentofacial intervention combined with functional exercises.	Treat as multi-component care; avoid attributing the total effect to OMT.
T5	Inspiratory or general breathing training	Training directed at respiratory muscles or breathing pattern without a defined orofacial exercise protocol.	Do not treat as equivalent to OMT.
T6	Multi-component rehabilitation	Program combining several therapeutic targets, often with lifestyle or behavioral components.	Use for context unless the independent OMT contribution is extractable.

The categories separate mechanism and patient activity. They are not an efficacy ranking.

3.2. Evidence gradient across outcome domains

The second result is an evidence gradient rather than a uniform positive or negative finding. Across the pediatric interventional studies, functional and selected symptomatic outcomes were reported more consistently than durable polysomnographic resolution. Guilleminault and colleagues reported a favorable long-term signal among children who underwent myofascial reeducation in the course of treatment, but the retrospective design and treatment sequence make it difficult to isolate OMT from adherence, prior orthodontic or surgical care, and family-level factors [15]. Villa and colleagues tested oropharyngeal exercises after AT in a small pediatric sample; the study is central to the topic because it addresses the postoperative setting, yet the Cochrane synthesis judged the certainty of pediatric AHI evidence from this comparison as low [16,21].

Other pediatric work from the same group reported changes in tongue tone, mouth breathing, symptoms, and oxygenation-related measures in children with sleep-disordered breathing, but the diagnostic spectrum and short follow-up limit direct inference for persistent OSA after AT [17]. The most recent study of childhood residual OSA compared treated and untreated children and included polysomnographic, quality-of-life, OMES, and open-mouth outcomes; however, because the accessible information is limited to the abstract and the study is small and nonrandomized, it cannot close the efficacy question [18]. Table 2 summarizes how the key studies contribute to the map.

Table 2. Pediatric interventional evidence map for active orofacial myofunctional therapy.

Study	Population and design	OMT and duration	Sleep outcomes	Functional or symptomatic outcomes	Main limitation
Guilleminault et al. [15]	Children treated for sleep-disordered breathing; retrospective long-term comparison.	Myofascial reeducation within a broader treatment trajectory.	Favorable long-term signal, but the independent effect is not separable.	Supports plausibility of functional rehabilitation.	Self-selection, small sample, previous treatments, and adherence confounding.
Villa et al. 2015 [16]	Children with OSA symptoms after AT; small controlled pediatric study.	Oropharyngeal exercises for approximately 2 months.	AHI effect remains uncertain under critical appraisal [21].	Short-term symptom and functional signal.	Small sample, short duration, and wide uncertainty.
Villa et al. 2017 [17]	Children with sleep-disordered breathing; comparative intervention study.	Myofunctional therapy targeting tongue tone and related functions.	Physiological outcomes reported, but not a definitive postoperative OSA trial.	Improvement reported in tongue tone, mouth breathing, and symptoms.	Mixed SDB spectrum and short follow-up.
Ye et al. 2025 [18]	Children with residual OSA; treated and untreated groups.	Active OMT; details interpreted within abstract-access limits.	Included PSG outcomes; study design was nonrandomized.	Included OMES, OSA-18, and open-mouth outcomes.	Small nonrandomized design; extraction limited to open abstract.

Direction of evidence is shown qualitatively. The table does not report a pooled effect size.

The review-level literature explains why different conclusions can be drawn from the same small pediatric base. The early meta-analysis by Camacho and colleagues helped establish the hypothesis that myofunctional therapy may reduce AHI, but most of the quantitative weight came from mixed or adult data rather than children with residual postoperative disease [19]. Later reviews continued to support biological plausibility and possible clinical utility while also documenting heterogeneity in protocols, age groups, and outcome definitions [20,22-25]. The Cochrane review remains the most cautious source for pediatric postoperative AHI outcomes because it focused on comparative certainty rather than direction of reported change [21].

The evidence gradient is also shaped by timing. Short courses of exercises may plausibly change awareness, posture, and performance during daytime assessment before they produce a measurable and sustained reduction in upper-airway obstruction during sleep. Conversely, a child may show modest improvement in AHI while still having mouth breathing or low tongue endurance that could matter for long-term stability. For that reason, endpoint selection should match the hypothesized mechanism. A trial designed to test symptom relief, functional rehabilitation, or polysomnographic disease control should not use the same primary endpoint without justification.

This gradient is important for interpretation. Functional gains may matter clinically, especially if they improve nasal breathing habit, lip closure, tongue posture, swallowing coordination, or child and parent-reported symptoms. Nevertheless, those gains cannot be treated as equivalent to normalization of oAHI, oxygen desaturation index, or minimum oxygen saturation during sleep. The evidence is therefore strongest for defining a research target and weakest for claiming a generalized treatment effect.

3.3. Functional phenotype and structural-functional pathway

The third result is a proposed phenotype for future study and cautious clinical discussion. The candidate child is not defined by mouth breathing alone. The entry point is objectively confirmed persistent OSA after AT. After that confirmation, structural and functional factors should be evaluated separately. OMT becomes a plausible adjunct only if nasal airway adequacy has been assessed, a dominant untreated anatomical obstruction is not the primary explanatory factor, and an orofacial functional problem is documented.

Figure 1 presents this structural-functional pathway. The model integrates guideline-based postoperative reassessment [1-6], the professional requirement that nasal breathing capacity be available before OMT is expected to succeed [12], and observational evidence that children with OSA may have measurable orofacial motor impairments [13,14]. It also incorporates the practical lesson from interventional studies: adherence and program dose cannot be treated as administrative details because they may influence both exposure and apparent response [15-18].

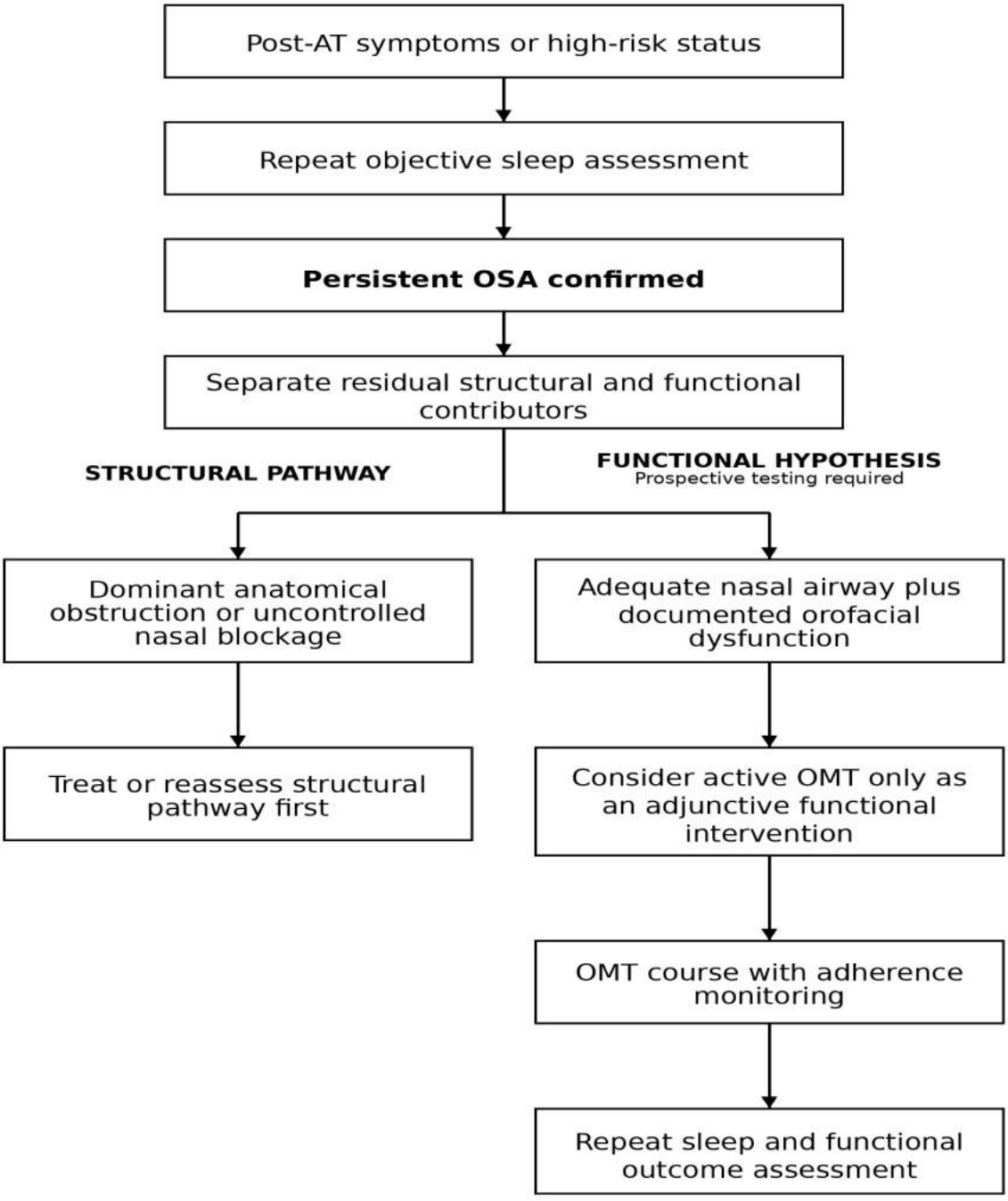

Figure 1. Structural-functional model for positioning OMT after adenotonsillectomy.

The model is a research framework. Solid steps reflect accepted clinical sequencing; the functional branch is a hypothesis that requires prospective testing.

Four variables should therefore be reported in every future pediatric OMT study. First, nasal airway status must be assessed because exercises intended to support nasal breathing cannot compensate for a mechanical nasal obstacle. Second, residual structural obstruction, including lingual tonsil hypertrophy or dentofacial restriction, should be described before a functional intervention is interpreted. Third, the orofacial phenotype

should be operationalized using an observed breathing pattern, lip and tongue posture, and, when feasible, a structured tool such as OMES or instrumental tongue-pressure measurement. Fourth, adherence should be quantified because children who complete therapy may differ systematically from those who do not.

The age boundary deserves particular caution. The lower working threshold of approximately four years is not a biological cut-off; it reflects the practical requirement that a child understand instructions, repeat exercises, and participate in self-monitoring with family support. Adolescents may complete exercises more reliably, but they may also have longer-standing oral habits, orthodontic histories, or obesity-related contributors. Future studies should therefore report developmental readiness and family-supported adherence rather than rely on chronological age alone.

These variables are candidate modifiers, not validated predictors. The model should not be converted into a scoring system or an automatic indication for therapy. Its value lies in forcing investigators to specify whether they are studying residual airway collapse, persistent habit, or a mixed structural-functional pattern.

3.4. Minimum outcome set for future studies

The fourth result is a minimum outcome set intended to make future studies comparable. Current evidence is difficult to synthesize because the outcome domains are not consistently separated. Some studies emphasize AHI or oxygenation, others emphasize mouth posture or tongue strength, and others emphasize symptoms or quality of life. Without a shared core, a trial can appear positive by improving a functional marker while leaving the sleep disorder unresolved.

Table 3 proposes a minimum set that separates disease activity, function, patient-centered outcomes, and treatment process. The set is not a formal consensus statement. It is an analytical recommendation derived from the mapped gaps in the pediatric interventional studies, the outcomes listed in newer protocols, and the diagnostic priorities of clinical guidelines [1,16-18,21,24-26].

Table 3. Proposed minimum outcome set for studies of OMT in persistent pediatric OSA.

Domain	Required measures	Timing	Interpretive rule
Objective sleep breathing	oAHI or AHI; oxygen desaturation index; mean and minimum SpO2; proportion below predefined oAHI thresholds.	Baseline; end of treatment; 6-month follow-up; preferably 12-month follow-up.	Polysomnographic response is evaluated separately from symptom or functional response.
Orofacial function	Predefined mouth-breathing criterion; lip and tongue posture at rest; OMES or comparable structured assessment; IOPI where available.	Baseline; end of treatment; follow-up.	Functional change does not by itself prove OSA resolution.
Patient-centered outcomes	OSA-18 or comparable quality-of-life measure; snoring; daytime symptoms; tolerability.	Baseline; end of treatment; follow-up.	Symptom improvement should be interpreted with sleep and functional measures.
Treatment process and safety	Prescribed dose; completed sessions; home-practice logs; dropout; adverse events; concurrent therapies.	Throughout treatment and at each assessment point.	Adherence is both exposure information and a potential source of selection bias.
Phenotype descriptors	Age; body mass status; nasal airway status; residual anatomical obstruction; syndrome status; previous and concurrent treatment.	Before inclusion and whenever treatment changes.	Descriptors are required for transferability and subgroup interpretation.

The outcome set separates objective sleep-breathing outcomes from functional and process outcomes.

3.5. Clinical-research positioning

The fifth result is a practical research pathway (Figure 2). It translates the evidence map into a sequence that remains consistent with pediatric sleep and otolaryngology guidance. The pathway begins with persistent symptoms or high-risk status after AT, moves through objective confirmation, separates anatomical from functional contributors, and uses OMT only as an adjunct when a functional target and adequate capacity for participation are present.

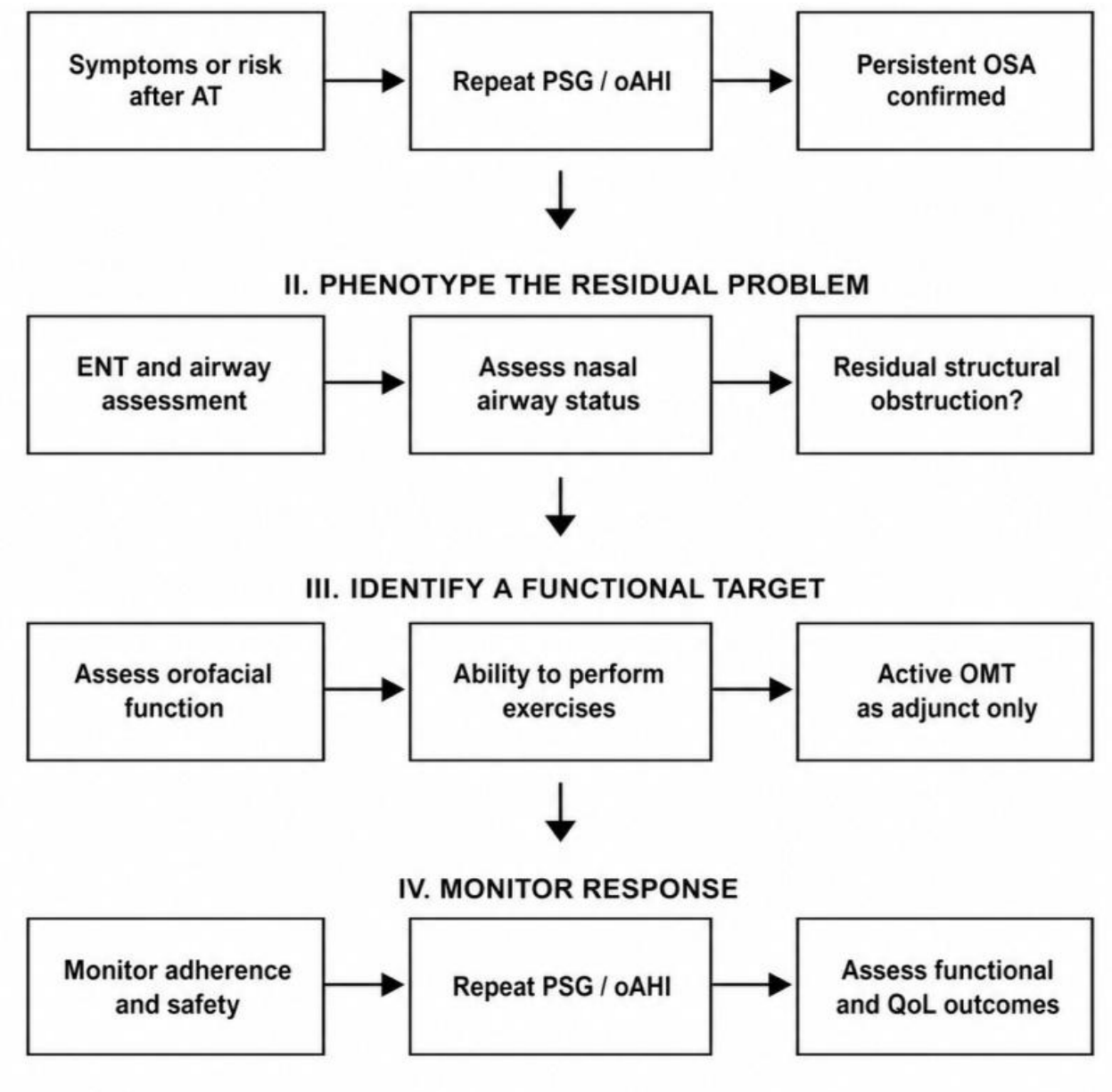

Figure 2. Proposed assessment and monitoring sequence for research on OMT.

The sequence is not a clinical guideline. It indicates where objective diagnosis, anatomical assessment, functional assessment, and adherence monitoring should occur.

The pathway also clarifies the role of emerging research. A published protocol now applies OMT to pediatric OSA populations with and without Down syndrome and uses a broader set of sleep, functional, and quality-of-life outcomes [26]. A registry record comparing two oral myofunctional reeducation methods in children with persistent sleep apnea similarly shows that the field is moving toward protocolized comparative work [27]. These documents are important for research planning, but they do not constitute efficacy evidence until results are reported and peer reviewed.

A research design consistent with this pathway would avoid enrolling children solely because they mouth-breathe after surgery. It would first confirm persistent OSA, then document whether the dominant residual

problem is anatomical, inflammatory, dentofacial, neuromuscular, behavioral, or mixed. Only after this sequence would the child enter an active OMT arm. The control condition would need to distinguish usual postoperative care, sham or attention control, and other active treatments. Follow-up should extend beyond the immediate end of training because the question is not only whether a child can perform exercises, but whether a learned functional pattern persists.

4. Discussion

The evidence map changes the form of the question. The available literature cannot answer whether OMT treats pediatric OSA as a single disease entity. It can answer a narrower and more clinically defensible question: whether active exercises have a plausible and partially supported role in a subset of children whose residual postoperative disease includes a modifiable functional component. This shift matters because the target of OMT is not adenotonsillar tissue, craniofacial anatomy, or obesity. Its direct target is motor behavior and the resting or functional configuration of the orofacial complex.

This interpretation reconciles rather than simply rejects the optimistic and cautious lines of evidence. Early reviews were correct to recognize a signal of improvement and a biologically plausible mechanism [19,20]. The Cochrane synthesis was also correct to treat pediatric AHI evidence after AT as low certainty because small samples, short follow-up, and inconsistent controls do not justify a firm therapeutic claim [21]. Recent reviews add breadth but do not fully solve the problem because adult and pediatric samples, active and passive interventions, and postoperative and non-postoperative contexts remain difficult to separate [22-25].

The proposed taxonomy has a direct methodological consequence. Pooling active therapist-guided OMT with passive appliances or orthodontic interventions may increase the number of included studies, but it weakens interpretability. In a child with persistent OSA, the hypothesized mechanism of an exercise program is learning, strength, coordination, and habitual posture. The hypothesized mechanism of an appliance or orthodontic expansion is different. A future trial should either isolate these exposures or explicitly test a multi-component package without attributing the entire effect to OMT.

This approach also clarifies why OMT is absent from major recommendations as a stand-alone therapy for persistent OSA. The absence does not prove inefficacy; it indicates that existing evidence has not yet reached the certainty, standardization, and directness usually required for guideline endorsement. A therapy can be plausible, safe in many contexts, and worthy of research while still being premature as a universal recommendation. This distinction is particularly relevant in pediatrics, where undertreatment of residual OSA and overtreatment with poorly specified behavioral programs are both avoidable risks.

Adherence is another reason why the field requires careful design. Completion of a daily exercise program by a child and family may indicate exposure to an adequate therapeutic dose. It may also identify families with higher health literacy, better access to care, fewer comorbid barriers, or stronger motivation. If adherence is used to define the treatment group after the fact, apparent benefit can reflect both a dose-response relationship and selection bias. Future trials should prospectively specify adherence thresholds, record prescribed and completed dose, and preserve intention-to-treat interpretation where possible.

For otolaryngology, the practical implication is not to replace standard postoperative care with exercise therapy. Rather, persistent symptoms after AT should trigger objective reassessment and anatomical evaluation. Orofacial assessment may then identify a rehabilitation target that is relevant but secondary to the diagnosis of residual disease. This framing protects children from two opposite errors: overlooking a residual anatomical obstruction because mouth breathing is treated as a habit, and dismissing functional dysfunction because surgery has already addressed the most obvious tissue obstruction.

For families, the distinction should be communicated in concrete language. Exercises may be discussed

as a possible way to address a functional pattern, not as proof that the airway problem has been solved. Improvement in snoring or lip closure should prompt reassessment of outcomes rather than termination of follow-up. This is not a conservative rhetorical stance; it follows from the mismatch between the target of OMT and the diagnostic construct of OSA. The safety of a low-risk adjunct does not remove the need to verify whether the sleep disorder remains active.

The minimum outcome set is meant to support this more disciplined research pathway. Objective sleep measures remain necessary because the disorder under consideration is sleep-related airway obstruction. Functional measures are equally necessary because they represent the target most directly affected by OMT. Patient-centered outcomes are needed because improvements in snoring, sleep quality, and daytime function may matter even when oAHI change is modest. Process outcomes are needed because a behavioral intervention cannot be interpreted without knowing whether the child actually performed it.

The framework also has a publication-level implication. Future authors should report negative or neutral findings with the same detail as positive functional change. A small study in which children complete exercises but show no meaningful AHI change can still be informative if it documents phenotype, dose, adherence, and concurrent care. Without such reporting, the literature will continue to overrepresent attractive mechanisms and underrepresent the boundary conditions under which rehabilitation does not alter sleep-breathing physiology.

The limitations of this review follow from both the corpus and the method. The primary pediatric interventional studies are few, small, and heterogeneous. One recent study could be used only within the bounds of its open abstract [18]. No new meta-analysis, individual patient-data analysis, formal risk-of-bias scoring, or publication-bias assessment was performed. Several systematic reviews re-use overlapping primary studies, so their conclusions are not independent pieces of evidence. In addition, syndromic and nonsyndromic children should not be merged without explicit stratification, because neuromuscular, craniofacial, and adherence conditions can differ substantially.

A further limitation is geographical and disciplinary transferability. Although the source corpus was searched internationally, most clinical and research infrastructure reflected settings with access to PSG, pediatric sleep specialists, orthodontic assessment, or speech-language professionals trained in orofacial function. In health systems where PSG access is limited, the temptation to use symptoms or mouth breathing as substitutes for objective diagnosis is greater. The framework proposed here remains applicable as a research logic, but implementation will depend on local diagnostic capacity and multidisciplinary referral pathways.

These limitations do not negate the functional hypothesis. They set the level of confidence. Current evidence supports investigation of OMT as an adjunct for a defined phenotype and supports standardization of outcome reporting. It does not support a universal recommendation, a pooled numerical effect estimate for children after AT, or the claim that OMT prevents recurrence of OSA.

5. Conclusions

Persistent pediatric OSA after adenotonsillectomy requires a move from binary surgical outcome assessment to structured phenotyping of residual disease. The mapped literature suggests that active OMT has a biologically plausible and partially supported functional target, especially when mouth breathing, altered lip posture, or tongue dysfunction persists after airway evaluation. The strength of evidence, however, differs by outcome domain. Short-term functional and symptom changes are more consistently reported than durable polysomnographic resolution.

The main scientific result of this review is a bounded analytical framework. OMT should be studied as a potential adjunct for children with objectively confirmed residual OSA, assessed nasal airway patency, documented orofacial dysfunction, absence of dominant untreated structural obstruction, and measurable

capacity to adhere to a program. The proposed intervention taxonomy, structural-functional model, assessment sequence, and minimum outcome set can guide more comparable studies. Until multicenter randomized data with longer follow-up are available, OMT should not be presented as a substitute for repeat polysomnography, treatment of residual anatomical obstruction, positive airway pressure, orthodontic or pharmacological care, or other standard therapies.

Declarations

Author contributions. Karan Olga Anatolyevna: Conceptualization; Methodology; Investigation; Formal Analysis; Data Curation; Visualization; Writing - Original Draft; Writing - Review & Editing; Project Administration. The author has read and approved the final version of the manuscript.

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Ethics approval. Not applicable. This review did not involve author-generated data from human participants, animals, or clinical interventions.

Conflicts of interest. The author declares no conflicts of interest.

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