

Nasal Mucociliary Dysfunction in Children with Adenoid Hypertrophy Mechanisms and Postoperative Recovery

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Abstract: *Adenoid hypertrophy is associated with impaired nasal mucociliary transport, but the mechanisms and meaning of postoperative improvement remain incompletely resolved. This critical narrative review examines pediatric studies of transport time, ciliary activity, adenoidal morphology and bacterial biofilms. The findings suggest contributions from nasopharyngeal obstruction, epithelial dysfunction and the local microbial environment; their interactions remain incompletely established. Several studies report faster transport after adenoidectomy, and one prospective investigation found improved nasal ciliary beat frequency at six months. These functional findings do not demonstrate epithelial regeneration, which requires structural assessment. Adenoid size does not consistently correlate with mucociliary impairment, and biofilm findings differ between clinical phenotypes. Early shortening of transport time may reflect changes in drainage or secretions, but the mechanism cannot be inferred from timing alone. Existing studies are limited by heterogeneous populations, combined operations, variable follow-up and incomplete assessment of reproducibility. An interpretive framework distinguishes transport improvement, cellular recovery, symptom benefit and prognostic usefulness. Nasal transport testing can contribute to research on adenoid disease, but current evidence does not establish it as an independent indication for surgery or a validated predictor of postoperative benefit.*

Keywords: adenoid hypertrophy; adenoidectomy; mucociliary clearance; ciliary function; postoperative recovery

1. Introduction

Adenoid disease is usually assessed through symptoms, nasopharyngeal examination and associated ear or sleep problems. The S2k guideline describes pathological adenoid hyperplasia in relation to mechanical obstruction and chronic inflammation, rather than size alone [1]. Mucociliary transport adds a functional dimension to this assessment. A child can have an anatomically narrow nasopharynx, altered epithelial activity and retained secretions at the same time, but the relative contribution of each process is difficult to establish.

Studies spanning several decades associate adenoid hypertrophy with slower nasal transport, altered adenoidal surface morphology or reduced nasal ciliary beat frequency [2–4]. These observations support a biological relationship. They leave important questions unanswered: whether obstruction directly causes transport impairment, whether inflammation independently affects the nasal epithelium, and whether a shorter postoperative transit time represents sustained recovery.

A previous review by Dinev and Atanasov examined the historical development of clearance assessment and the role of visible markers [5]. The present review addresses a different question: how should mucociliary dysfunction and postoperative improvement be interpreted in children with adenoid disease? Its purpose is to separate evidence for mechanisms from evidence for treatment-associated change, and to identify the measurements needed to connect

functional findings with clinical benefit. No unpublished patient results are reported.

2. Review Approach

This is a critical narrative review. Targeted web-assisted searches were completed on 24 October 2026 using combinations of adenoid hypertrophy, adenoidectomy, nasal mucociliary clearance, mucociliary transport, ciliary beat frequency and adenoid biofilm. The principal bibliographic source was PubMed, accessed through web-indexed records. PubMed Central, original journal publisher websites and university repositories provided additional accessible material. Individual study titles and author names were also searched to verify bibliographic details and clarify study design. These targeted searches were not a systematic search of the complete PubMed database.

Priority was given to pediatric primary studies directly examining adenoid disease, nasal transport or ciliary activity. Studies of adenoidal biofilms and experimental airway physiology informed the appraisal of possible mechanisms. The final set of 22 cited sources comprises 18 primary empirical studies, one methodological paper, two clinical guidelines and one previous narrative review. These counts describe the included references, not the number of records screened; a formal screening count was not recorded. Evidence from airway models and adults is identified as indirect when applied to pediatric adenoid disease. Full articles were examined where accessible, while some reports were assessed from indexed abstracts or repository summaries. Detailed risk-of-bias assessment was

therefore not possible for every report. No duplicate screening, formal evidence grading or meta-analysis was undertaken, and the included studies do not constitute an exhaustive evidence set.

Physiological basis of transport dysfunction

Mucus movement depends on coordinated ciliary activity and a suitable physical environment. In cultured airway systems, Matsui and colleagues demonstrated coupling between mucus movement and periciliary liquid transport [6]. Button and colleagues described a periciliary brush that separates the mucus layer from the epithelial surface [7]. Their work helps explain how mucus concentration can influence transport. These experiments support considering secretion properties alongside ciliary activity. They do not establish that the same molecular changes occur in children with adenoid hypertrophy.

A transport-time test records marker passage through the sampled pathway. Ciliary beat frequency measures one aspect of ciliary activity under specified sampling and laboratory conditions. Morphological examination describes tissue structure. These measurements address different domains, so improvement in one cannot establish improvement in the others. Transport can become faster without a demonstrated change in epithelial architecture.

Nasopharyngeal obstruction may alter secretion drainage. Mucus is propelled by ciliary action, however, and should not be conceptualized as simply carried by inspiratory airflow. After surgery, nasal resistance may decrease while transport becomes faster. Observing both changes does not establish that improved airflow caused the transport improvement. Establishing that pathway requires a study designed to assess mediation.

Adenoid size and epithelial changes

Maurizi and colleagues examined nasal function and adenoidal surface morphology in 86 children aged 4–10 years [2]. Their findings connected hypertrophic adenoids with impaired clearance and surface changes, including loss of ciliated epithelium in markedly hypertrophic tissue. This provides direct evidence that adenoid disease can involve more than a restricted airway lumen. However, the examined adenoidal surface is not equivalent to the inferior turbinate or the entire nasal transport pathway.

More recent findings caution against assuming a simple relationship between size and functional severity. Berkiten and colleagues compared 31 children with adenoid hypertrophy with 30 controls aged 5–12 years [3]. Clearance time was longer in the affected group, but hypertrophy size did not correlate with transport time, and routine nasal cytology did not differ between groups. Similarly, Marusiakova and colleagues found no correlation between adenoid size and nasal ciliary beat frequency [4]. These findings do not make size clinically irrelevant; they indicate that size alone cannot fully characterize mucociliary dysfunction.

Ohashi and colleagues studied ciliary activity in adenoidal epithelium from patients with recurrent otitis media with effusion and described recovery of reduced activity under

culture conditions [8]. This supports the possibility of a reversible functional component. Culture findings cannot establish the time course of nasal recovery after adenoidectomy, and they should not be translated into evidence that every child has reversible epithelial injury.

Microbial reservoirs and biofilms

Adenoidal microbial communities provide a second plausible connection between adenoid disease and impaired clearance. Coticchia and colleagues compared adenoidal surfaces from children undergoing surgery for chronic rhinosinusitis or obstructive sleep apnea [9]. Biofilm coverage was markedly greater in the rhinosinusitis group. The comparison suggests that clinical phenotype influences the microbial environment. It does not support treating every enlarged adenoid as having the same biofilm burden.

Winther and colleagues demonstrated bacteria and biofilm within mucus overlying excised adenoids and emphasized the importance of surface secretions and crypts [10]. Their study also acknowledged uncertainty about whether biofilms cause hypertrophy. Nistico and colleagues used molecular identification and microscopy to demonstrate polymicrobial biofilms in adenoids from children with chronic otitis media or obstructive sleep apnea [11]. Together, these studies support the reservoir concept, while differences in sampling and microscopy complicate direct comparisons.

A reasonable mechanistic hypothesis is that retained secretions, microbial persistence and local epithelial dysfunction can reinforce one another. The direction and strength of these relationships remain uncertain in individual children. Biofilm microscopy is not a measurement of nasal transport, and a reduction in transport time after surgery does not prove biofilm eradication. Direct testing of this hypothesis would require microbial or tissue measurements linked to repeated functional assessments in the same participants.

Evidence for postoperative change

The principal clinical studies are summarized in Table 1. Maurizi and colleagues evaluated 34 children before adenoidectomy and at six months, reporting faster mucociliary transport together with lower nasal resistance [12]. Hellin Meseguer and colleagues investigated 30 children aged 3–13 years using charcoal and reported improvement at two weeks [13]. The latter study is relevant to early postoperative observation, but its accessible English abstract provides limited procedural detail. Neither a detailed modern test protocol nor numerical effect estimates should be reconstructed from that abstract.

Ranga and colleagues compared 50 children with adenotonsillar hypertrophy with 50 healthy children and repeated testing one month after adenotonsillectomy [14]. Mean transport time decreased from 16.97 to 8.70 minutes; the control mean was 8.55 minutes. The combined operation limits attribution to adenoid removal alone. Arnaoutakis and Collins also reported postoperative improvements in transport and symptoms [15]. Although 35 children entered their investigation, only 10 completed the paired transport assessment. This distinction between recruitment and

analyzable paired measurements is essential when weighing the result.

Yazici and colleagues evaluated 64 children with adenoid hypertrophy, with or without otitis media with effusion, before surgery and at one month [16]. Both groups improved, but postoperative transport remained slower in children with effusion. This finding supports stratification by middle-ear phenotype and cautions against assuming a uniform recovery pattern. In a separate prospective study, Marusiakova and colleagues evaluated nasal ciliary activity in 64 affected children and 43 controls [4]. At six months, postoperative beat frequency was higher than before surgery and was not statistically different from the control value. This is evidence of recovery in a measured cellular function at that visit, rather than direct evidence of epithelial regeneration or a complete recovery trajectory.

Parker and colleagues examined a separate clinical question [17]. In children with established otitis media with effusion, preoperative saccharin transport measurements did not predict otoscopic clearance at one year. Their repeatability investigation also identified substantial variation. These findings concern prognostic performance and should be distinguished from studies assessing postoperative change in transport itself.

Interpreting early and later recovery

The literature contains observations at two weeks, one month and six months, but these are generally different cohorts assessed with different methods [4,12–16]. They cannot be joined into a single empirical recovery curve. The available evidence supports studying recovery over time; it does not establish a universal sequence or the point at which recovery is complete.

For interpretation, an early postoperative interval may be considered a period in which altered drainage, secretion burden and local healing could influence transport. Later measurements can assess whether improvement persists and whether it coincides with changes in ciliary activity or symptoms. This temporal framework is a hypothesis for study design. Direct evidence is needed before assigning a particular biological mechanism to a particular postoperative day.

Interpretation should therefore specify the measured domain and the relevant comparator. Normalization of transport requires an age- and protocol-compatible reference distribution. Cellular recovery requires a cellular measurement, while recovery of epithelial architecture requires structural evidence. Table 2 summarizes these inferential boundaries.

A single postoperative measurement also cannot show whether a change is transient, sustained or influenced by an intercurrent respiratory illness. Repeated assessment is particularly useful when the scientific question concerns durability. A pragmatic research schedule could include baseline, an early visit at approximately 2–4 weeks and a later visit at approximately 3–6 months. These intervals are proposed for comparative research and are not a clinical follow-up standard derived from validated outcome data.

Reference values and measurement uncertainty

Pediatric reference findings vary considerably between studies. Corbo and colleagues assessed 295 schoolchildren aged 11–14 years and found a six-minute coefficient of repeatability in a subgroup of 50 [18]. Yadav and colleagues reported a mean saccharin clearance time of 5.7 minutes in 100 healthy children aged 4–15 years in a tropical setting [19]. Differences in populations and protocols make it inappropriate to treat either study as a universal reference for every pediatric transport test. The age range also matters: findings in adolescents cannot automatically define normal values for preschool children.

Visible markers can reduce dependence on the child's report of taste, but they introduce their own endpoint and observation procedures. Austero and Gelera compared saccharin and charcoal testing in 50 adults aged 19–77 years [20]. Similar group means provide useful comparative information, but do not validate pediatric thresholds. Their assignment of saccharin to one side and charcoal to the other also makes it difficult to separate a marker effect from a side effect.

Evidence of similar means is insufficient to establish that two methods agree closely enough for use in the same child. Bland and Altman described why correlation and mean comparison alone are inadequate for evaluating clinical measurement agreement [21]. For studies of postoperative recovery, repeatability and observer agreement should be established under the actual pediatric protocol. Video recording can permit review of a visible endpoint, but the existence of a recording does not establish reliability until independent readings are compared.

Investigators should preserve the temporal resolution of the test in their reporting. If the endpoint is checked intermittently, the arrival occurs within an interval between observations; the recorded inspection time is not an exact continuously observed transit time. Confidence intervals and measurement uncertainty should accompany estimates of change. Statistical significance alone cannot determine whether an improvement exceeds measurement variation or matters to the child.

Clinical implications and persistent dysfunction

Current evidence supports mucociliary testing as a complementary research outcome in adenoid disease. It does not establish a transport threshold that independently identifies children who should undergo surgery. Surgical decisions should remain based on the clinical syndrome, examination and established indications [1]. A study showing average postoperative improvement cannot establish that operating on an otherwise asymptomatic child with a prolonged transport time will provide benefit.

Persistent symptoms or delayed transport after surgery should prompt reassessment of the clinical phenotype and test conditions. Intercurrent infection, concomitant rhinitis, residual obstruction and the reliability of the measurement may be relevant considerations. A prolonged nasal transport result by itself is nonspecific and cannot diagnose a primary disorder of ciliary function.

When the history suggests primary ciliary dyskinesia, the 2025 joint European Respiratory Society and American Thoracic Society guideline recommends an integrated specialist approach using appropriate combinations of diagnostic investigations [22]. Simple transport testing cannot replace that evaluation, and an apparently improved postoperative result should not override a strongly suggestive clinical history. This distinction protects against attributing all persistent airway disease to adenoid anatomy.

Priorities for future studies

Study populations should be defined according to the primary research question. Isolated adenoidectomy should be distinguished from adenotonsillectomy and additional procedures. Middle-ear effusion and chronic rhinosinusitis should be characterized prospectively because these phenotypes may affect dysfunction and recovery. Investigators should report exclusion criteria, reasons for exclusion and the number of children unable to complete testing. This makes the clinical applicability of a tightly selected cohort easier to assess.

Measurement reliability requires specific assessment. Repeated baseline tests can quantify spontaneous variability before surgery, and independent endpoint readings can assess observer agreement. Investigators should report paired change with its confidence interval. Age, respiratory illness, relevant treatment and smoke exposure warrant prespecified consideration. Comparator groups or repeated preoperative observations can help distinguish surgical change from maturation, repeated-test familiarity and temporal fluctuation.

Clinical outcomes should be assessed alongside physiological measurements. Studies combining transport tests with ciliary analysis and patient-reported outcomes could examine whether changes are concordant within the same children. Discordance may identify distinct biological processes or measurement limitations. Tissue or biofilm analysis can add mechanistic information when clinically obtained material is available; additional invasive sampling requires separate justification. The minimum important change in transport time should ultimately be anchored to outcomes meaningful to children and families.

3. Limitations of this Review

The narrative selection of evidence is vulnerable to retrieval and interpretation bias. Access to several older reports was

limited to abstracts or repository summaries, and detailed methods could not always be checked. No pooled effect estimate or formal certainty rating is presented. Biofilm studies, epithelial experiments and clinical transport studies address related but different questions. Their synthesis here provides a framework for interpretation and hypothesis generation, rather than proof of a unified causal mechanism.

4. Conclusion

Children with adenoid hypertrophy can have impaired nasal transport and altered ciliary activity, and several studies report postoperative improvement. The findings are compatible with anatomical, epithelial and microbial contributions, but do not establish the relative importance of each mechanism in an individual child. Functional improvement should be distinguished from structural recovery and from the ability to predict surgical benefit. Standardized longitudinal studies linking physiological measurements with patient-relevant outcomes are needed before transport testing can guide surgical selection or define complete postoperative recovery.

Declarations

Ethics approval and consent: This review reports no newly collected participant data and includes no identifiable patient material.

Data availability: No new dataset was generated. The sources discussed are identified in the reference list.

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Table 1: Selected pediatric studies relevant to dysfunction and recovery

Study	Population and assessment	Observation	Interpretive limit
Maurizi 1984 [2]	86 children aged 4–10 years; transport and adenoidal morphology	Functional impairment accompanied surface abnormalities	Adenoidal morphology does not represent the whole nasal epithelium
Berkiten 2023 [3]	31 affected children and 30 controls; ages 5–12	Longer transport time; no size correlation or group cytology difference	Case–control association does not establish cause
Marusiakova 2020 [4]	64 affected children and 43 controls; nasal ciliary analysis at 6 months	Postoperative beat frequency approached control values	Cellular function measured at one later visit
Maurizi 1986 [12]	34 children; transport and rhinomanometry at 6 months	Faster clearance and lower nasal resistance	Parallel changes do not establish mediation
Hellín Meseguer 1994 [13]	30 children aged 3–13; charcoal testing at 2 weeks	Postoperative improvement reported	Limited procedural detail in accessible abstract
Ranga 2000 [14]	50 affected and 50 healthy children; saccharin at 1 month	Mean 16.97 to 8.70 min; control 8.55 min	Adenotonsillectomy prevents attribution to adenoidectomy alone

Arnaoutakis 2011 [15]	35 recruited; 18 paired surveys; 10 paired transport tests	Symptoms and transport improved	Small paired subset and mixed procedures
Yazici 2014 [16]	64 children; with or without effusion; assessment at 1 month	Both groups improved; longer postoperative transit with effusion	Phenotype and co-interventions affect interpretation
Parker 1992 [17]	44 children with effusion recruited; 40 transport-time analyses	Baseline transport did not predict effusion clearance at 1 year	Prognostic outcome differs from postoperative transport change

This is a selected evidence map, not an exhaustive systematic review. Study populations, procedures, methods and follow-up differ; the findings should not be pooled without full-text methodological assessment.

Table 2: Framework for interpreting postoperative findings

Observed finding	Supported interpretation	Additional evidence needed
Shorter postoperative transit time	Improvement in the specified transport endpoint	Repeatability, temporal resolution and repeated follow-up
Value within a reference range	Consistency with the selected reference population	Age and protocol compatibility; individual uncertainty
Higher ciliary beat frequency	Change in sampled ciliary activity under test conditions	Beat pattern, sample handling and links to in vivo transport
Less obstruction after surgery	Anatomical or airflow improvement	Direct evidence connecting airflow change to transport
Better symptoms or quality of life	Patient-relevant clinical benefit	Magnitude, durability and relationship to functional outcomes
Biofilm in removed adenoid tissue	Microbial aggregates were present in the sampled material	Same-patient longitudinal evidence for mechanism or eradication
Baseline test associated with outcome	Potential prognostic information	Prespecified prediction model and independent validation

This framework is an interpretive proposal derived from the reviewed evidence. It is not a validated clinical decision rule or consensus guideline.

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