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ORIGINAL RESEARCH

Impact of Upper Airway Comorbidities and Tonsil/Adenoid Synergistic Effects on Pediatric OSA Severity

Yiwei Feng[1,*], Weisong Cai[1,*], Qiang Xie[1,2,*], Xiaoping Ming[1], Xiuping Yang[1,2], Xiong Chen[1,2]

[1]Department of Otorhinolaryngology, Head and Neck Surgery, Zhongnan Hospital of Wuhan University, Wuhan, People's Republic of China; [2]Sleep Medicine Center, Zhongnan Hospital of Wuhan University, Wuhan, People's Republic of China

*These authors contributed equally to this work

Correspondence: Xiong Chen, Zhongnan Hospital of Wuhan University, No. 169, Donghu Road, Wuhan, 430071, People's Republic of China, Email zn_chenxiong@whu.edu.cn

Purpose: To investigate the factors influencing the severity of obstructive sleep apnea (OSA) in children and to elucidate the synergistic effects of upper airway comorbidities (chronic rhinosinusitis, nasal septum deviation and malocclusion) with tonsillar and adenoid hypertrophy on ventilation efficiency.

Patients and Methods: A total of 404 pediatric patients with OSA aged 6–10 years diagnosed between January 2022 and January 2025 were included in this retrospective cohort study and underwent logistic regression analysis to identify the risk factors for pediatric OSA. Three-dimensional upper airway models under various comorbidity states were constructed via cone–beam computed tomography (CBCT), and postoperative airway resistance changes were calculated via computational fluid dynamics (CFD) simulation and simulated surgery.

Results: Grade 3 tonsillar or adenoid hypertrophy, the presence of comorbidities, and overweight/obesity were identified as independent risk factors for increased OSA severity. A dose–response relationship was observed between the number of comorbidities and OSA severity, with the highest odds ratio (OR=9.392, 95% CI=2.459–35.875) for triple-positive comorbidities. CFD simulations demonstrated that tonsillectomy and adenoidectomy (T&A) significantly improved ventilation function across different OSA severities. Comorbidities influence airway resistance, with postoperative resistance in children with triple-positive comorbidities approaching preoperative levels in those without comorbidities.

Conclusion: T&As hold therapeutic value for pediatric OSA patients, but comorbidities significantly mitigate surgical efficacy through synergistic effects.

Keywords: obstructive sleep apnea, children, tonsillectomy and adenoidectomy, computational fluid dynamics, synergistic effects

Introduction

Obstructive sleep apnea (OSA) is a common sleep-disordered breathing condition, and its pathogenesis is closely associated with structural abnormalities and functional dysregulation of the upper airway (UA).[1] The tonsils and adenoids, as critical anatomical structures in the pediatric UA, are recognized as primary etiological factors for OSA due to their hypertrophy.[2] Current pediatric OSA diagnostic and treatment guidelines advocate tonsillectomy and adenoidectomy (T&A) as a first-line therapeutic approach for children with confirmed tonsillar and adenoid hypertrophy.[3–5] However, for patients who do not meet the criteria for hypertrophy, the guidelines recommend comprehensive evaluation of the oral, nasal, and laryngeal airways but do not address the synergistic effects among these anatomical regions. Emerging evidence suggests that even in the absence of significant hypertrophy, the anatomical positioning and dynamic interactions of the tonsils and adenoids with surrounding tissues can alter aerodynamic properties and affect ventilation efficiency.[6–8]

In addition to tonsillar and adenoid factors, the role of UA comorbidities in the development and progression of OSA is increasingly recognized. Chronic rhinosinusitis (CRS)-induced mucosal edema,[9] nasal septum deviation (NSD)-

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mediated asymmetric nasal cavity narrowing,[10] and malocclusion-related maxillofacial skeletal abnormalities can exacerbate airway obstruction.[11] Notably, these comorbidities may synergize with tonsillar and adenoid abnormalities through multiple mechanisms to influence airflow distribution and pressure gradients; however, quantitative assessments of such multifactorial interactions remain scarce. Clinical observations indicate a significant positive correlation between the number of comorbidities and OSA severity, underscoring the necessity for integrated treatment strategies. However, the specific pathways and effects of these comorbidities on T&A outcomes remain to be systematically explored.

This study leverages a large-scale clinical cohort and computational fluid dynamics (CFD) simulation techniques to elucidate the synergistic impact of tonsillar and adenoid hypertrophy with UA comorbidities on pediatric OSA. By analysing polysomnography (PSG) data and aerodynamic parameters before and after T&A across different OSA severity levels and comorbidity states, we aimed to validate the universal value of T&A from both a clinical efficacy and biomechanical perspective. The research also seeks to establish an innovative methodological framework for the quantitative assessment of multifactorial interactions in UA.

Materials and Methods

Case Selection

A retrospective analysis was conducted on pediatric patients diagnosed with OSA at Wuhan University Zhongnan Hospital between January 2022 and January 2025. The inclusion criteria were as follows: (1) age between 6 and 10 years; (2) no prior history of T&A; (3) preoperative PSG-confirmed OSA; (4) exclusion of patients with other acute/chronic organic or nonorganic diseases; (5) receipt of T&A as core treatment during hospitalization, without receiving correction for NSD or malocclusion; and (6) postoperative follow-up including PSG at 6–8 months. A total of 404 children were included. The study complies with the Declaration of Helsinki, and the research protocol was approved by the Zhongnan Hospital of Wuhan University (No. 2025048K), and all participants and their parents or legal guardians provided written informed consent.

Endoscopy and PSG

The nasal cavity and nasopharynx were examined via STORZ electronic nasendoscopy. Tonsillar size was classified into three grades on the basis of the palatoglossal arch and posterior pharyngeal midline boundaries. Adenoid size was assessed via a four-grade classification system.[12] PSG monitoring was conducted via the Philips Alice 6 system, with all patients undergoing ≥7 hours of nocturnal sleep respiratory monitoring. The reports were verified by two physicians.

CBCT Image Acquisition and Measurement

CBCT images were acquired via a NewTom VG (Quantitative Radiology, Verona, Italy). Patients sat upright with the Frankfurt horizontal plane parallel to the ground and facial midline aligned with the machine's long axis, holding breath at end-exhalation. The exposure parameters were 80 kV, 2 mA, and 17s (axial thickness of 1 mm). Mimics 21.0 (Materialise, Belgium) was used for image measurement and UA reconstruction (from the nasal vestibule to the glottis), excluding sinuses, owing to its minimal impact on nasal airflow.[13,14]

3D Reconstruction and Mesh Generation

The UA models were surface optimized in Geometric Warp 2015 (Geomagic, USA). The polyhedral meshes were generated in Ansys Fluent Mesh 2022 R1 (ANSYS, USA). Five-layer prism grids were applied at the boundaries, with mesh independence testing confirming negligible velocity differences when the mesh count exceeded 2.6 million; all the models retained mesh counts above this threshold.

CFD Simulation

In this study, the incompressible Navier–Stokes equations were employed as the governing equations for simulating steady-state inspiratory airflow in the nasal airway. The second-order upwind algorithm was used to calculate airflow characteristics, with an inhalation flow rate of 12.5 L/min representing resting respiratory conditions in children.[15]

A velocity inlet boundary condition was applied at the nasal cavity entrance, where the no-slip velocity condition and rigid wall assumption were implemented. A pressure outlet boundary condition was established below the glottis. Simulations were performed via the k-ω SST turbulence model in Ansys Fluent 2022 R1 (ANSYS, Inc., Canonsburg, Pennsylvania) coupled with the SIMPLE algorithm for pressure–velocity interactions. The residual values were set to 1e-5 to ensure numerical convergence, which was achieved in all the models. Finally, lateral view analyses were used to quantify airflow streamlines and wall pressure distributions, with pressure differences measured across the inlet and outlet boundaries.

Statistical Analysis

The data were analysed via SPSS 25.0. Paired t tests (two-tailed) were used to compare normally distributed variables, whereas Mann–Whitney *U*-tests and Kruskal–Wallis *H*-tests were used to compare nonnormally distributed data. Pearson linear regression was used to assess correlations. Logistic regression included univariate analysis ($p<0.2$ covariates selected for multivariable modelling) and stepwise variable addition ($p<0.05$). GraphPad Prism 7.0 was used to generate the statistical plots.

Results

Patient Information

A total of 404 pediatric patients (231 males and 174 females) were enrolled in this study (Table 1). Patients were categorized into normal weight (261 cases) and overweight/obese (143 cases) groups on the basis of the International Obesity Task Force (IOTF)[13] and the Working Group on Obesity in China (WGOC)[14] criteria. According to the obstructive apnea-hypopnea index (OAHI), 296 patients had mild OSA (OAHI 1--5), 81 had moderate OSA (OAHI 5--10), and 27 had severe OSA (OAHI >10).[15] Hypoxemia (lowest SpO_2 <92%) was diagnosed in 256 patients.[15] The most frequent chief complaints were open-mouth breathing (78.22% reported ≥3 nights/week) and snoring (49.26% reported ≥3 nights/week). Endoscopic and CBCT evaluations revealed that 51.49% of the patients had CRS, 37.13% had NSD, and 19.8% had malocclusion (Figure 1A). The distributions of tonsils and adenoids across different OSA severity levels were generally consistent, except for a lower proportion of Grade 3 tonsils observed in patients with mild OSA (Figure 1B). This analysis further revealed a strong correlation between comorbidity count and OSA severity (Figure 1C). Specifically, 99 patients had double-positive comorbidities (20.61%, 34.57%, and 37% across the mild, moderate, and severe OSA groups, respectively), whereas 18 patients had triple-positive comorbidities (2.36%, 8.64%,

Table 1 Basic Information of the Included Patients

	Category	Number & Mean ± SD
Gender	Male	231
	Female	173
Age (y)	6–7	214
	8–9	95
	10–12	95
BMI (kg/m2)	Total	16.79 ± 3.35
	Male	17.10 ± 3.62
	Female	16.37 ± 2.88
Weight Status	Normal-weight	261
	Overweight & Obese	143
OAHI	1–5	296
	5–10	81
	>10	27
$LSaO_2$ < 92%	Yes	256
	No	148

Abbreviations: OAHI, obstructive apnea-hypopnea index; $LSaO_2$, Lowest Saturation Oxygen.

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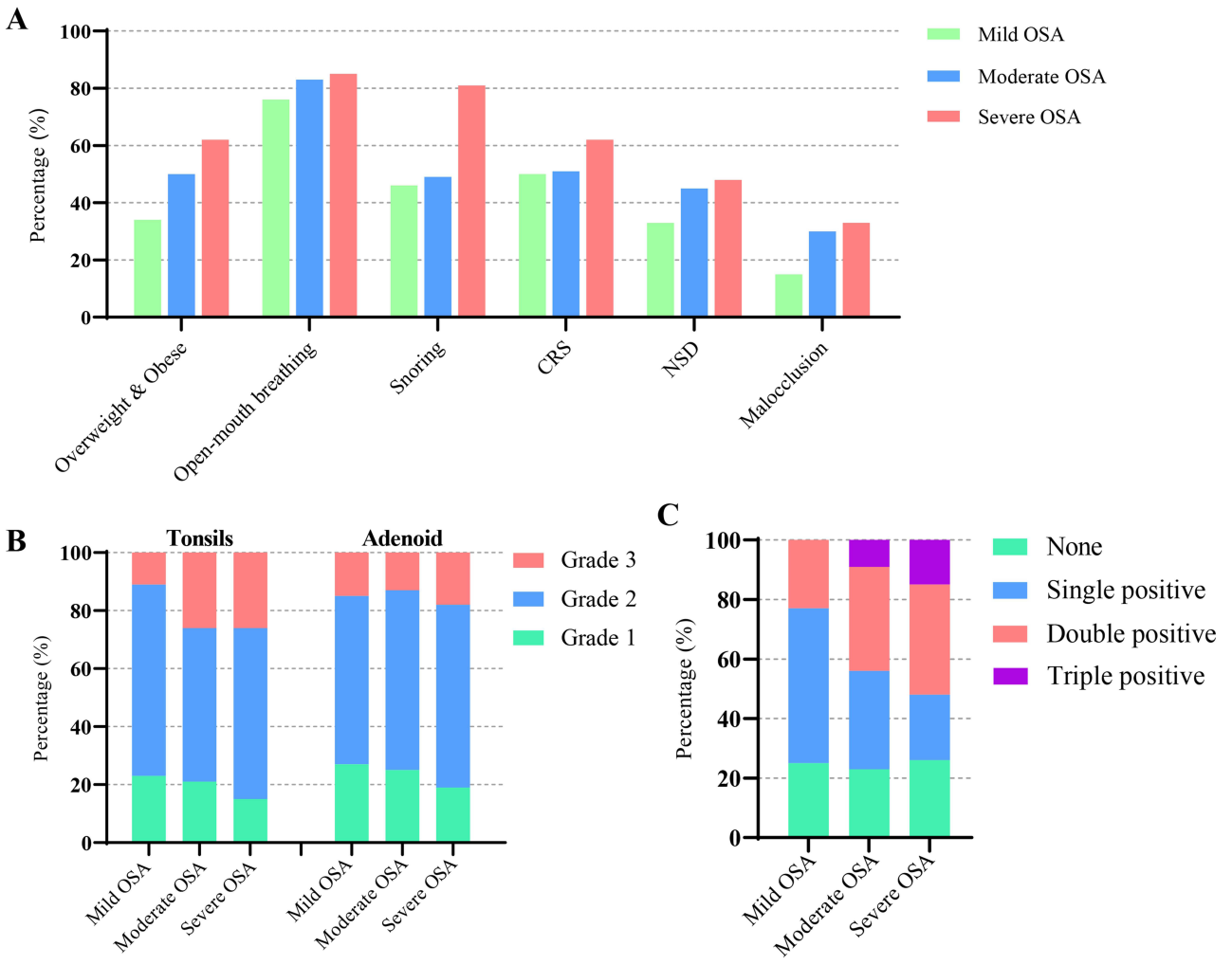

Figure 1 Statistical profile of pediatric OSA patients. (**A**) Symptom and comorbidity incidence rates across OSA severity groups. (**B**) Tonsil/adenoid size distribution in pediatric OSA patients stratified by severity. (**C**) Comorbidity count distribution patterns in pediatric OSA patients.
Abbreviations: CRS, chronic rhinosinusitis; NDS, nasal septum deviation.

and 14.8%, respectively). Conversely, single-positive comorbidities were most prevalent in patients with mild OSA. Figure 2A–D illustrates a child with three comorbidities (severe maxillary retrusion, CRS, and NDS), while anterior mandibular protrusion was also a common malocclusion type (Figure 2D–F). These findings suggest that tonsils/adenoids restrict ventilation efficiency in pediatric OSA patients, that comorbidities amplify this effect, and that the number of comorbidities strongly correlates with OSA severity.

Logistic Regression and Synergistic Analysis

To test our hypothesis, univariate logistic regression excluded low-correlation variables (gender, $p>0.2$) before adjusting for the remaining covariates in multivariate analysis. Weight status, tonsil size, adenoid size, CRS, NDS, and malocclusion emerged as independent risk factors for OSA severity (Table 2). Notably, only grade 3 tonsils/adenoids served as independent risk factors. The interactions between tonsils and adenoids were categorized into three levels: Level 1 (one of the tonsils/adenoids was grade 0 or 1, and neither exceeded Grade 2), Level 2 (both tonsils/adenoids were Grade 2), and Level 3 (at least one of the tonsils/adenoids was Grade 3). The comorbidity count was classified into four categories (none, single, double, triple positive), which were included in the multivariate logistic regression analysis alongside weight status (Table 3). Multivariate analysis confirmed weight (OR=2.349, CI=1.336–4.131), double comorbidity (OR=5.072, CI=1.985–12.956), and triple positive comorbidity (OR=9.392, CI=2.459–35.875) as independent risk

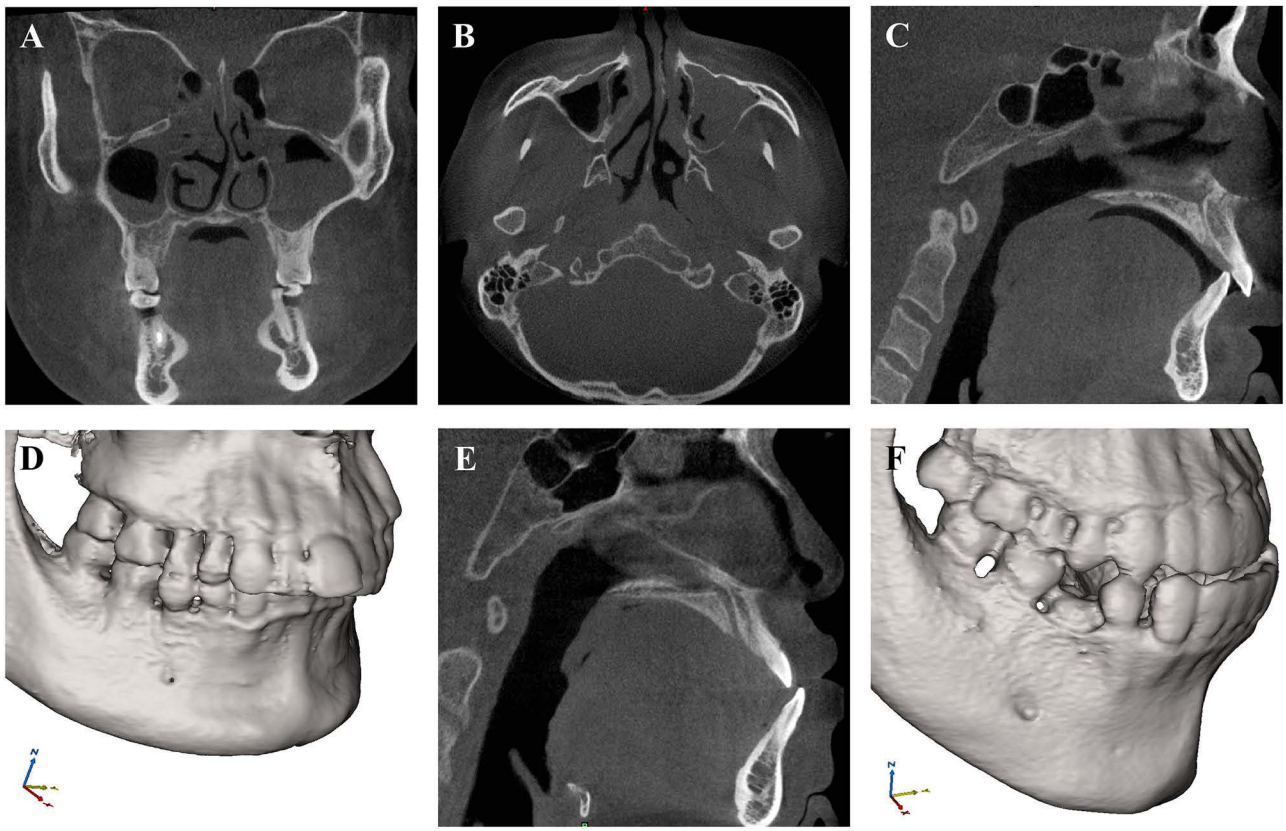

Figure 2 Upper airway comorbidities via CBCT and 3D reconstruction. (**A**–**C**) A patient with concurrent CRS, NDS, and malocclusion. (**D**) 3D reconstruction of the same patient's malocclusion, showing severe mandibular retrusion. (**E** and **F**) Another patient with malocclusion characterized by mandibular protrusion.

factors, whereas the level 3 tonsil/adenoid interaction (OR=3.704, CI=1.745–7.861) was also significant. This analysis confirmed our hypothesis that the comorbidity count is strongly correlated with OSA severity.

To further investigate the impact of tonsils/adenoids on OSA severity and the correlation between comorbidity count and OSA severity, we stratified patients by OSA severity and comorbidity count to compare pre/postoperative symptom

Table 2 Univariate and Multivariate Logistic Regression Analyses of Pediatric OSA Severity

Variable	Category	Univariate Model		Multivariate Model	
		OR & 95% *CI*	*p*	OR & 95% *CI*	*p*
Gender	Male	Reference		–	
	Female	1.373 (0.801–2.352)	0.249	–	–
Weight Status	Normal-weight	Reference		Reference	
	Overweight & Obese	2.174 (1.266–3.735)	0.005*	2.361 (1.332–4.186)	0.003†
Age (y)	6–7	Reference		Reference	
	8–9	0.731 (0.393–1.358)	0.322	0.752 (0.388–1.456)	0.397
	10–12	0.504 (0.229–1.129)	0.196*	0.6 (0.254–1.421)	0.246
Tonsillar Hypertrophy	Grade 1	Reference		Reference	
	Grade 2	0.96 (0.412–1.957)	0.912	1.742 (0.775–3.913)	0.179
	Grade 3	2.914 (1.291–6.58)	0.01*	11.237 (3.629–34.792)	<0.001†
Adenoid Hypertrophy	Grade 0 and 1	Reference		Reference	
	Grade 2	0.817 (0.334–1.998)	0.657	1.75 (0.825–3.713)	0.145
	Grade 3	0.929 (0.501–1.723)	0.815	3.993 (1.269–12.562)	0.018†

(*Continued*)

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Table 2 (Continued).

Variable	Category	Univariate Model		Multivariate Model	
		OR & 95% *CI*	***p***	**OR & 95% *CI***	***p***
CRS	Yes	Reference		Reference	
	No	1.316 (0.867–2.262)	0.189*	2.41 (1.271–4.57)	0.007†
NDS	Yes	Reference		Reference	
	No	1.45 (0.844–2.493)	0.178*	2.302 (1.211–4.375)	0.011†
Malocclusion	Yes	Reference		Reference	
	No	1.997 (1.097–3.645)	0.024*	2.75 (1.374–5.506)	0.004†

Notes: In univariate logistic regression, the p value threshold for variable selection was 0.2(*), whereas the threshold for significance testing was 0.05(†). "Reference" means using this group as the benchmark for comparison.
Abbreviations: CRS, chronic rhinosinusitis; NDS, nasal septum deviation.

Table 3 Multivariate Logistic Regression Analysis Incorporating the Synergistic Effects of Tonsils and Adenoids on OSA Severity

Variable	Category	OR	95% *CI*	*p*
Weight Status	Normal-weight	Reference		
	Overweight & Obese	2.349	1.336–4.131	0.003*
Tonsillar-adenoidal Interplay	Level 1	Reference		
	Level 2	0.805	0.376–1.721	0.576
	Level 3	3.704	1.745–7.861	0.001*
Comorbidity Count	None	Reference		
	Single	1.483	0.676–3.252	0.325
	Double	5.072	1.985–12.956	0.001*
	Triple	9.392	2.459–35.875	0.001*

Notes: *$p < 0.05$. "Reference" means using this group as the benchmark for comparison.

prevalence and sleep study data (Tables 4 and 5). After T&A, significant improvements in symptom positivity and sleep study parameters were observed across all OSA severity groups, confirming the efficacy of the procedure in enhancing ventilation efficiency in pediatric OSA patients. Notably, while T&A conferred substantial benefits across all comorbidity categories, triple-positive comorbidity patients presented relatively worse postoperative outcomes than did those with no or single-positive comorbidities.

Table 4 Preoperative and Postoperative Symptom Outcomes and Sleep Study Data Comparison Stratified by OSA Severity

Category	Mild OSA		Moderate OSA		Severe OSA	
	Preoperation	**Postoperation**	**Preoperation**	**Postoperation**	**Preoperation**	**Postoperation**
Symptom						
Open-mouth Breathing ≥ 3 night/week, No. (%)	225 (76.01%)	31 (10.47%)*	68 (83.95%)	11 (13.58%)*	23 (85.19%)	4 (14.81%)*
Snoring ≥ 3 night/week, No. (%)	137 (46.28%)	14 (4.73%)*	40 (49.38%)	6 (7.4%)*	22 (81.48%)	3 (11.1%)*
Sleep study data						
OAHI [event/hour], (SD)	2.14 (1.05)	0.37 (0.29)*	6.85 (1.29)	0.57 (0.89)*	23.21 (15.68)	4.08 (6.76)*
Sleep efficiency [%], (SD)	86.44 (9.13)	89.97 (9.18)*	84.35 (10.25)	88.28 (9.7)*	84.68 (11.19)	88.78 (8.59)*
Microarousal frequency, (SD)	71.62 (27.03)	64.36 (25.17)*	91.93 (38.89)	76.94 (30.81)*	169.26 (73.12)	82.37 (35.8)*
Total Microarousal Index, (SD)	8.44 (2.92)	7.88 (3)*	11.14 (4.09)	9.46 (3.33)*	19.79 (8.4)	10.25 (4.09)*
$LSaO_2$ [%], (SD)	90.66 (2.69)	93.83 (1.37)*	87.96 (4.45)	92.86 (1.97)*	82.37 (8.72)	91.04 (3.68)*
Average Heart Rate [per minute], (SD)	74.32 (8.8)	74.54 (8.47)	75.33 (11.83)	74.69 (10.03)	80.46 (11.75)	75 (8.57)*

Notes:*$p < 0.05$.
Abbreviations: OAHI, obstructive apnea-hypopnea index; OSA, obstructive sleep apnea; SD, standard deviation; $LSaO_2$, Lowest Saturation Oxygen.

Table 5 Preoperative and Postoperative Symptom Outcomes and Sleep Study Data Comparison Stratified by Comorbidity Count

Category	None & Single Positive		Double Positive		Triple Positive	
	Preoperation	Postoperation	Preoperation	Postoperation	Preoperation	Postoperation
Symptom						
Open-mouth Breathing ≥ 3 night/week, No. (%)	223 (77.7%)	29 (10.1%)*	79 (79.8%)	13 (13.13%)*	15 (83.33%)	4 (22.22%)*
Snoring ≥ 3 night/week, No. (%)	138 (48.08%)	14 (4.88%)*	51 (51.52%)	6 (6.06%)*	11 (61.11%)	3 (16.67%)*
Sleep study data						
OAHI [event/hour], (SD)	3.94 (5.57)	0.62 (1.17)*	5.4 (8.89)	1.18 (3.71)*	8.4 (8.91)	0.86 (0.74)*
Sleep efficiency [%], (SD)	86.11 (9.33)	90.42 (9.22)*	85.55 (10.09)	90.88 (10.27)*	84.46 (9.81)	89.85 (6.15)*
Microarousal frequency, (SD)	80.79 (41.34)	69.05 (27.21)*	82.91 (39.91)	63.95 (29.18)*	101.83 (63.54)	75.39 (29.24)*
Total Microarousal Index, (SD)	9.53 (4.47)	8.43 (3.24)*	9.92 (4.69)	8.01 (3.29)	12.1 (7.99)	9.07 (3.06)*
$LSaO_2$ [%], (SD)	89.6 (4.64)	93.59 (1.75)*	89.68 (3.45)	93.09 (2.18)*	88.44 (4.68)	93.11 (2.18)*
Average Heart Rate [per minute], (SD)	75.44 (10.25)	74.88 (9.06)	73.61 (8.45)	73.12 (8.17)	73.02 (9.09)	72.89 (7.82)

Notes:*$p < 0.05$.
Abbreviations: OAHI, obstructive apnea-hypopnea index; SD, standard deviation; $LSaO_2$, Lowest Saturation Oxygen.

CFD Simulation and Airway Resistance

Five normal-weight pediatric OSA patients with Grade 2 tonsils/adenoids were selected to simulate pre/postoperative airflow under different comorbidity scenarios (no comorbidities, only CRS, only NDS, only malocclusion, and triple-positive comorbidities). They were selected due to the excellent symmetry of UA and the high similarity in the dimensions and structures of their UA, which effectively minimized interference with airflow. Figure 3 visualizes airflow

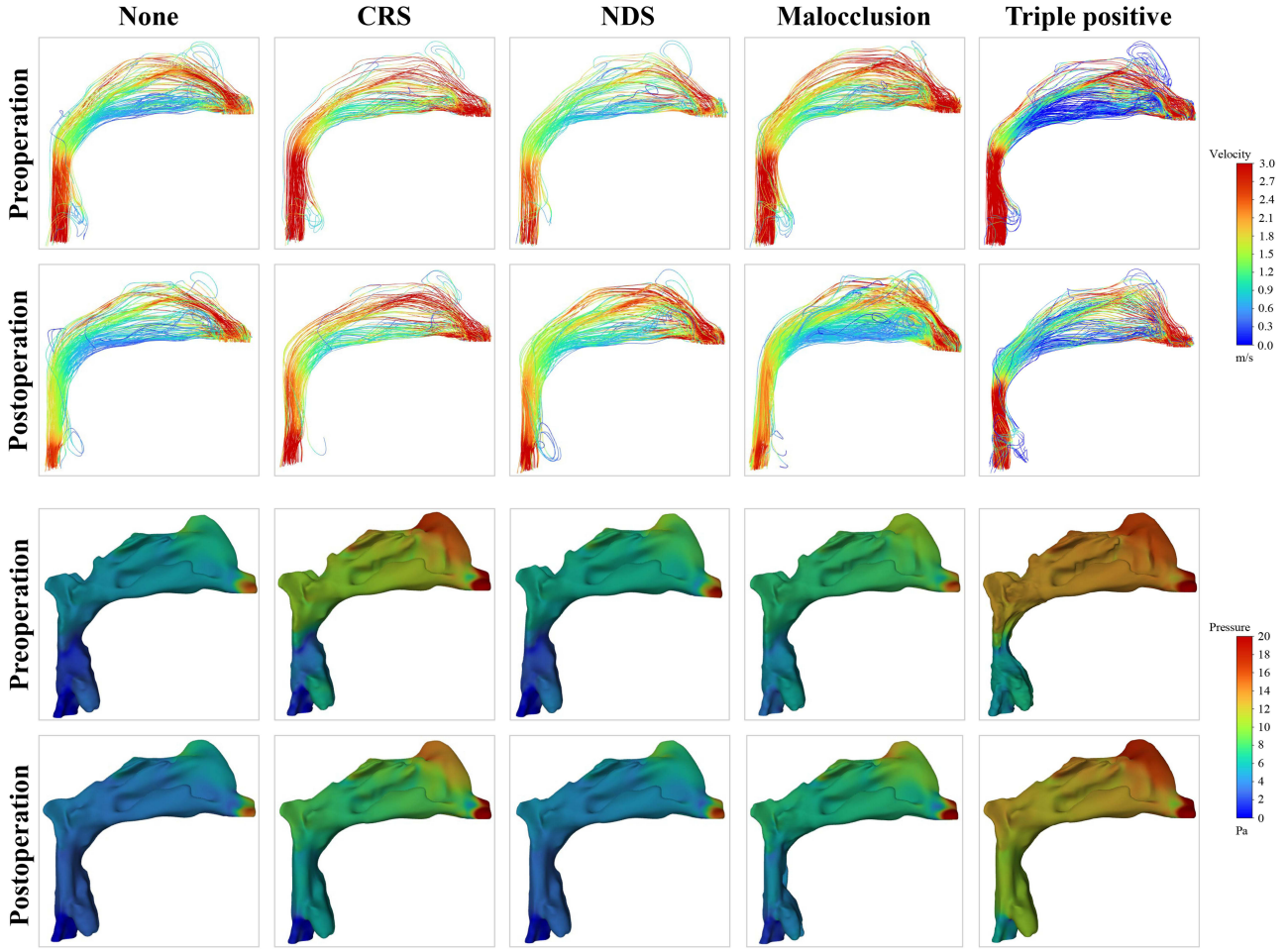

Figure 3 Simulated surgical interventions: airflow streamlines and airway wall pressures in pediatric OSA patients with different comorbidity profiles.
Abbreviations: CRS, chronic rhinosinusitis; NDS, nasal septum deviation.

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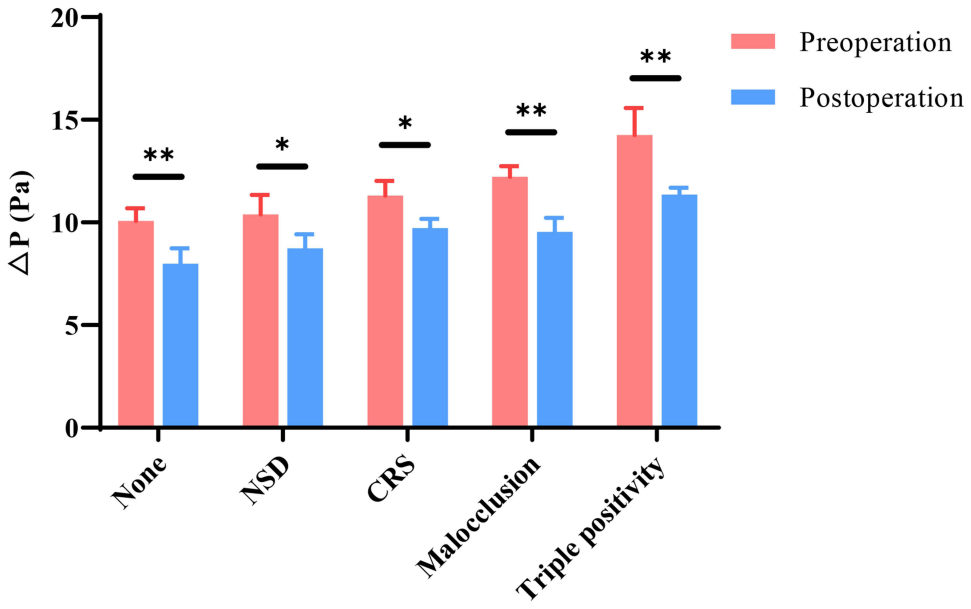

Figure 4 Simulated preoperative and postoperative airway pressure drop changes. **p<0.01, *p<0.05.
Abbreviations: ΔP, pressure drop; CRS, chronic rhinosinusitis; NDS, nasal septum deviation.

velocity and wall pressure distributions across multiple models. Following T&A, regions of high-speed airflow within the nasal cavity and nasopharynx exhibited a transition from turbulent to laminar flow behavior. This transformation substantially reduced peak airflow velocities and wall shear stresses, thereby enhancing overall ventilation efficiency in the upper airway. The pressure drop (ΔP) is commonly used to measure the UA resistance. As shown in Figure 4, we calculated ΔP across different models from the nasal vestibule to the glottis. Surgical simulation significantly reduced ΔP in all the models, confirming that T&A effectively alleviated airway resistance. Notably, in triple-positive comorbidity cases, postoperative pressure drops closely resembled those of preoperative models without comorbidities, highlighting the necessity of addressing comorbidities to achieve maximal therapeutic benefits.

Discussion

This retrospective study provides evidence, to a certain extent, of the therapeutic value of T&A in pediatric OSA, and the modulatory role of comorbidity synergy in treatment outcomes. This study integrated CFD analysis with clinical cohort data to reveal the universal therapeutic value of T&A in pediatric OSA and the modulatory role of comorbidity synergy in treatment outcomes. While conventional wisdom posits that T&A is primarily effective for children with significantly enlarged tonsils and adenoids,[5] our CFD simulations demonstrated that even moderate hypertrophy (grade 2) of these structures can induce pharyngeal turbulence through dynamic interactions with surrounding soft tissues, thereby increasing airway resistance. The presence of comorbidities may amplify such interactions. This turbulence activates the Venturi effect, leading to palatal collapse and secondary obstruction.[16,17] Marcus et al[18] investigated the impact of T&A on the growth and development of OSA pediatric and found that T&A can alleviate clinical symptoms while improve secondary outcomes of behavior, quality of life, and polysomnographic findings, thus providing evidence of beneficial effects of early adenotonsillectomy. Chervin et al[19] also confirmed that baseline predictors of OSA resolution included lower AHI, better oxygen saturation, smaller waist circumference or percentile, higher-positioned soft palate, smaller neck circumference, and non-black race (each $p < 0.05$). Therefore, T&A is of positive significance in reducing nocturnal hypoxemic events and improving cognitive function in OSA pediatric.

Some clinical physicians might express concerns about long-term complications associated with early tonsillectomy and adenoidectomy. The advent of cryoablation technology has expanded the indications for T&A by minimizing tissue damage while preserving partial immune function, particularly in young patients.[20] Research by Blackshaw et al on partial tonsillectomy indicates that retaining 30–50% of tonsillar tissue maintains local immune activity, accelerates

functional recovery, and effectively resolves airway obstruction.[21] A prospective longitudinal study involving 1,257 children demonstrated that only 2.1% of participants required reoperation for recurrent tonsillar hypertrophy-induced airway obstruction within 1–4 years after the initial T&A,[22] suggesting that for immunocompromised or recurrently infected pediatric OSA patients, partial resection may serve as an effective option for balancing airway patency and local immune function preservation.

Another key finding of this study is that comorbidities may attenuate the anatomical benefits of T&A, with the comorbidity count demonstrating a dose–response relationship with OSA severity (Table 3). This conclusion aligns closely with the multiplanar obstruction model,[23] suggesting that CRS, NDS, and malocclusion interact through pathophysiological mechanisms to amplify airway resistance in pediatric OSA patients. CRS-induced mucosal edema reduces the nasal cross-sectional area, whereas the release of IL-6 and TNF-α via trigeminal-vagal reflexes increases pharyngeal muscle tone and decreases compliance, accelerating airflow velocity.[9,24] NSD-induced asymmetric narrowing of the nasal cavity exacerbates airway resistance abnormalities caused by high-speed airflow, forming a "double-peaked obstruction" in the nasopharynx and thereby intensifying pediatric respiratory dysfunction.[16] Malocclusion compresses the effective oropharyngeal airspace, creating "clamping narrowing" between the tongue base and adenoids. This mechanically elevates peak airflow velocities in the oropharynx, inducing a transition from laminar to turbulent flow in both nasopharyngeal and oropharyngeal cavities.[25] The interaction of these three factors results in a "nasal acceleration–nasopharyngeal negative pressure–oropharyngeal turbulence" cascade of blockages. Therefore, multidimensional approaches addressing both anatomical and pathological mechanisms are essential to further reduce airway resistance and optimize ventilation efficiency.

This study has some main limitations. This study has several main limitations. Firstly, CFD simulations are based on steady-state inspiratory conditions and the rigid wall assumption in an awake, sitting posture. However, during sleep, factors such as muscle relaxation, posterior displacement of the tongue base, and periodic movement of the uvula can worsen the ventilation conditions in the pharyngeal area compared to the awake state. As a result, the airway resistance obtained through simulation is lower than in real conditions and cannot fully represent the dynamic characteristics of airway collapse during sleep. Secondly, due to variations in age, gender, clinical presentation, and UA anatomical morphology among pediatric patients, it is challenging for clinicians to accurately assess the outcomes of comprehensive interventions such as T&A and treatment of comorbidities (eg, CRS medication). Future research could establish personalized surgical prognosis models through dynamic airway impedance monitoring combined with real-time pressure-flow analysis. Additionally, some stratified analyses have small sample sizes, which may affect the reliability of our statistical results. Lastly, as a retrospective study with a small sample size, there may be selection bias that limits causal inference regarding comorbidity interactions. Larger-scale prospective studies are needed in the future to further validate our findings. In conclusion, our investigation consolidates the universal efficacy of T&A in pediatric OSA management. These findings reveal that comorbidities act as critical modulators of postoperative outcomes, necessitating integrated multidimensional interventions that address both structural and pathophysiological dimensions. Furthermore, our work establishes the feasibility of digital healthcare modalities in pediatric upper airway disorders, equipping clinicians with precision-driven tools to tailor evidence-based therapies for individual OSA patients.

Conclusion

T&A demonstrates consistent efficacy in optimizing airway resistance and enhancing ventilation function across all severity levels of pediatric OSA. Comorbidities, including CRS, NDS, and malocclusion, interact synergistically with tonsils/adenoids to exacerbate airway obstruction.

Data Sharing Statement

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

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Author Contributions

Yiwei Feng: Conceptualization, Formal Analysis, Writing - Original Draft, Methodology.
Weisong Cai: Conception, Data Curation, Investigation, Writing - Original Draft.
Qiang Xie: Data Curation, Investigation, Writing – Original Draft.
Xiuping Yang: Resources, Software, Validation, Writing – Original Draft.
Xiaoping Ming: Supervision, Validation, Project Administration, Writing – Review & Editing.
Xiong Chen (corresponding author): Methodology, Supervision, Validation, Funding Acquisition, Writing – Review & Editing.

All authors have drafted or substantially revised the manuscript, reviewed and agreed on all versions of the article before submission, during revision, and in the final stage before publication, and approved the journal to which the article was submitted. Additionally, all authors agree to be accountable for the content of the manuscript and any significant changes introduced at the proofing stage.

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Disclosure

The author(s) report no conflicts of interest in this work.

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