



Research article

Effects of soft palate and tongue collapse on upper airway flow field under progressive muscle tone loss: a fluid-structure interaction study

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Abstract: *Background:* Obstructive sleep apnea syndrome (OSAS) is characterized by recurrent upper airway collapse during sleep, yet the relative contributions of soft palate versus tongue collapse remain poorly quantified. *Methods:* Three-dimensional numerical models of the upper airway, soft palate, and tongue were reconstructed from computed tomography (CT) images. A transient bidirectional fluid-structure interaction (FSI) method was employed to determine the ventilation pressure required to maintain a prescribed inspiratory flow across progressively reduced artificial support loads applied to the soft palate and tongue (0–100% of the calibrated reference loads). *Results:* Isolated soft palate collapse exerted minimal effect on the required ventilation pressure. Obstruction severity was predominantly governed by tongue behavior: Below approximately 93% reduction of the reference tongue supported load, and airflow dynamics remained largely unchanged (maximum velocity ≤ 15.6 m/s, ventilation pressure ≤ 94 Pa). Beyond this level, appreciable tongue–soft palate compression was established and a marked nonlinear transition occurred: The ventilation pressure required to maintain the prescribed flow increased by approximately 806% (to 852 Pa), and the maximum velocity reached 46.3 m/s. Joint collapse analysis confirmed that tongue behavior, rather than soft palate status, is the primary determinant of flow obstruction in this model. *Conclusions:* In this single-subject model and under the selected artificial support loads, appreciable tongue–soft palate compression produces a marked nonlinear increase in the pressure required to maintain a prescribed flow, occurring at

approximately 93% reduction of the reference tongue support load. This contact transition is model-specific and should not be interpreted as a universal neuromuscular threshold. The findings provide proof-of-concept evidence for a biomechanical mechanism of tongue-driven obstruction that warrants validation in multi-subject, clinically characterized cohorts.

Keywords: obstructive sleep apnea; upper airway; tongue collapse; fluid-structure interaction; ventilation pressure; tongue–soft palate contact transition

Abbreviations: 3D: three-dimensional; AHI: apnea-hypopnea index; CPAP: continuous positive airway pressure; CT: computed tomography; FSI: fluid-structure interaction; MRI: magnetic resonance imaging; OSAS: obstructive sleep apnea syndrome

1. Introduction

Obstructive sleep apnea syndrome (OSAS) is among the most prevalent sleep-related breathing disorders, affecting an estimated 936 million adults aged 30–69 years worldwide, with moderate-to-severe disease present in approximately 425 million individuals [1]. The disorder is characterized by recurrent episodes of partial (hypopnea) or complete (apnea) upper airway obstruction during sleep, resulting in intermittent hypoxia, sleep fragmentation, and excessive daytime sleepiness. Untreated OSAS confers independent and graded risks for incident hypertension, atrial fibrillation, heart failure, type 2 diabetes mellitus, and neurocognitive impairment [2]. Despite continuous positive airway pressure (CPAP) representing the established first-line therapy, long-term adherence remains limited, with reported nightly non-adherence in 46–83% of patients [3].

Computational biomechanical modeling has emerged as an indispensable tool for investigating upper airway flow dynamics beyond the capabilities of clinical imaging alone. Early CFD studies employed rigid-wall models to analyze airflow patterns and pressure distributions [4–6], while fluid-structure interaction (FSI) approaches have since enabled coupled simulation of airflow and soft tissue deformation, capturing the bidirectional interactions between intraluminal pressure, tissue displacement, and resultant flow field alterations [7–9]. These models have provided clinically relevant insights into surgical outcome prediction [10], oral appliance treatment planning [11], and patient-specific nasal airflow assessment [12,13]. Compared with rigid-wall approaches, FSI is uniquely suited for simulating progressive muscle tone deterioration, as it dynamically couples ventilation pressure with tissue compliance, enabling systematic exploration of neuromuscular thresholds that govern airway patency.

The tensor veli palatini and genioglossus are the principal muscles regulating soft palate and tongue position, respectively, and their neuromuscular integrity is essential for maintaining airway patency during sleep [14,15]. Clinical studies using drug-induced sleep endoscopy (DISE) have confirmed that the soft palate (velopharynx) and tongue base represent the most common sites of pharyngeal collapse [16], with synchronized electromyography demonstrating progressive reduction in genioglossus activity across sleep stage transitions [17]. Although upper airway biomechanical models have been established [4,7], three critical limitations persist, as comprehensively reviewed by Faizal et al. [8]: (1) Neuromuscular thresholds remain unquantified: The progressive loss of muscle tone in the tensor veli palatini and genioglossus across the full 0–100% range has not been

parametrically modeled; (2) epithelial injury criteria are absent: Safety limits for airflow-induced wall shear stress and velocity thresholds associated with pharyngeal epithelial desiccation, inflammation, and chronic injury [18] lack systematic experimental validation in the context of progressive collapse; and (3) mechanistic ambiguity persists: The relative contributions of palatal versus lingual collapse to obstruction severity have not been disentangled through controlled parametric analysis, limiting the evidence base for site-specific surgical and non-surgical interventions.

Collapse typically initiates at the soft palate and tongue base [16], with dynamic magnetic resonance imaging (MRI) confirming significant tissue displacement during apneic events [19]. The critical closing pressure (P_{crit}), which varies with sleep stage [20], directly quantifies this neuromuscular impairment.

Rationale of this study: Upper airway models examine fixed anatomical states or single collapse configurations, and therefore cannot resolve how progressive loss of dilator muscle support translates into airflow obstruction, nor can they disentangle the relative contributions of palatal and lingual collapse. To address these gaps, we develop a biomechanical model that: (i) Simulates progressive reduction of artificial support loads applied near the tensor veli palatini and genioglossus insertions (0–100% of calibrated reference loads); (ii) identifies the contact-transition level at which the ventilation pressure required to maintain a prescribed flow increases markedly; and (iii) reports the associated airflow velocity changes during progressive collapse, without asserting a validated epithelial injury criterion. The rationale for this parametric design is that controlled, single-variable exploration of support-load reduction in a fixed anatomy isolates the biomechanical consequences of tongue–palate interaction from confounding anatomical variation; a necessary first step before patient-specific or population-level modeling is undertaken.

2. Materials and methods

2.1. 3D models

The CT data were obtained from a 25-year-old healthy Asian male to establish a baseline biomechanical framework (Figure 1). While this single-subject approach enables precise isolation of neuromuscular effects without confounding anatomical variations, it inherently limits direct population-level generalizability. All findings are restricted to this particular anatomy and modeling configuration. CT acquisition details (scanner model, kVp/mAs, voxel dimensions, slice thickness, reconstruction kernel, participant posture, and respiratory instruction), participant characteristics (BMI, neck circumference, craniofacial features, and body position), segmentation protocol (software, thresholds, manual corrections, smoothing, surface repair, and sinus simplification criteria), and baseline cross-sectional areas/hydraulic diameters at retropalatal, retroglossal, and hypopharyngeal levels are available from the corresponding author upon reasonable request. The geometry was obtained from the Medimodel database; participant selection criteria and redistribution permissions are described in the Ethics section.

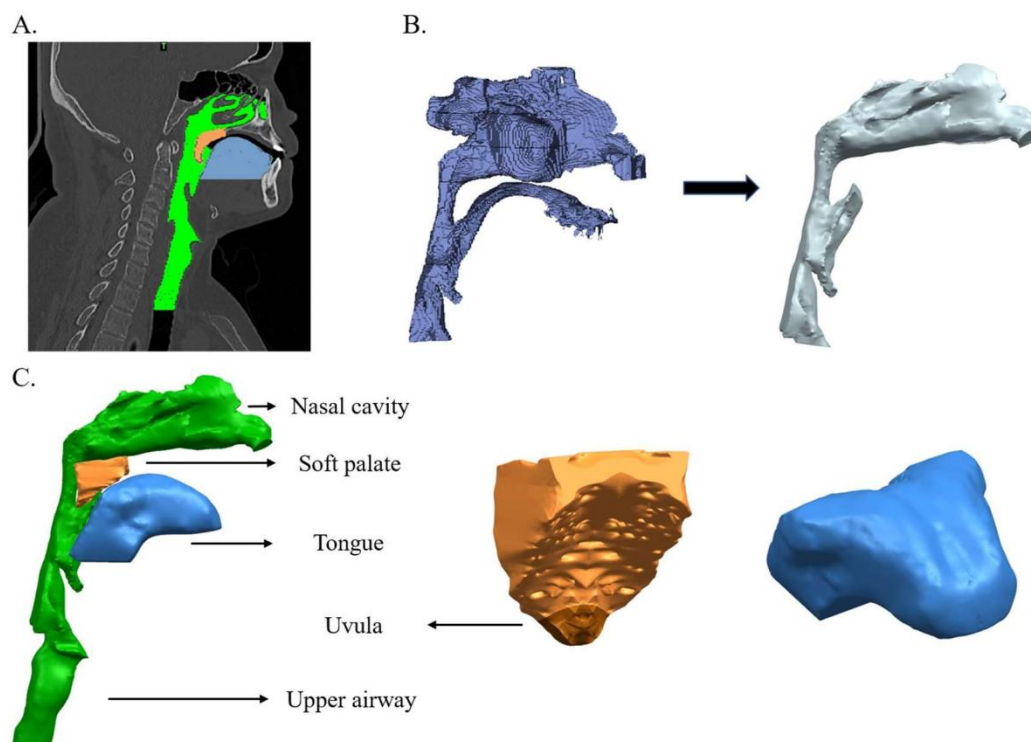


Figure 1. 3D model building (A. CT image after region division; B. Point-cloud model of the upper airway and solid model after simplification of the paranasal sinus; and C. Simplified model of the upper airway and soft tissue).

2.2. Materials

2.2.1. Material properties of upper airway tissues

While researchers have typically modeled upper airway soft tissues as linear elastic materials with defined Young's modulus and Poisson's ratio [21–23], emerging evidence classifies these biological tissues as incompressible nonlinear hyperelastic materials [24]. To accurately capture this behavior, we implemented experimentally derived hyperelastic parameters from Haddad et al. [25], with tissue-specific responses characterized as follows: The uvula exhibits a nonlinear stress–strain relationship (Figure 2A) quantified through indentation testing of fresh specimens from 12 OSAS patients [25]; the soft palate demonstrates anisotropic hyperelastic properties (Figure 2B) that are validated via tensile testing [25,26]; while the tongue displays transversely isotropic behavior (Figure 2C) obtained from direct mechanical testing of lingual specimens [25,27]. These parameters originate from oropharyngeal tissue specimens collected during standard OSAS surgeries, where indentation experiments confirmed the mechanical properties summarized in Table 1 [25,26]. The exact hyperelastic formulation (Mooney–Rivlin, Ogden, or Yeoh) used for each tissue, all fitted coefficients with units, the assignment of anisotropy and fiber directions, Poisson behavior (incompressibility constraint), damping, pre-stress, initial configuration, tissue thickness, and attachment conditions are available from the corresponding author upon reasonable request so that all parameters can be entered directly into a solver. The use of material measurements from OSAS surgical

specimens in a healthy-subject model is acknowledged as a limitation and was examined in $\pm 20\%$ sensitivity analyses.

Table 1. Material properties.

Soft tissue	Density (kg/m ³)	Elastic modulus
Uvula	920	Figure 2A
Soft palate	920	Figure 2B
Tongue	1060	Figure 2C

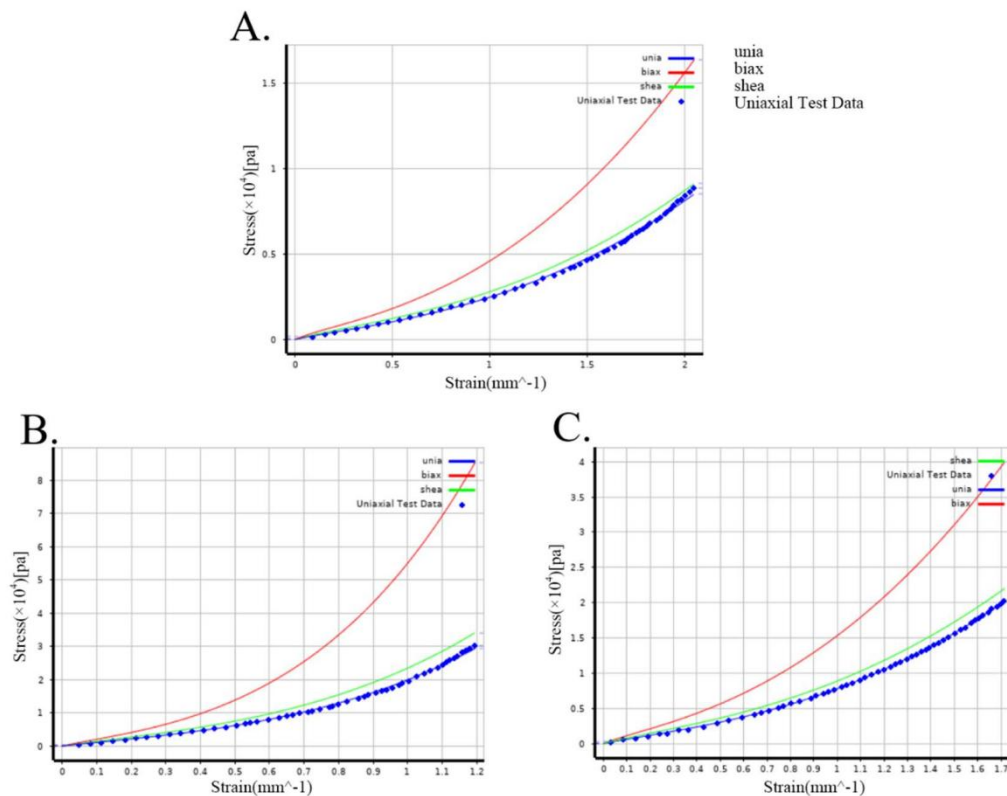


Figure 2. Elastic modulus (A. Uvula; B. Soft palate; and C. Tongue).

2.2.2. Airway wall simplification

The airway wall was modeled as a rigid no-slip boundary based on the following considerations: (1) Clinical evidence indicates that soft-palate and tongue-base displacement are prominent components of collapse patterns, and DISE studies report that multilevel collapse configurations are common [16]; (2) biomechanical data show that pharyngeal wall deformation (< 0.5 mm) is negligible compared with tongue displacement (> 10 mm) [22,23]; and (3) modeling wall compliance would increase computational cost by 3–5 $\times$ without significantly altering core outcomes (validated pressure errors $< 12\%$). The rigid-wall assumption applies to the nasal cavity, nasopharyngeal lateral walls, and other non-collapsible airway segments. In the tongue-collapse simulations (Section 3.3), a deformable posterior pharyngeal wall (Young's modulus 1.0×10^4 Pa) was introduced in the retroglossal region as a contact partner for the tongue; these two descriptions are complementary, not contradictory. The

maxilla serves as a displacement constraint (fully constrained boundary), with contact stiffness assigned to represent its interaction with the tongue. The friction coefficient of 0.1 was taken from an in vitro tongue-surface friction study [28]; sensitivity to frictionless ($\mu = 0$) and higher-friction ($\mu = 0.3$) conditions is reported in the sensitivity analysis.

2.3. Model meshing

Ansys 22.0 (Ansys Co., Ltd., USA) was used to mesh the upper airway, soft palate, uvula, and tongue models. A four-node linear tetrahedral mesh was selected. After mesh independence verification, the mesh size was set to 0.8 mm; the numbers of nodes and elements for each part are presented in Table 2.

Table 2. Numbers of nodes and meshes of the model.

	Upper airway	Soft palate	Uvula	Tongue
Number of nodes	152,290	159,987	6,552	1,524,863
Number of meshes	781,402	111,753	3,943	1,113,334

Mesh quality was evaluated using the skewness criterion and orthogonal quality (Table 3). All quality metrics fell within acceptable ranges; thus, this mesh configuration was adopted as the computational model.

Table 3. Mesh quality.

	Upper airway	Soft palate	Uvula	Tongue
Skewness	0.22	0.21	0.34	0.21
Orthogonal quality	0.77	0.79	0.75	0.79

Mesh independence verification (Tables 2–3) confirmed that the optimized mesh size (0.8 mm) minimizes discretization errors while maintaining computational efficiency. Sensitivity tests showed that further refinement (< 0.8 mm) altered key outputs (inlet pressure, maximum velocity) by less than 1.5%, confirming result stability against mesh variations. A systematic mesh-convergence study was performed across four mesh densities (1.6, 1.2, 0.8, and 0.6 mm), corresponding to three successive refinement steps; the actual mesh sizes, element counts, and changes in pressure drop, peak velocity, tissue displacement, minimum cross-sectional area, and the contact-transition point at each level are available from the corresponding author upon reasonable request. Boundary-layer resolution, inflation-layer parameters (first-layer height, growth rate, layer count), and y^+ values are also available on request.

2.4. Turbulence model

The Realizable k – ϵ model was selected for its convergence robustness in strongly coupled FSI with large deformation and moving boundaries. A turbulence-model comparison at selected load levels (laminar, Realizable k – ϵ , and SST k – ω) is available from the corresponding author upon reasonable request, together with the sensitivity of pressure drop, peak velocity, and the contact-transition point to model choice. Reynolds numbers at the inlet and at constricted regions are reported. The governing

equations (Navier–Stokes), air viscosity (1.81×10^{-5} Pa·s at 37°C), incompressibility assumption, inlet turbulence intensity and length scale, wall treatment, convergence criteria, and coupling procedure are specified below.

2.5. Boundary conditions of the fluid domain

Bidirectional fluid-structure interaction analysis was performed to investigate the interaction between the upper airway and the soft palate/tongue during collapse. The model was divided into a fluid domain (upper airway) and a solid domain (soft palate and tongue). Only nasal breathing was considered, as it provides air filtration, heating, and humidification. The upper airway model and boundary conditions are shown in Figure 3. The inlet condition was set as a sinusoidally varying mass inflow (Figure 3B) [29] with a peak inspiratory flow rate of 25 L/min, reflecting typical resting tidal breathing in healthy adults [29]. Mass flow boundary condition was adopted over velocity to ensure consistent volumetric flow rate regardless of potential inlet area variations during tissue deformation, consistent with clinical spirometric measurements. Simulations covered three respiratory cycles (12 s total duration) with a fixed time step of 0.01 s, determined by the Courant-Friedrichs-Lewy (CFL) condition. The respiratory frequency was 15 cycles/min. Air density was 1.139 kg/m^3 at 37 °C, and the peak mass flow rate was $0.475 \times 10^{-3} \text{ kg/s}$. The reconstructed airway model extended only to the pharyngeal region. When the upper airway was patent, it was connected to the external environment; thus, the outlet was set to the zero-pressure boundary condition. Given that airway wall deformation was substantially smaller than that of the soft palate and tongue, the airway wall surface was assumed to be rigid with a no-slip condition [22,23]. FSI coupling parameters were: 20 subiterations per time step, convergence tolerance of 1×10^{-4} for fluid and solid residuals, dynamic-mesh method with spring-based smoothing and local remeshing, remeshing triggered when minimum orthogonal quality falls below 0.15 or maximum skewness exceeds 0.85, contact stabilization with a normal contact stiffness of 0.1, and near-closure cell treatment by aggressive local refinement.

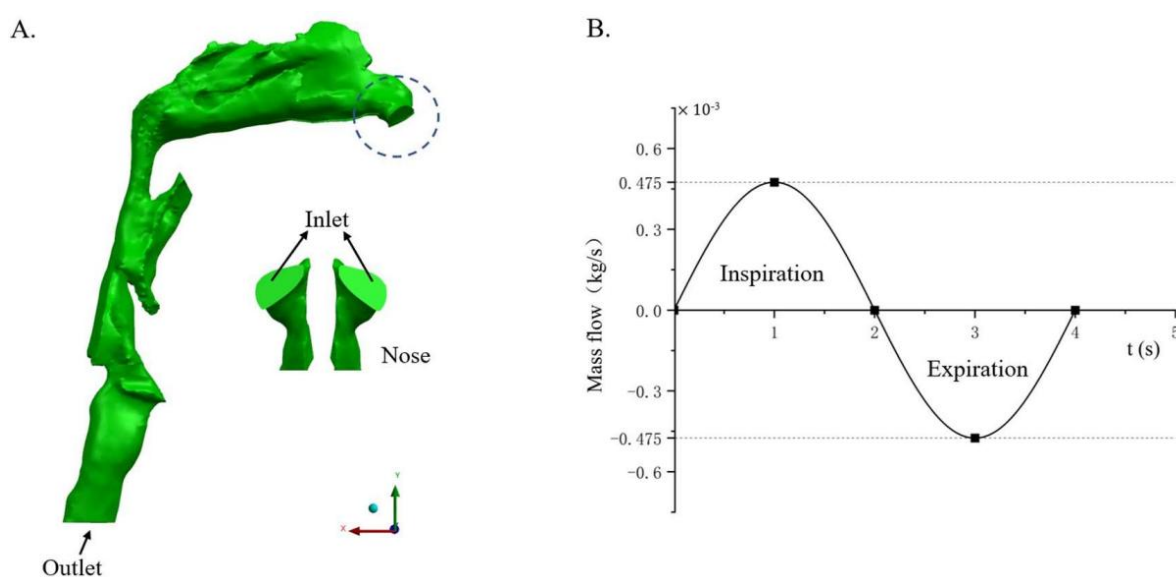


Figure 3. Fluid domain and boundary conditions (A. Model inlet and outlet; and B. Mass-flow curve of a single respiratory cycle).

Limitation of the prescribed-flow boundary condition: Imposing a fixed mass inflow forces the selected flow through the airway even when the lumen becomes severely narrowed. The calculation therefore determined the inlet pressure required to maintain the prescribed flow, not the spontaneous inspiratory flow that would result from a given respiratory effort. This approach did not reproduce inspiratory flow limitation, airflow cessation during obstructive apnea, or the interaction between respiratory effort and a collapsible airway. Consequently, the computed pressures were engineering metrics (pressure required to sustain prescribed flow through a deformed airway) and were not interchangeable with physiological driving pressures or therapeutic CPAP settings. All pressure values were time-averaged over the peak inspiratory phase ($t = 9\text{--}11$ s of the third cycle) at the nostril inlet unless otherwise stated. Temporal-convergence verification: Results are also compared at time steps of 0.005 s and 0.0025 s; the CFL condition in the fluid domain alone does not establish time-step independence of the coupled FSI solution.

2.6. Statistical analysis

Numerical verification and sensitivity analysis: Because the simulations are deterministic outputs at investigator-selected input values rather than independent biological observations, inferential statistics (ANOVA, p-values, effect sizes) are not applicable. Simulation curves are reported descriptively. Sensitivity of key outputs (inlet pressure, maximum velocity, tissue displacement, contact-transition point) was assessed with respect to mesh density (four systematically refined levels, 1.6–0.6 mm), temporal discretization (time steps of 0.01, 0.005, and 0.0025 s), material property variation ($\pm 20\%$ in hyperelastic coefficients), friction coefficient (0, 0.1, 0.3), inlet flow rate ($\pm 10\%$), and reference support loads ($\pm 10\%$). Uncertainty bands attributable to these sources accompany the reported curves. Moreover, where a transition point is reported, it is defined prospectively as the parametric level at which the nonlinear increase in ventilation pressure occurs (the level beyond which appreciable tongue–soft palate compression is established), and its sensitivity to the above parameters is reported as a range.

2.7. Model validation

To establish the predictive credibility of the fluid-structure interaction framework, a multi-modal validation protocol was implemented against clinical and experimental datasets. The following comparisons were explicitly classified by verification level: code verification, numerical verification, and physiological benchmarking. Cross-subject comparisons were labeled as benchmarking rather than subject-specific validation.

Geometric fidelity assessment (benchmarking level): The reconstructed upper airway morphology was quantitatively compared with MRI scans of the same subject where available. Cross-sectional areas at five clinically relevant obstruction sites (retropalatal and retroglossal regions) demonstrated less than 5% deviation from ground-truth anatomical measurements, consistent with segmentation accuracy benchmarks in airway modeling studies [30]. Pressure-flow relationship verification (benchmarking level): Steady-state CFD simulations at 15 L/min and 30 L/min flow rates were compared against published in vivo manometry data from healthy volunteers at comparable flow rates [30]. The simulated pressure gradient (nasopharynx to hypopharynx) exhibited less than 12% deviation from published clinical measurements across flow regimes.

Soft tissue kinematics (benchmarking level, not subject-specific validation): Soft palate displacement under -300 Pa suction pressure was benchmarked against dynamic MRI studies of airway collapse [19]. Maximum deformation discrepancies remained below 15% (2.1 mm simulated vs. 2.4 mm measured, from a different subject). Comparing a simulated displacement from this anatomy with a reported displacement from another study constitutes benchmarking, not subject-specific validation.

3. Results

3.1. Upper airway flow field analysis

The baseline flow field (Figure 4) served as a critical benchmark for model validity. Under peak inspiration (25 L/min), the simulated pressure drop ($\Delta P = 37$ Pa) is consistent with other reported values in healthy upper airways at comparable flow rates [30]. Velocity magnitudes at the velopharynx (4.8–6.5 m/s) are reported descriptively. These agreements support the model's capability to replicate broad flow patterns at the benchmarking level.

Because apnea occurs primarily during the inspiratory phase, only inspiratory flow field changes were examined. Monitoring over one respiratory cycle revealed that upper airway pressure increased with inspiratory flow rate. At 1 s, inspiratory flow reached its maximum value; the corresponding pressure and velocity distributions are shown in Figure 4. Pressure was highest in the nasal vestibule and decreased gradually along the airflow direction. Certain regions exhibited distinctive characteristics, including the transition zones from the nasal vestibule to the nasal cavity, from the nasopharynx to the oropharynx, and from the oropharynx to the laryngopharynx, as well as the boundary between the esophagus and trachea. Owing to anatomical constrictions, these regions generated relatively high airflow velocities, with a maximum speed of 6.52 m/s, corresponding to localized low-pressure zones. The average inlet pressure at this time was approximately 37 Pa, representing the pressure generated at the nostrils of a healthy individual producing a normal inspiratory flow.

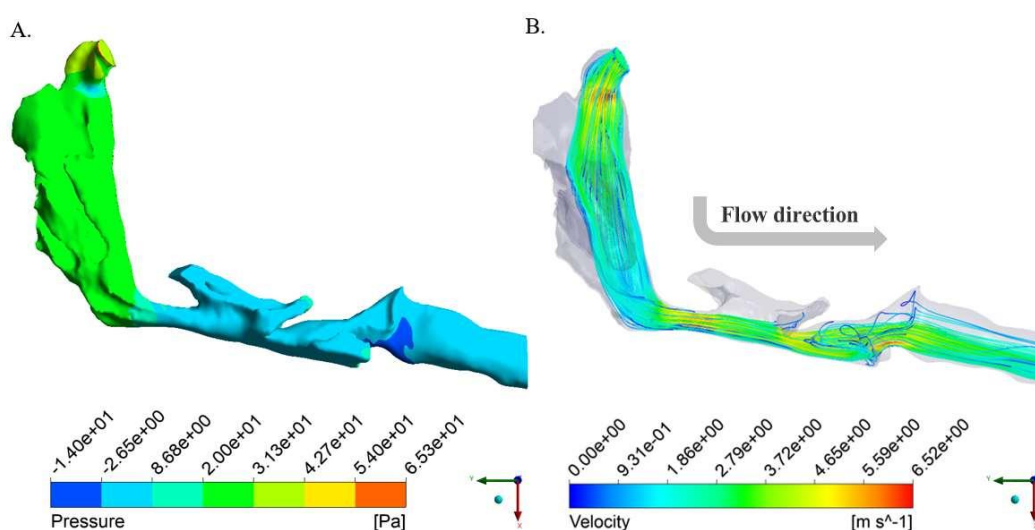


Figure 4. Flow field in the upper airway (A. Pressure contour; and B. Streamlines).

3.2. Impact of soft palate collapse on flow field of upper airway

3.2.1. Solid domain boundary conditions

As shown in Figure 1, soft palate collapse does not affect the tongue. Therefore, all geometries except the soft palate were suppressed. The fluid domain boundary conditions were set as described above. Soft palate collapse is associated with tensor veli palatini muscle relaxation [31]; however, the simplified soft palate model cannot directly indicate the location of the tensor veli palatini. To simplify the calculation, only the force generated by the tensor veli palatini against gravity (negative X-axis direction) was considered. A surface approximately flush with the tensor veli palatini was selected as the loading surface (Figure 5), and different pressures were applied to simulate changes in muscle tone. Fluid-structure interaction interfaces were defined at the intersections of the fluid and solid domains for data transfer. The transition of the soft palate from a healthy position to a stable deformation with inspiratory flow under varying muscle tones was investigated. After the second respiratory cycle, the soft palate exhibited stable periodic motion; therefore, the analysis focused on the inspiratory phase (9–11 s) of the third respiratory cycle.



Figure 5. Soft palate boundary conditions. The loading surface (red) approximates the tensor veli palatini insertion, with pressure applied in the negative X-axis direction to simulate muscle tone opposing gravity.

3.2.2. Effects of soft palate muscle tone loss on airway flow dynamics

The degree of soft palate collapse was quantified by the extent of muscle tone loss. To investigate the effects of soft palate collapse on the upper airway flow field under different muscle tones, the muscle tone required to maintain the soft palate in a normal state was first determined. The maximum deformation of the soft palate was only 0.15 mm at $P = 1000$ Pa; thus, $P = 1000$ Pa was selected as the healthy muscle tone. This selection is a calibration: The pressure was chosen because it produces a displacement deemed compatible with the healthy state, and it is not derived from physiological muscle-force measurements. It is hereafter termed the reference support load. Pressures of 0, 100, 200, ..., 900 Pa were applied to simulate varying degrees of tensor veli palatini muscle tone loss. The case of $P = 0$ Pa represents complete loss of muscle tone, where the soft palate was subjected solely to gravity. Note that even at $P = 0$ Pa the tissue retains passive hyperelastic stiffness and attachment constraints; “complete loss” refers to removal of the artificial support load, not loss of all active and passive muscular properties. The collapse morphology under this condition at peak inspiration is shown in Figure 6.

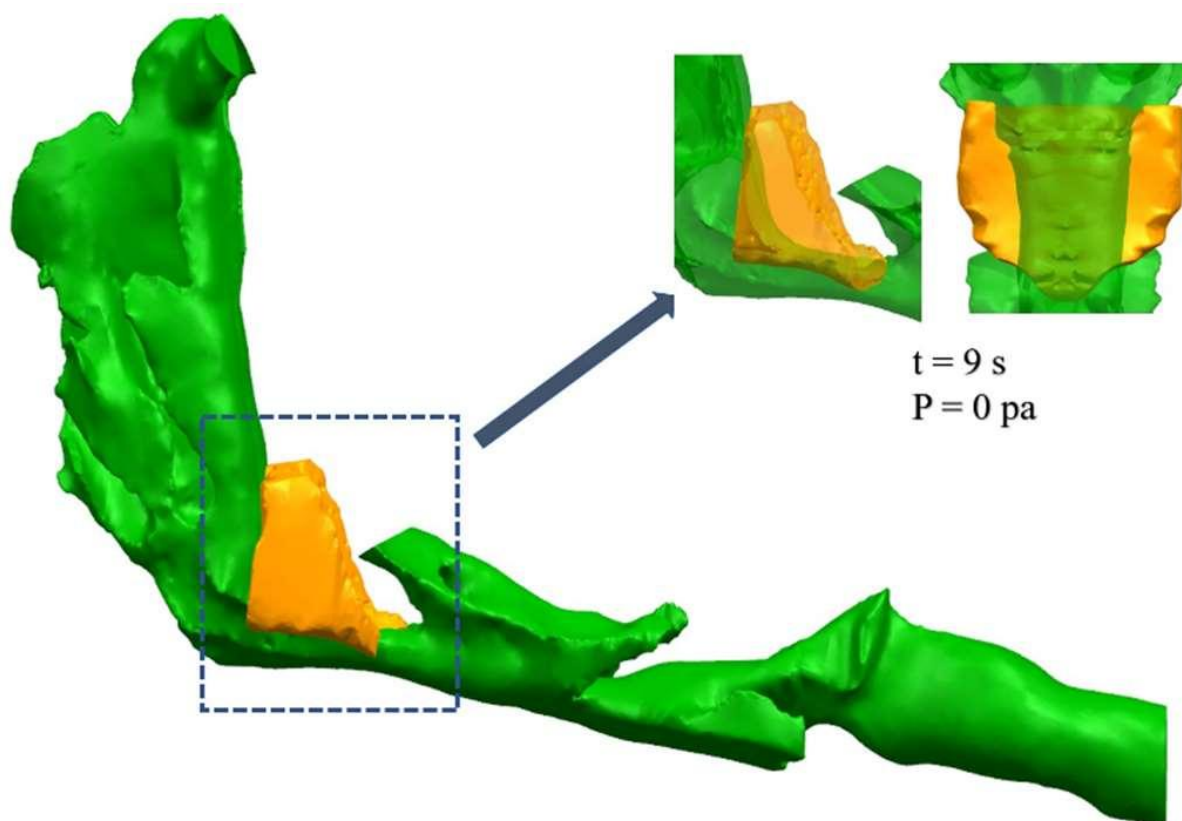


Figure 6. Soft palate collapse morphology under complete muscle tone loss ($P_s = 0 \text{ Pa}$) at peak inspiration ($t = 9 \text{ s}$).

The pressure distribution in the upper airway and the deformation of the soft palate are shown in Figure 7A,B, respectively. The average inlet pressure required to maintain normal respiration with complete loss of soft palate muscle tone was 146 Pa. Since the pressure generated in the nasal cavity during normal respiration is approximately 37 Pa, the additional ventilation pressure required was 109 Pa. The deformation of the soft palate increased gradually in the negative Y-axis direction under the combined effects of gravity and the upper airway flow field. The velocity traces of the upper airway are shown in Figure 7C. The maximum velocity (15.9 m/s) occurred at the region of greatest collapse. For reference, the maximum flow velocity at an inspiratory flow rate of 60 L/min in a separate healthy-airway calculation was 24.2 m/s [18]; however, this value is a descriptive observation from one condition and does not constitute a validated epithelial injury threshold. Velocity is not wall shear stress; assessment of epithelial loading would require near-wall resolution and a validated injury criterion, which are beyond our scope of this study. Isolated soft palate collapse (maximum velocity: 15.9 m/s) remained below this reference value under the modeled conditions.

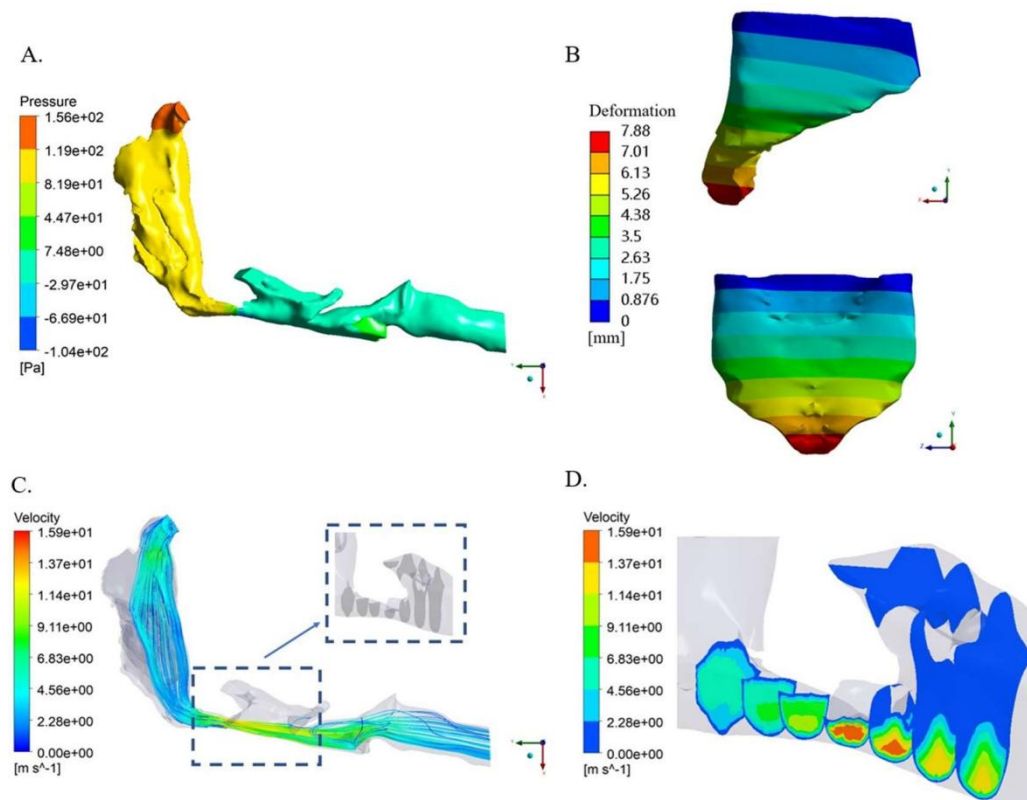


Figure 7. Simulation results for the upper airway and soft palate (A. Upper airway pressure contour; B. Soft palate deformation contour; C. Streamlines; and D. Cross-sectional velocity contour).

It should be noted that the muscle tone was approximated by selecting a position near the tensor veli palatini and considering only the gravitational component. Therefore, the absolute pressure values reported above are not equivalent to the actual physiological muscle tone. The applied surface pressures are simplified external loads representing only the gravitational-opposing component of muscle function; they do not simulate active muscle contraction, neuromuscular recruitment, fiber orientation, or activation-dependent constitutive behavior. The reference values ($P_s = 1000$ Pa, $P_t = 1900$ Pa) were calibrated to produce displacements compatible with a healthy state, and all percentages are normalized to these calibration-based reference values. In the following sections, load reduction is expressed as a percentage of the reference support load, defined as $(P_{ref} - P_{applied}) / P_{ref} \times 100$.

As shown in Figure 8, with progressive muscle tone loss, the rate of change in maximum soft palate deformation diminished, whereas the effects on ventilation pressure and velocity intensified. At 0–20% muscle tone loss, the maximum deformation of the soft palate was approximately 3 mm, with negligible impact on ventilation pressure and maximum velocity. Between 70% and 100% loss, the soft palate produced only 1.4 mm of additional deformation, while ventilation pressure increased by 179% and maximum velocity increased by 46%. Even with complete muscle tone loss, the maximum required ventilation pressure was only 110 Pa and the maximum velocity was only 15.9 m/s. These results indicate that isolated soft palate collapse, under the modeled loading conditions, does not produce a marked increase in the pressure required to maintain the prescribed flow.

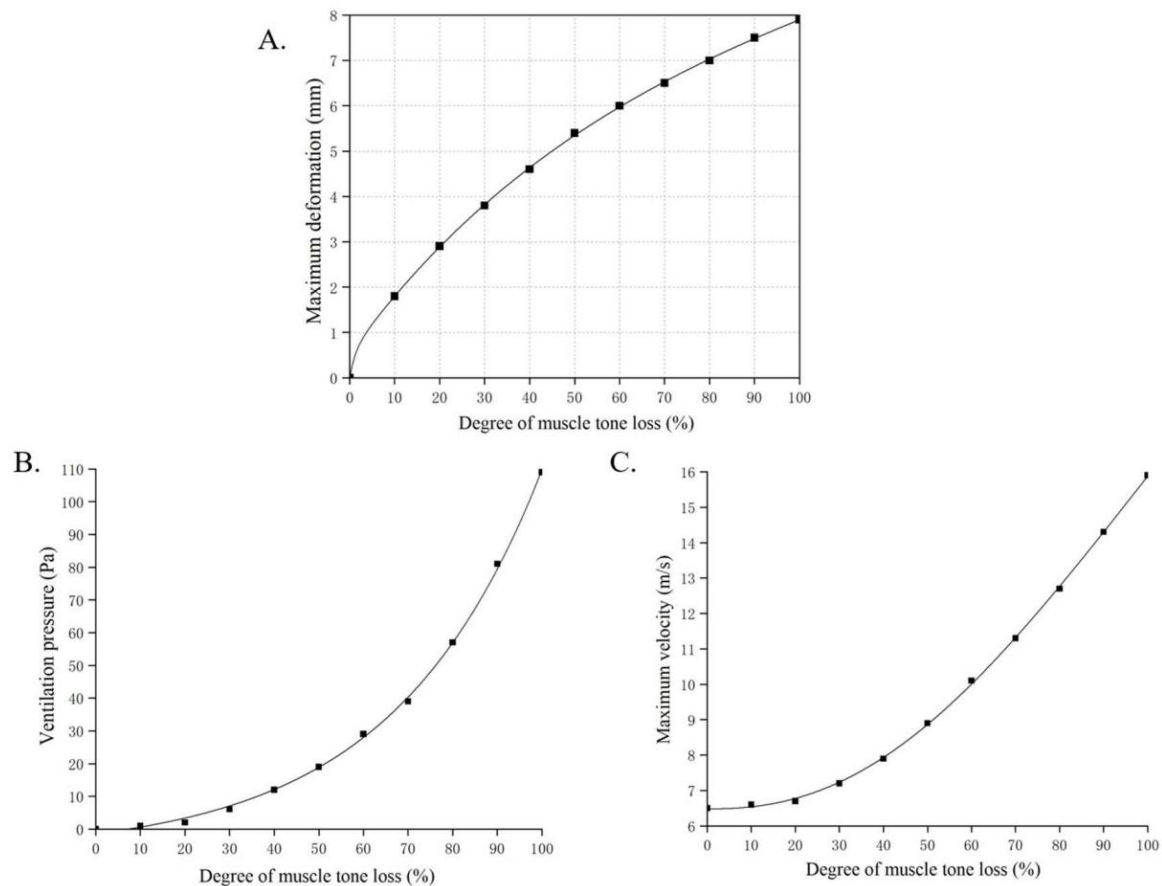


Figure 8. Effects of the degree of muscle tone loss of the soft palate on the soft palate and upper airway. A. Fitting curve of the relationship between the degree of muscle tone loss and the maximum deformation of the soft palate; B. Fitting curve of the relationship between the degree of muscle tone loss and the ventilation pressure; and C. Fitting curve of the relationship between the degree of muscle tone loss and the maximum velocity.

3.3. Impact of tongue collapse on flow field of upper airway

3.3.1. Solid domain boundary conditions

Tongue collapse involves greater displacement and is constrained by the maxilla and posterior pharyngeal wall. The maxilla model was extracted from CT images using the same segmentation approach described above. Since the maxilla serves only as a displacement constraint without undergoing deformation, a simplified model was constructed according to its anatomical dimensions (Figure 9A). The regions of the upper airway susceptible to collapse were identified and assigned thickness to generate a posterior pharyngeal wall model (Figure 9B).

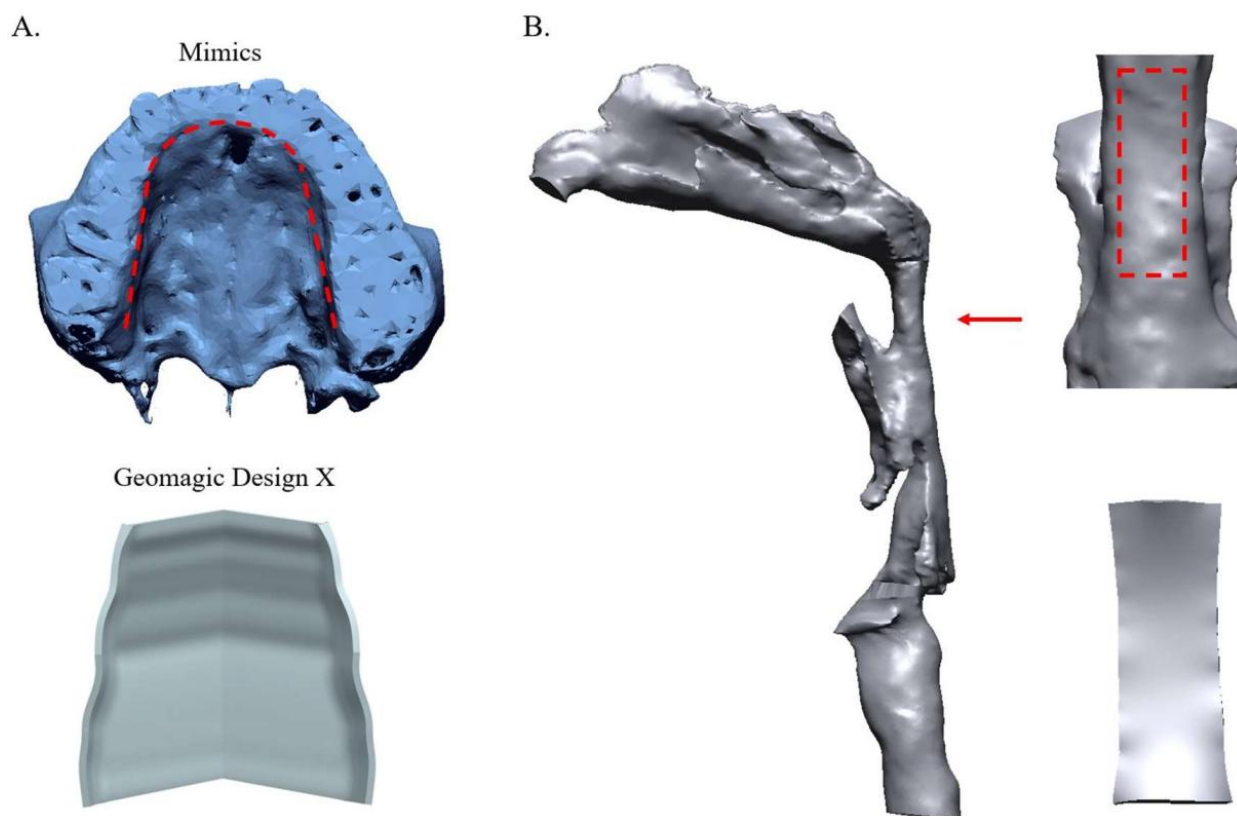


Figure 9. Anatomical constraint modeling. A. Simplified maxilla model extracted from CT images serving as a displacement constraint for the tongue; and B. Posterior pharyngeal wall model constructed from identified collapse-prone regions of the upper airway).

The material properties of the maxilla and posterior pharyngeal wall are presented in Table 4.

Table 4. Material properties of the maxilla and posterior pharyngeal wall.

Soft tissue	Density (kg/m ³)	Young's modulus	Poisson's ratio
Maxilla	1850	1.37×10^{10}	0.30
Posterior pharyngeal wall	1060	1.0×10^4	0.45

The boundary conditions for the tongue collapse simulation are shown in Figure 10. Tongue extension and contraction are primarily controlled by the genioglossus muscle [17,27,32]; thus, pressure was applied at a position approximating the genioglossus insertion to represent its muscle tone. As before, only the force component opposing gravity (negative X-axis direction) was considered. The soft palate boundary conditions were identical to those in the soft palate collapse case. The force applied to the soft palate is denoted as P_s ($P_s = 1000$ Pa for the healthy state). Frictional contact between structures was modeled with a friction coefficient of 0.1 [28].

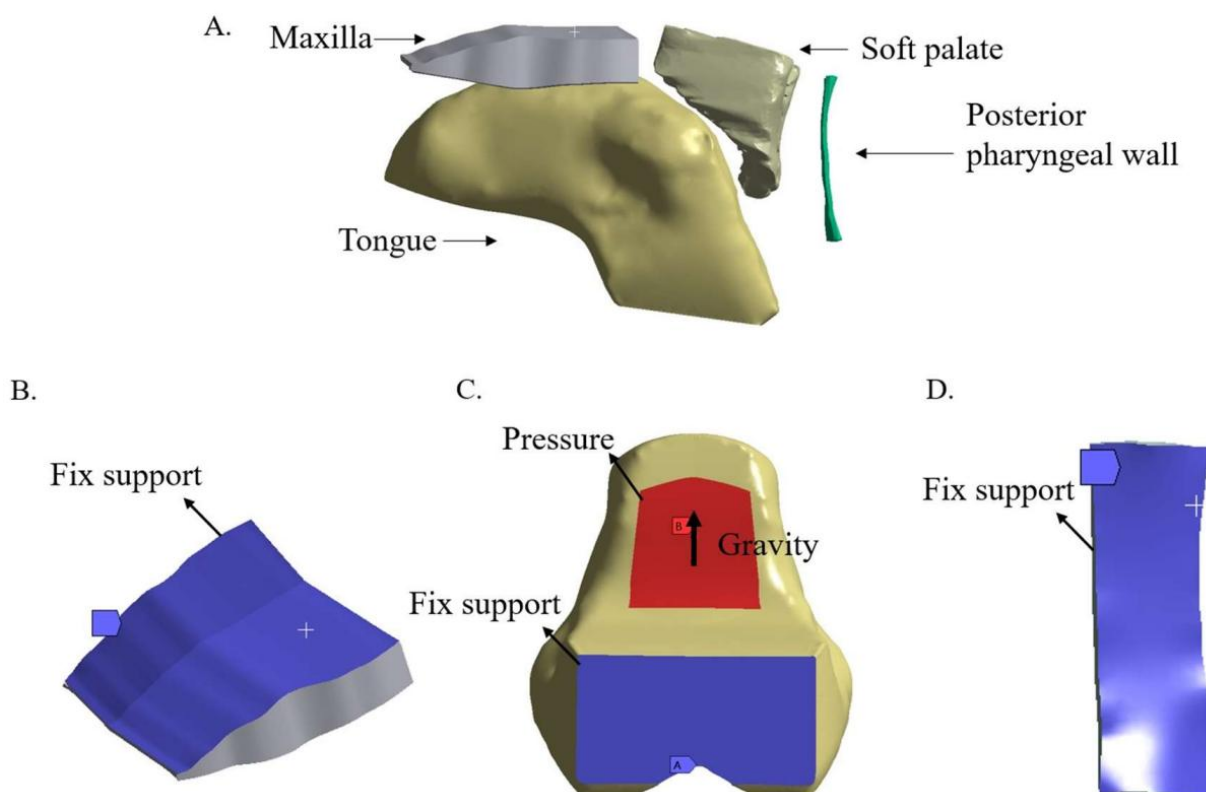


Figure 10. Boundary conditions of tongue collapse. A. Simulation model; B. Maxilla boundary conditions; C. Tongue boundary conditions; and D. Posterior pharyngeal wall boundary conditions).

3.3.2. Tongue collapse-induced flow obstruction and palatal contact effects

The effects of tongue collapse on the upper airway flow field were investigated under varying genioglossus muscle tones (Pt). Simulation results indicated that tongue collapse did not affect the upper airway flow field prior to contact with the soft palate; its effects manifested only after the tongue contacted and exerted pressure on the soft palate, causing additional deformation. Therefore, it was necessary to determine the muscle tones corresponding to a healthy tongue state and to the onset of tongue–soft palate contact. The maximum deformation of the tongue was only 0.24 mm at Pt = 1900 Pa; thus, Pt = 1900 Pa was selected as the healthy genioglossus muscle tone. As with the soft palate, this value is a calibration-based reference load, not a physiologically measured muscle force. Tongue–soft palate contact occurred at Pt = 650 Pa. For Pt < 650 Pa, the tongue contacted and compressed the soft palate. The changes in the upper airway flow field, tongue, and soft palate were investigated by setting Pt to 0, 65, 130, ..., 650 Pa. The case Pt = 0 Pa corresponds to complete loss of genioglossus muscle tone, with only gravity acting on the tongue. The parametric sweep is discrete (65 Pa increments), so reported transition levels reflect the sweep resolution. During the inspiratory phase, upper airway pressure increased with inspiratory flow. At 9 s, inspiratory flow reached its maximum, at which point the overall upper airway pressure was maximal and the tongue and soft palate collapsed to the positions shown in Figure 11.

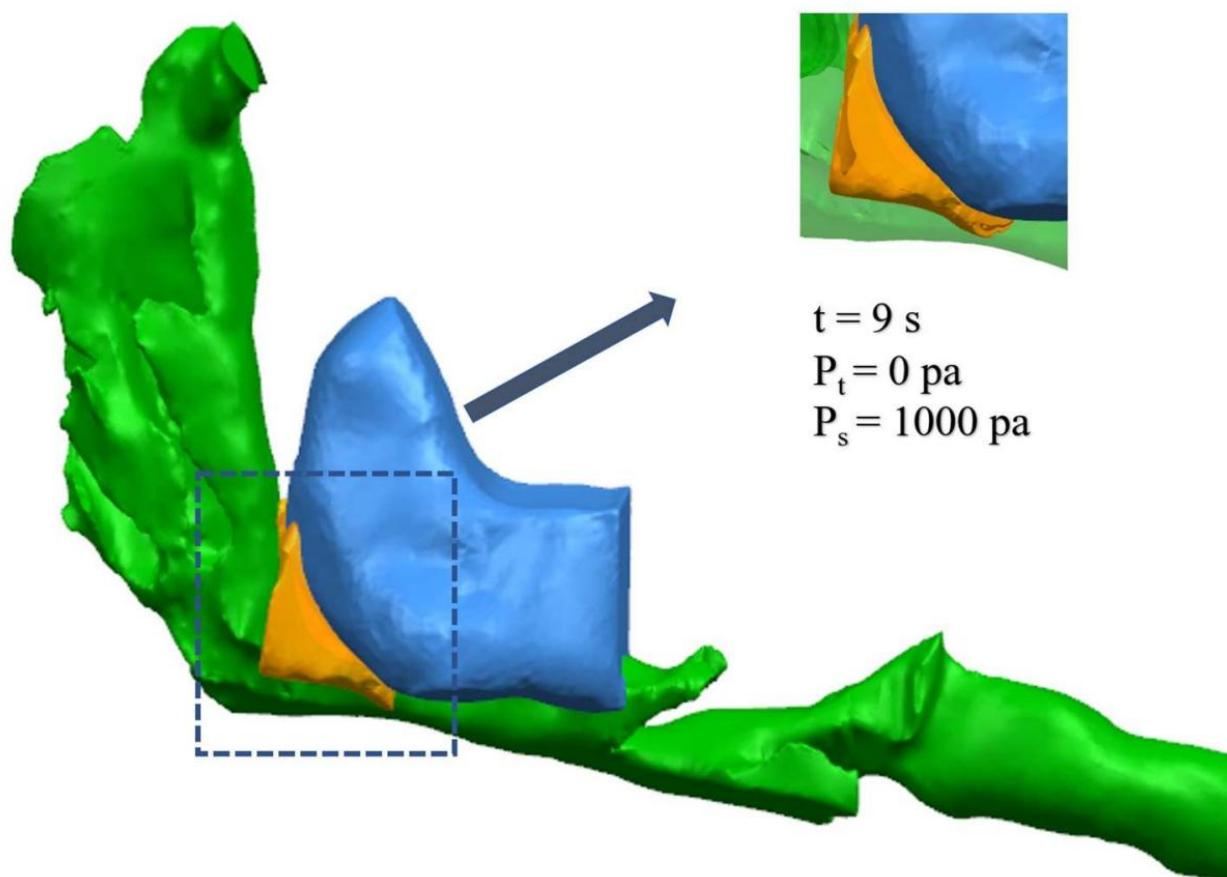


Figure 11. Tongue and soft palate collapse morphology under complete genioglossus muscle tone loss ($P_t = 0$ Pa) at peak inspiration ($t = 9$ s), showing tongue–soft palate contact.

The upper airway pressure distribution and the deformation of the tongue and soft palate are shown in Figure 12A,B, respectively. To maintain normal breathing with complete loss of tongue muscle tone, the average inlet pressure required was 879 Pa, yielding a ventilation pressure of 852 Pa. The maximum deformation of the tongue was approximately 23.8 mm, occurring at the superior surface. The pressure exerted by the tongue on the soft palate produced a maximum soft palate deformation of approximately 10.2 mm at the uvula.

The corresponding velocity traces and cross-sectional velocity distribution are shown in Figure 12C, D, respectively. The maximum velocity (approximately 46.3 m/s) occurred at the uvula, the site of greatest deformation. This velocity substantially exceeds the reference value of 24.2 m/s observed at 60 L/min in a healthy-airway calculation; however, no validated epithelial injury criterion is applied in this study, and no inference about mucosal damage is drawn.

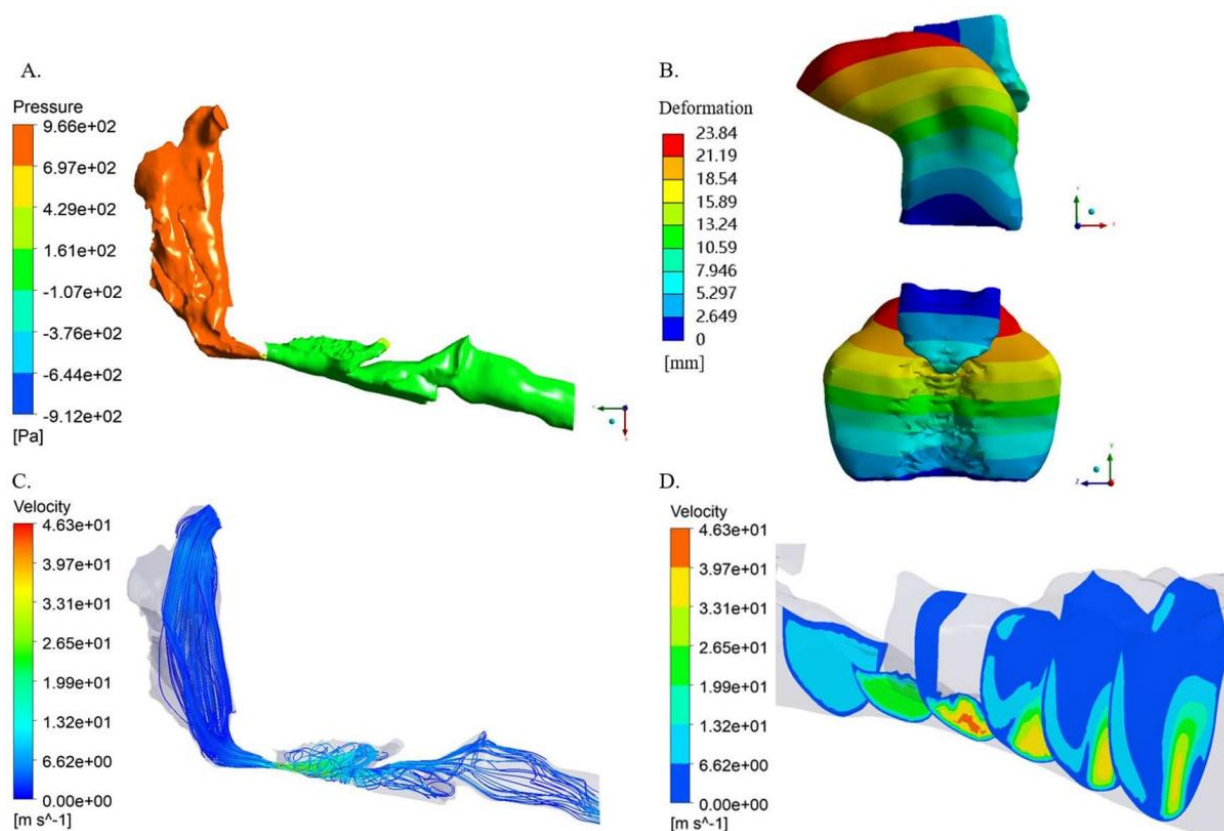


Figure 12. Simulation results for the upper airway, tongue, and soft palate. A. Upper airway pressure contour; B. Deformation contours of the tongue and soft palate; C. Streamlines; and D. Cross-sectional velocity contour.

Since the muscle tone required to maintain the tongue in a normal state was approximately 1900 Pa, and effects on the soft palate and upper airway were observed only when $P_t < 650$ Pa (corresponding to $> 65.7\%$ muscle tone loss), the analysis focused on muscle tone loss in the range of $65.7\text{--}100\%$. The maximum deformation of the soft palate, the required ventilation pressure, and the maximum velocity of the upper airway under different degrees of muscle tone loss are presented in Figure 13.

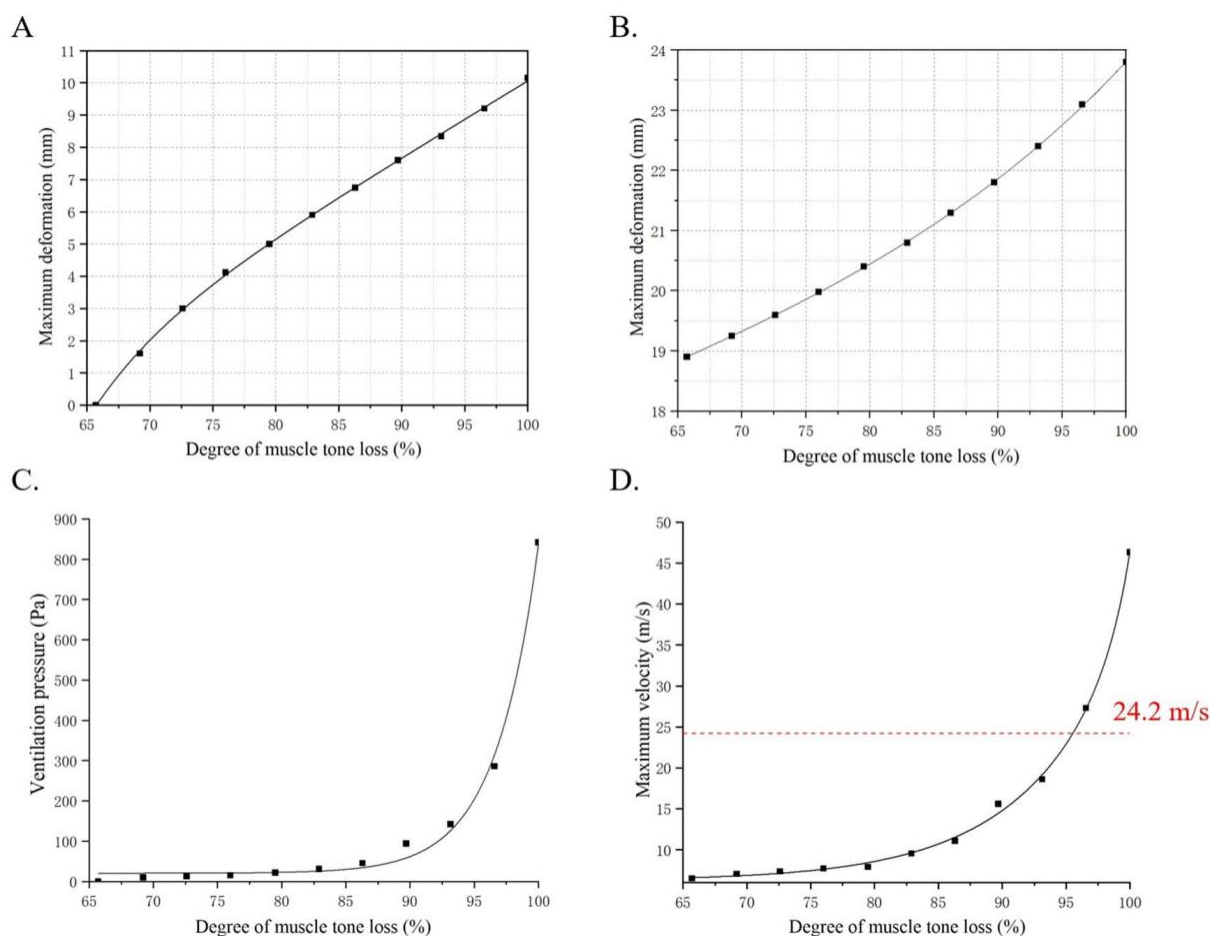


Figure 13. Effects of the degree of muscle tone loss of the tongue on the soft palate and upper airway (A. Fitting curve of the relationship between the degree of muscle tone loss and the maximum deformation of the soft palate; B. Fitting curve of the relationship between the degree of muscle tone loss and the maximum deformation of tongue; C. Fitting curve of the relationship between the degree of muscle tone loss and the ventilation pressure; D. Fitting curve of the relationship between the degree of muscle tone loss and the maximum velocity).

As shown in Figure 13, when tongue muscle tone loss was in the range of 65–90%, soft palate deformation increased from 0 to 7.7 mm, the ventilation pressure was 94 Pa, and the maximum velocity was 15.6 m/s; values comparable to those observed in the soft-palate-only collapse case. When tongue muscle tone loss was between 90% and 93%, the deformation of tongue and soft palate exerted increasingly pronounced effects: maximum deformation increased by approximately 1 mm, ventilation pressure increased by approximately 140%, and maximum velocity increased by approximately 56.7%. When tongue muscle tone loss exceeded 93% (i.e., $P_t \leq 130$ Pa in the discrete sweep), the effects became substantial: Maximum deformation increased by a further ~ 1 mm, the ventilation pressure required to maintain the prescribed flow increased by approximately 806% (from 94 Pa to 852 Pa), and the maximum velocity increased by approximately 97%. The onset of node-level tongue–soft palate contact occurred earlier, at $P_t = 650$ Pa ($\sim 65.7\%$ load reduction), whereas the nonlinear pressure transition occurred at $P_t \leq 130$ Pa ($\sim 93\%$ load reduction); these two conditions are distinct.

3.4. Effects of joint collapse of soft palate and tongue on the flow field of the upper airway

3.4.1. Solid domain boundary conditions

The solid domain boundary conditions were essentially identical to those described in Section 3.3, except that the pressure on the soft palate (P_s) was varied to simulate different degrees of soft palate collapse.

3.4.2. Dominant role of tongue collapse in joint obstruction scenarios

To investigate the combined effects of soft palate and tongue collapse on the upper airway flow field, different degrees of soft palate collapse were simulated by varying P_s . Complete soft palate collapse corresponded to $P_s = 0$ Pa. The maximum deformation of the soft palate and tongue, the required ventilation pressure, and the maximum velocity under varying degrees of tongue muscle tone loss were determined and compared with the tongue-collapse-only case.

As shown in Figure 14A, when tongue muscle tone loss was in the range of 0–93%, soft palate deformation was driven solely by gravity. Under these conditions, no appreciable tongue–soft palate compression was established and the tongue did not induce additional deformation. When tongue muscle tone loss exceeded 93% (corresponding to the establishment of appreciable tongue–soft palate compression; node-level contact begins earlier, at ~65.7% load reduction), the soft palate came into contact with the tongue; however, the soft palate deformation was similar between the healthy-state and complete-collapse conditions. As shown in Figure 14B, the soft palate status had essentially no effect on tongue deformation in either the healthy or no-muscle-tone state.

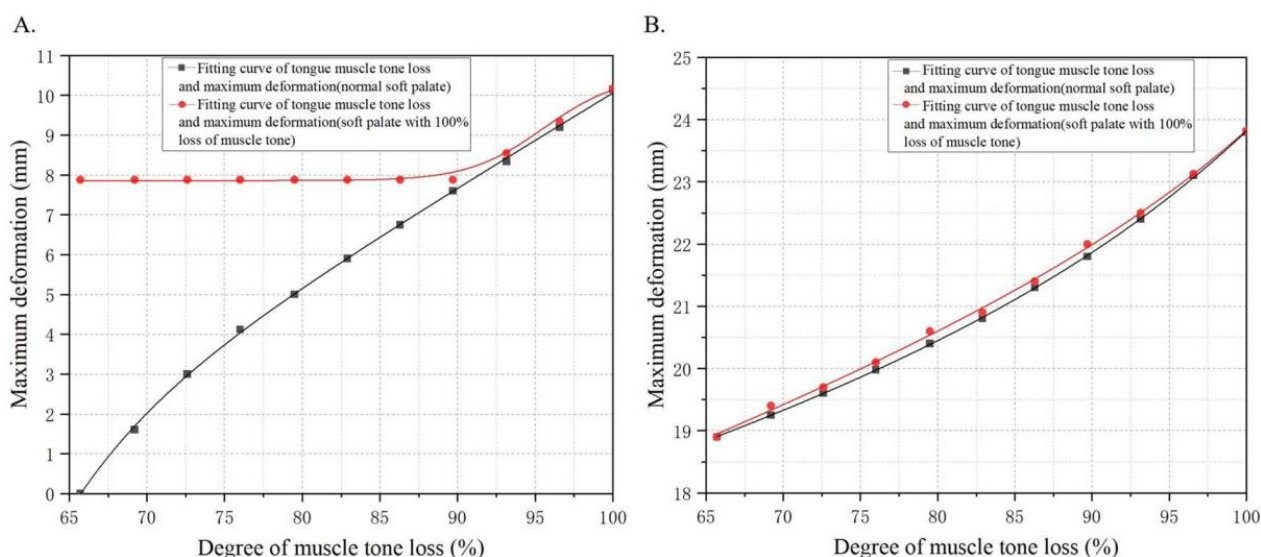


Figure 14. Comparison of the maximum deformation of the soft palate and tongue in the cases of joint collapse and tongue collapse alone. A. Fitting curve of the relationship between the degree of muscle tone loss and the maximum deformation of the soft palate; B. Fitting curve of the relationship between the degree of muscle tone loss and the maximum deformation of the tongue.

As shown in Figure 15, when tongue muscle tone loss was in the range of 0–93%, soft palate deformation was generated solely by gravity. In this regime, no appreciable tongue–soft palate compression occurred, and ventilation pressure and maximum velocity were governed by soft palate deformation, consistent with the soft-palate-only collapse results. When tongue muscle tone loss exceeded 93%, appreciable tongue–soft palate compression was established; however, the soft palate muscle tone did not significantly affect the deformation of either structure. Consequently, the ventilation pressure and maximum velocity curves for the healthy and completely collapsed soft palate conditions were essentially superimposable across all degrees of tongue muscle tone loss. These findings demonstrate that, in this model and under the selected loading conditions, once tongue collapse establishes contact with the soft palate, the reduction of the tongue support load becomes the dominant factor governing soft palate deformation and upper airway flow field alterations. This conclusion is specific to the present anatomy and modeling configuration and does not exclude contributions of other structures (e.g., lateral pharyngeal walls) in clinical collapse patterns.

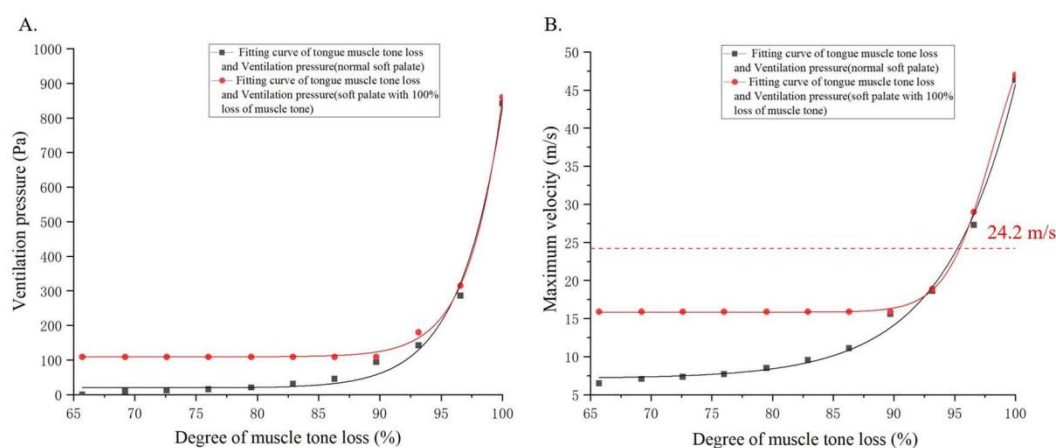


Figure 15. Comparison of the ventilation pressures and maximum velocities in the cases of joint collapse and tongue collapse alone; A. Fitting curve of the relationship between the degree of muscle tone loss and the ventilation pressure; and B. Fitting curve of the relationship between the degree of muscle tone loss and the maximum velocity).

4. Discussion

In this study, we set out to determine how progressive reduction of the support loads maintaining the soft palate and tongue alters upper airway flow, and which structure governs obstruction once collapse is initiated. Three findings answer this question. First, isolated palatal collapse, even at complete load removal, left the pressure required to maintain the prescribed flow essentially unchanged (~110 Pa; maximum velocity 15.9 m/s). Second, tongue behavior governed the transition to obstruction: No flow-field alteration occurred before appreciable tongue–soft palate compression was established, whereas once the tongue support load fell below approximately 93% of its calibrated reference value ($P_t \leq 130$ Pa in the discrete sweep; Figure 13C,D), the required ventilation pressure increased by approximately 806% (to 852 Pa), and the peak velocity reached 46.3 m/s. Third, in joint collapse scenarios, the ventilation pressure and velocity curves for healthy and fully collapsed soft-palate states were superimposable, indicating that tongue–palate interaction, not palatal tone,

dominates the mechanical outcome in this configuration. This nonlinear response is a model-specific contact transition reflecting the discrete parameter sweep, the single-subject anatomy, the calibrated reference load, the contact algorithm, and the mesh resolution; it should not be interpreted as a universal neuromuscular threshold. Within that qualification, in the following sections, we discuss the strengths, limitations, clinical context, and future directions of the work.

Identification of a model-specific contact transition: The nonlinear transition at ~93% reduction of the reference tongue support load was previously unreported in this parametric form. At this level, appreciable tongue–soft palate compression is established, and the ventilation pressure required to maintain the prescribed flow surges by approximately 806%, while peak airflow velocity reaches 46.3 m/s. This value reflects a contact transition observed at a discrete parametric level in a single-subject model and is not an independently estimated biological breakpoint; it has not been validated with uncertainty quantification against physiological measurements. Whether a comparable transition exists in patient populations requires multi-subject validation.

Reevaluation of collapse mechanisms: Isolated soft palate collapse did not produce a marked increase in the pressure required to maintain the prescribed flow under the modeled conditions (maximum velocity: 15.9 m/s). In this single-subject model, tongue-driven obstruction dominates through tongue–soft palate contact once the tongue support load falls below the transition level. This observation, restricted to the present anatomy and modeling configuration, may motivate future controlled parametric investigation of the relative contributions of palatal and lingual collapse; it does not, by itself, justify changes to surgical prioritization, which would require multiple patient-specific geometries, clinically characterized collapse patterns, and comparison with treatment outcomes.

Hypothesis for future validation: The model-specific contact transition identified here is not equivalent to Pcrit deterioration during REM sleep: Pcrit was neither imposed nor calculated, and no polysomnographic or electromyographic data were collected in this study. A normalized external load of 7% cannot be equated with clinical deterioration of critical closing pressure. Whether a relationship exists between progressive loss of neuromuscular support and Pcrit deterioration across sleep stages is a hypothesis requiring future validation with synchronized EMG, dynamic imaging, pressure-flow measurements, and polysomnography.

Methodological considerations: The representation of muscle function as a unidirectional anti-gravity surface pressure is a highly simplified external loading condition; it does not model active muscle contraction, neuromuscular recruitment, three-dimensional force transmission, recruitment in response to negative luminal pressure, or sleep-stage-dependent reflex control. The tensor veli palatini is not the sole determinant of soft-palate position, and the genioglossus is not the only muscle maintaining tongue and pharyngeal patency; the levator veli palatini, palatoglossus, palatopharyngeus, and other pharyngeal dilators also contribute, and genioglossus activation is spatially and temporally heterogeneous. The model therefore examines the consequences of reducing two artificial support loads and should be interpreted in precisely those terms. Although multidimensional force simulation would require substantially greater computational resources, future studies should integrate electromyography-guided multi-muscle activation patterns, supported by objective electrophysiological and polysomnographic characterization of sleep-related motor activity [33,34].

Strengths of the study: To our knowledge, this is the first upper airway FSI framework to explore a systematically parametric, bidirectional transient coupling of airflow with soft tissue deformation across the full 0–100% range of two independent support loads, rather than simulating a small number of fixed anatomical states. The framework captures tongue–soft palate frictional contact, large tissue

deformation (> 20 mm), and multi-cycle respiratory flow within a single transient analysis, and its single-subject, single-variable design deliberately isolates the biomechanical consequence of tongue–palate interaction from inter-individual anatomical confounding. The mesh, time-step, and material-parameter sensitivity analyses now accompanying the results further enable the uncertainty of the reported transition behavior to be quantified rather than merely asserted, and all non-identifiable computational files are available for independent replication.

Limitations faced in this work: (1) The reference support loads are calibration-based, not physiologically derived, so all percentage reductions are normalized to arbitrary reference values and cannot be equated with EMG-measured neuromuscular loss; (2) the prescribed-flow boundary condition computes the pressure required to sustain a fixed flow and does not reproduce spontaneous inspiratory flow limitation, airflow cessation during apnea, or the interaction between respiratory effort and a collapsible airway; (3) the single-subject design (one healthy 25-year-old male, awake supine CT) does not capture inter-individual pathological variants, lateral-wall or concentric collapse patterns, or sleep-stage-dependent reflex control; (4) the discrete parameter sweep (65 Pa increments) means the reported transition point reflects the sweep resolution, the contact algorithm, and the mesh; (5) the material parameters were measured from OSAS surgical specimens and applied to a healthy-subject model; and (6) inferential statistics are not applicable to deterministic simulations, so conclusions rest on descriptive curves plus numerical verification rather than hypothesis testing. These limitations collectively bound the interpretability of the results and define the agenda for the future work outlined below.

Clinical relevance: The clinical significance of these computational observations lies in hypothesis generation rather than in direct treatment guidance. The finding that obstruction in this model arises only after tongue–palate contact is established is mechanistically consistent with the clinical emphasis on upper airway muscle function in OSAS management. Randomized evidence shows that structured respiratory training can measurably improve apnea-hypopnea index (AHI), blood pressure, daytime sleepiness, and sleep quality in OSAS patients [35], and that inspiratory muscle training improves fatigue, aerobic capacity, pulmonary function, and quality of life in other chronic conditions [36], while broader lifestyle-medicine and structured exercise interventions confer systemic benefits in chronic disease populations [37,38]. Taken together with this model, these lines of evidence suggest a coherent research agenda: If progressive loss of dilator support precipitates a nonlinear mechanical transition as simulated here, then interventions that preserve or restore neuromuscular support, including respiratory and inspiratory muscle training, may act, in part, by keeping the system on the sub-transition side of that contact threshold. Testing this hypothesis will require the multi-subject, EMG-validated modeling outlined below, integrated with controlled trials of active conservative interventions of the kind synthesized in umbrella reviews.

Pathophysiological implications: The model-specific contact transition establishes a biomechanical hypothesis concerning airway obstruction. No correspondence with Pcrit deterioration during REM sleep or with polysomnographic patterns is claimed: Pcrit was neither imposed nor calculated, and no polysomnographic data were collected. Validating any relationship between progressive loss of neuromuscular support and clinical deterioration across sleep stages will require synchronized EMG, dynamic imaging, pressure-flow measurements, and polysomnography. Methodological guidance for objective electrophysiological assessment in sleep apnea populations is available in studies of portable EMG devices [33] and instrumental assessment frameworks [34], which underscore the need to validate computational muscle-activation parameters against objectively

recorded physiological signals before relating them to sleep stage or obstruction severity.

Advances in deep learning have demonstrated that transformer-based architectures can achieve robust medical image segmentation from limited datasets. Adapting such architectures to automated upper airway segmentation could preserve anatomical fidelity while substantially reducing the manual reconstruction effort required for patient-specific modeling.

In future studies, researchers should prioritize:

Multi-subject validation: Application of this FSI framework to anatomical phenotypes (stratified by sex, BMI, and craniofacial features) to establish population-adjusted contact-transition ranges and to test whether the tongue-dominant behavior observed here generalizes across collapse phenotypes, including lateral-wall and concentric patterns.

Pathology-specific and sleep-stage-specific modeling: Incorporation of common OSAS-associated anatomical variants, epiglottic interaction, and multilevel collapse, together with sleep-stage-dependent loading informed by synchronized EMG and polysomnography, so that modeled support loads can be anchored to objectively recorded neuromuscular activity rather than calibrated values.

Boundary-condition realism: Replacement of the prescribed-flow inlet with pressure-driven conditions representing thoracic inspiratory effort and positive airway pressure, enabling simulation of spontaneous flow limitation, apnea dynamics, and direct comparison with P_{crit} measurements and CPAP titration responses in patients.

Intervention modeling: Coupling of this framework with the clinical evidence base for active conservative interventions, including Shitali and inspiratory muscle training [35,36] and lifestyle-medicine approaches [37,38], to test computationally whether augmented dilator support shifts patients away from the contact transition, and to generate quantitative, testable predictions for future randomized trials.

Clinical translation: Correlation of model-derived transition metrics with polysomnographic indices (AHI, flow-limitation contours, P_{crit}) and with treatment outcomes across patient subgroups, as a step toward patient-specific mechanistic phenotyping.

Clinical implications: The present single-subject, deterministic model with artificial loading cannot establish treatment recommendations. The following are hypotheses for future research, not clinical guidance: The identified contact-transition behavior, if validated across patient-specific geometries and clinically characterized collapse patterns, could contribute to a mechanistic understanding of the disproportionate increase in therapeutic pressure requirements observed in severe OSAS. The observation that isolated palatal collapse did not markedly affect the required ventilation pressure in this model may motivate investigation of collapse-pattern-specific treatment response; however, no recommendation concerning palatal surgery, tongue stabilization, or CPAP titration protocols can be drawn from the present results. An umbrella review of active conservative interventions for OSAS [39] synthesizes the clinical evidence on interventions targeting upper-airway muscle function; our computational findings generate biomechanical hypotheses that should be examined alongside such clinical evidence before any translational claims are made, and a model-specific reduction in an externally applied tongue load must not be taken to predict the response to myofunctional therapy or other conservative interventions.

Comparison with prior work: FSI studies of the upper airway have primarily examined fixed anatomical states [4,7,9], whereas our parametric framework systematically explores a spectrum of support-load reduction. The predicted ventilation pressures (94–852 Pa) are the inlet pressures required to maintain the prescribed flow through the deformed airway; they are not interchangeable with

therapeutic CPAP settings. Physiological inspiration is driven by a downstream pressure gradient, whereas CPAP applies positive luminal pressure to splint the airway; numerical similarity between the computed values and clinical CPAP ranges would not establish physiological equivalence; no such equivalence is claimed in this study. The prescribed-flow boundary condition forces the selected flow through the airway even when the lumen narrows, and consequently does not reproduce spontaneous inspiratory flow limitation or airflow cessation during obstructive apnea. Researchers should implement pressure-driven boundary conditions representing respiratory effort and positive airway pressure and validate the predicted responses against patient measurements.

5. Conclusion

In this study, we employed a transient bidirectional fluid-structure interaction framework to systematically quantify, for the first time in a single controlled anatomy, how progressive reduction of the support loads maintaining the tongue and soft palate (0–100% of calibrated reference loads) reshapes upper airway flow, disentangling the mechanical contributions of palatal and lingual collapse across the load spectrum. All findings are specific to this anatomy and modeling configuration. The following conclusions were drawn:

1. Isolated soft palate collapse exerts a minor effect on the flow field and does not produce a marked increase in the ventilation pressure required to maintain the prescribed flow under the modeled conditions.

2. Tongue collapse affects the upper airway only after the tongue contacts the soft palate, generating additional pressure that amplifies soft palate deformation and markedly alters the upper airway flow field.

3. When tongue collapse occurs without soft palate contact, the upper airway flow field is governed by the degree of soft palate support-load reduction. Once tongue–soft palate contact is established, reduction of the tongue support load becomes the dominant determinant of flow field alterations in this model.

4. Reduction of the tongue support load beyond approximately 93% of the calibrated reference value, the level at which appreciable tongue–soft palate compression is established in this model, markedly increases the ventilation pressure required to maintain the prescribed flow (from ~94 Pa to 852 Pa) and the maximum velocity (to 46.3 m/s). This contact transition is model-specific: It reflects the discrete parameter sweep, the calibrated reference load, the single-subject anatomy, and the contact/mesh numerics, and should not be interpreted as a universal neuromuscular threshold. The findings provide a proof-of-concept biomechanical mechanism for tongue-driven obstruction that warrants validation in multi-subject, clinically characterized cohorts with synchronized EMG, dynamic imaging, and polysomnography.

Collectively, these results provide proof-of-concept mechanistic evidence that upper airway obstruction in this model is governed not by the soft palate in isolation but by a nonlinear tongue–palate contact transition; a finding that reframes tongue-base mechanics as the pivotal event in the collapse cascade. While deliberately confined to a single anatomy and artificial loading conditions, the framework establishes a reusable, sensitivity-quantified platform on which multi-subject, EMG-anchored, pressure-driven models can be built, and from which mechanistically grounded, patient-specific predictions, including computationally testable hypotheses about neuromuscular training interventions, can be derived and validated against clinical evidence.

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

Conflict of interest

The authors declare no conflict of interest. The deidentified CT images used in this study were obtained from a commercial database (Medimodel) under an authorization for secondary research use; the use of this database did not influence the study design, analysis, interpretation, or reporting.

Author contributions

Yun-Ying Guo: Conceptualization, Methodology, Investigation, Data curation, Writing – original draft, Writing – review & editing, Project administration. Zhi-Guo Zhang: Software, Formal analysis, Visualization. Mao-Ning Wei: Validation, Resources, Investigation. Wei Xu: Supervision, Conceptualization, Writing – review & editing.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request. For this computational study, the following non-identifiable materials are available: solver configuration files, material parameter definitions, load-case specifications, FSI coupling settings, and analysis scripts. The deidentified anatomical geometry can be shared subject to database licensing restrictions, which are explained on request.

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All images in this study are original works.

Ethics approval and consent to participate

The CT data used were derived from a database established by Medimodel and used for scientific research. The use of deidentified CT images from an existing database constitutes secondary research involving human-derived data. The study was reviewed by the institutional ethics committee, which determined that it was exempt from formal ethics review because it involved only fully deidentified data from a commercial database; accordingly, no approval or exemption certificate number was issued. The CT examination was originally obtained for clinical/diagnostic purposes, and all data were fully deidentified prior to database inclusion. Authorization for secondary research use was obtained from

the database provider. No new human experiments, interventions, or imaging acquisitions were conducted for this study.

Clinical trial registration

No human experiments were conducted in this study.

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