

Title: AI-Driven Lumped-Element Modeling of Human Respiratory System for Studying Voice Mechanics

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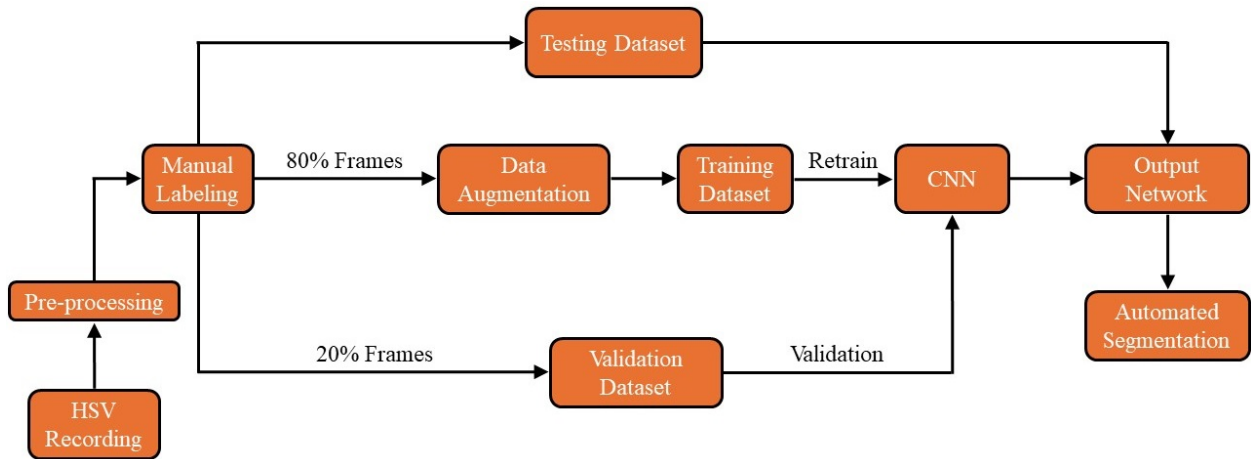
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Supplementary materials

Data Collection and Processing

The development of the present unified lump-element modeling framework begins with the recording of laryngeal high-speed videoendoscopy (HSV) data. HSV data provide the raw imaging data from which the time-resolved glottal area waveform is extracted. This process involves several steps, starting from the image acquisition, preprocessing and image enhancement, deep-learning-based segmentation, and quality control. An overview of the HSV segmentation workflow is shown in Fig. S1. The pipeline illustrates the complete processing sequence from raw HSV recordings through the preprocessing, manual annotation, dataset partitioning, data augmentation, and convolutional neural network (CNN) training, to automated glottal segmentation. These interconnected steps establish the experimental foundation that provides the physiological input to the lumped-element model.



Supplementary Figure S1. Workflow of the convolutional neural network (CNN)-based framework used for automated glottal area segmentation from laryngeal high-speed videoendoscopy (HSV) recordings. HSV recordings are first pre-processed and manually labeled to generate ground-truth masks. The labeled frames are then divided into training, validation, and testing subsets. Eighty percent of the labeled frames are used for data augmentation and training, while the remaining twenty percent are reserved for the validation. The trained network produces an output segmentation model, which is subsequently applied to the independent testing dataset to perform a fully automated glottal segmentation.

Participant Recruitment and Ethics

The present study analyzed HSV data from a 49-year-old male participant with normophonic voice characteristics. The participant was recruited as part of a larger investigation to study vocal fold dynamics during connected speech production. The data collection was conducted at Mayo Clinic-Scottsdale, AZ, under an Institutional Review Board (IRB) protocol approved by Mayo Clinic. The data usage and analysis was approved by the IRB at Michigan State University.

Laryngeal High-Speed Videoendoscopy (HSV) Acquisition

HSV recordings were acquired using a Photron FASTCAM mini AX200 monochrome high-speed camera (Photron Inc., San Diego, CA, USA) coupled with a flexible nasolaryngoscope (Kay Pentax, Lincoln Park, NJ, USA). The camera was configured to capture images at 4,000 frames per second (fps) with a spatial resolution of 256×224 pixels and 12-bit grayscale depth. A 300-W xenon light source provided continuous illumination throughout the recording session to ensure consistent image intensity and minimize motion artifacts. The flexible nasolaryngoscope was inserted transnasally and positioned approximately 1-2 cm above the glottal plane to optimize visualization of the vocal fold edges. Real-time monitoring during insertion ensured an adequate capturing of both vocal folds and surrounding laryngeal structures. The participant was instructed to read standardized speech stimuli from the Consensus Auditory-Perceptual Evaluation of Voice (CAPE-V) protocol [1] and the Rainbow Passage [2]. Recording sessions yielded approximately 200,000-400,000 frames per participant, corresponding to 50-100 seconds of continuous laryngeal imaging during natural speech production.

Image Preprocessing and Enhancement

Raw HSV image sequences were exported in uncompressed 12-bit grayscale format for subsequent analysis. To enhance glottal edge visibility and standardize image quality across frames, a multi-step preprocessing pipeline was applied. First, Gaussian spatial filtering with a kernel size of 3×3 pixels and standard deviation $\sigma = 0.5$ was used to reduce high-frequency noise while preserving edge information. Pixel intensity values were then linearly rescaled to span the full dynamic range $[0, 255]$ using

$$I_{\text{norm}}(x, y) = \frac{I(x, y) - I_{\min}}{I_{\max} - I_{\min}} \times 255 \quad (\text{S1})$$

where $I(x, y)$ represents the original pixel intensity at coordinates (x, y) , and $I_{\min}$ and $I_{\max}$ denote the minimum and maximum intensities within each frame, respectively. Finally, gamma correction with $\gamma = 1.2$ was applied to enhance mid-tone contrast:

$$I_{\text{gamma}}(x, y) = 255 \times \left(\frac{I_{\text{norm}}(x, y)}{255} \right)^{1/\gamma} \quad (\text{S2})$$

Deep Neural Network Architecture for Glottal Segmentation

We employed a U-Net convolutional neural network architecture[3] for automated pixel-wise segmentation of the glottal area in HSV frames. The U-Net consists of a contracting encoder path that captures hierarchical image features and an expansive decoder path that enables precise localization. The network architecture comprised five encoding blocks and five corresponding decoding blocks, with skip connections concatenating feature maps between corresponding encoder and decoder layers to preserve spatial information. Each encoding block contained two convolutional layers with 3×3 kernels, batch normalization, rectified linear unit (ReLU) activation functions, and a 2×2 max pooling operation for downsampling. The number of feature channels doubled after each pooling operation, following the sequence: 64, 128, 256, 512, 1024. Each decoding block performed 2×2 transposed convolution for upsampling, followed by concatenation with corresponding encoder features, and two convolutional layers with ReLU activations. The final layer employed a 1×1

convolution with softmax activation to produce binary segmentation masks distinguishing glottal area pixels (class 1) from background pixels (class 0).

Network Training and Validation

The network was trained on a manually annotated dataset comprising 738 HSV frames selected from six participants (three normophonic controls and three patients with adductor laryngeal dystonia) to capture diverse glottal configurations including abducted, adducted, and obstructed states. Two experienced raters independently delineated glottal boundaries using custom MATLAB software (MathWorks Inc., Natick, MA, USA). Inter-rater reliability was assessed using the Dice similarity coefficient, yielding a mean score of 0.94 ± 0.03 , indicating excellent agreement. The annotated dataset was randomly partitioned into training (80%, $n = 590$ frames) and validation (20%, $n = 148$ frames) subsets. Network training employed the Adam optimizer with an initial learning rate of 1×10^{-4} , batch size of 16, and 100 epochs. The loss function combined binary cross-entropy and Dice loss to address class imbalance:

$$\mathcal{L} = -\frac{1}{N} \sum_{i=1}^N [y_i \log(\hat{y}_i) + (1 - y_i) \log(1 - \hat{y}_i)] + \left(1 - \frac{2 \sum_{i=1}^N y_i \hat{y}_i}{\sum_{i=1}^N y_i + \sum_{i=1}^N \hat{y}_i}\right) \quad (\text{S3})$$

where y_i and $\hat{y}_i$ represent ground truth and predicted labels for pixel i , and N is the total number of pixels. Data augmentation techniques including random rotation ($\pm 15^\circ$), horizontal flipping, and elastic deformations were applied during training to improve generalization. The model achieving the lowest validation loss was selected for inference on test data.

Following completion and validation of the convolutional neural network, the trained model was applied to the HSV recordings from a single 49-year-old male participant with normophonic voice characteristics. The glottal area waveform extracted from this participant was used as the physiological input for the subsequent analyses. Glottal area waveforms derived from the remaining participants were used only for network development and validation and were not included in the downstream modeling or analysis.

Glottal Area Extraction

The trained U-Net model was applied to the entire HSV sequence from the target participant to generate binary segmentation masks for all frames. For each mask, the glottal area $A_g(t)$ at time t was computed as:

$$A_g(t) = \sum_{x=1}^W \sum_{y=1}^H M(x, y, t) S^2 \quad (\text{S4})$$

where $M(x, y, t)$ is the binary mask value at pixel coordinates (x, y) and time t , W and H are the image width and height, and S is the spatial calibration factor (mm/pixel) determined from known anatomical dimensions. The resulting glottal area waveform (GAW) $A_g(t)$ provides a quantitative time-series representation of vocal fold kinematics throughout the speech sample.

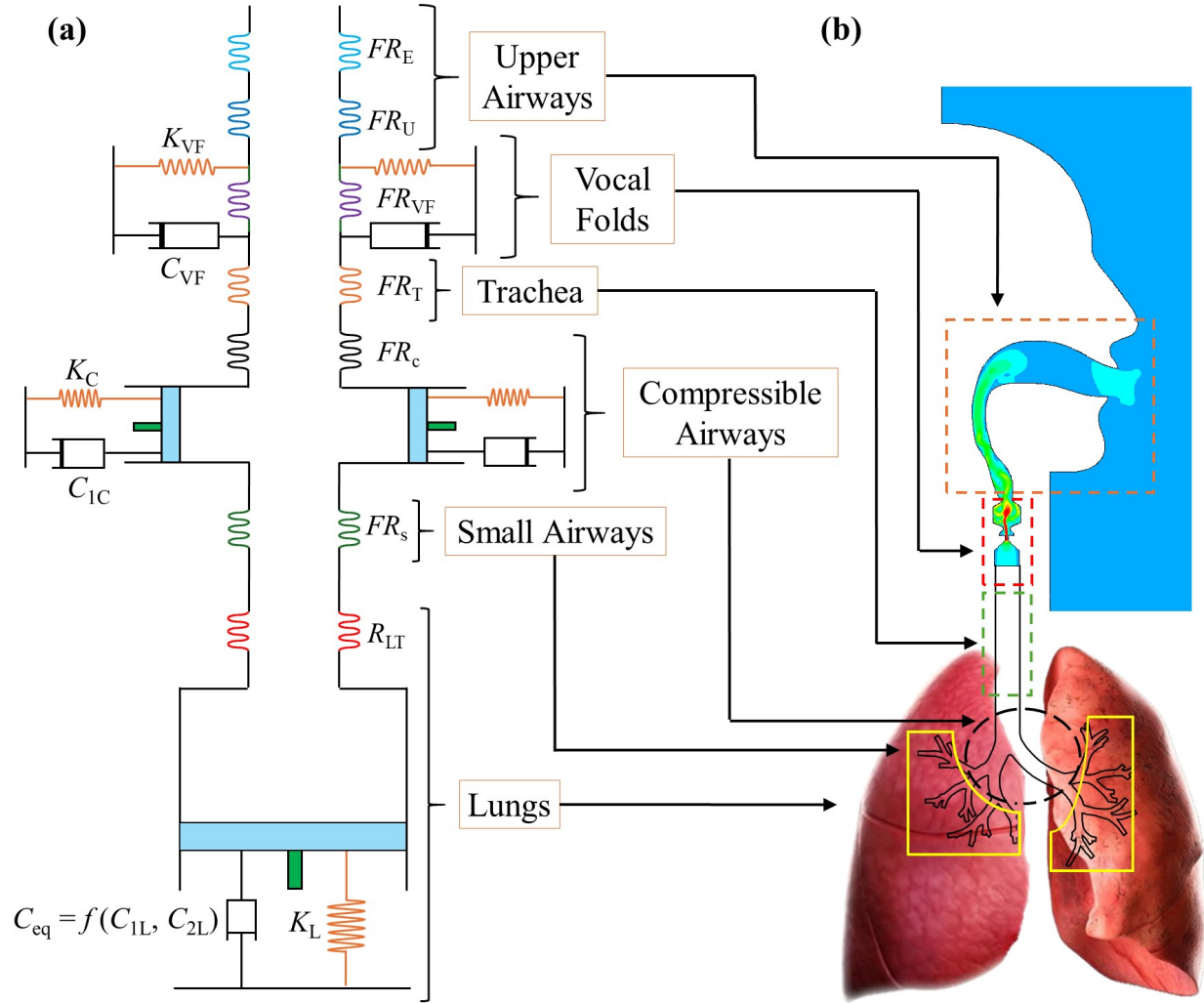
Quality Control and Validation

To ensure segmentation accuracy, two independent raters visually inspected a random sample of 20,000 frames (approximately 10% of the total dataset) representing diverse phonetic contexts and glottal configurations. Raters assessed whether automatically generated masks accurately captured glottal boundaries using a binary accept/reject criterion. Segmentation quality was deemed acceptable when at least 80% of reviewed frames received positive ratings from both raters, consistent with validation metrics reported in prior work[4, 5, 6]. Frames with poor segmentation quality (e.g., due to excessive mucus accumulation or specular reflections) were flagged for manual correction or exclusion from downstream analyses.

Lumped-Element System Overview

Figure S2 illustrates the unified lumped-element architecture developed in this study to describe human phonation as a coupled mechanical–aerodynamic process. The model comprises three dynamically interacting subsystems: the respiratory subsystem, the phonatory subsystem, and the articulatory subsystem. Together, these subsystems form a continuous airflow pathway extending from the lungs to the ambient environment. Each anatomical segment is represented using lumped mechanical elements and flow resistances that capture its dominant physiological function.

The modeling begins with the respiratory subsystem, which consists of the lungs, small airways, compressible airways, and the trachea. In the present formulation, the two lungs are combined into a single equivalent cylindrical chamber whose volumetric expansion and compression are governed by a piston. This unified representation allows the total airflow generated by both lungs to be treated as a single volumetric source, thereby neglecting the phase lag between the left and right lungs. This assumption is justified because inter-lung phase differences, occur on time scales associated with slow respiratory mechanics. In contrast, phonation is governed by much faster aerodynamic and biomechanical processes. Therefore, the influence of the phase lag between the lungs on the overall phonation flow dynamics is assumed to be insignificant. Although the two lungs are combined into a single equivalent chamber in the present formulation, the airflow in the physiological system is inherently distributed between the left and right lungs and each lung responds independently to the applied intrapleural pressure. To preserve this physiological behavior, the present model treats the combined lung chamber as an energetically equivalent system, where the total airflow and pressure response generated by the unified lung representation matches the aggregate behavior of the two lungs acting simultaneously. In this way, the model retains the correct global pressure–flow characteristics of the respiratory subsystem without explicitly resolving left–right lung asymmetries. The motion of this unified lung system is governed by a spring–mass–damper system. The spring stiffness K_L represents the elastic recoil of the lung parenchyma arising from the tissue elasticity. The damping coefficients C_{1L} and C_{2L} represent the viscous dissipation and nonlinear energy losses associated with the tissue viscosity and lung deformation, respectively. The lumped mass M_L accounts for the total mass of the lungs. The external mechanical force imposed upon the lung piston is the intrapleural force, $F_{pl,L}$, which is caused due to the intrapleural pressure, (P_{pl}), signifying the pressure within the intrapleural cavity situated between the lungs and the thoracic wall. As a consequence of the intrapleural force $F_{pl,L}$, the lung piston undergoes axial motion, resulting in volumetric compression of the lung chamber and the expulsion of air from the lungs. As the airflow exits the lung chamber, it first encounters the resistance associated with the deformation of the lung tissue itself. This resistance is represented in the model by the lung tissue resistance R_{LT} ,



Supplementary Figure S2. Unified mechanical-physiological representation of the human phonation system depicting: **(a)** Lumped-element architecture comprising three coupled subsystems of the respiratory subsystem (lungs, small airways, compressible airways, and trachea), the phonatory subsystem (vocal folds), and the articulatory subsystem (upper airways). Airflow is generated by lung contraction and propagates sequentially through the small airways, compressible airways, vocal folds, and upper airways before exiting at the mouth. Along this pathway, the flow encounters lung tissue resistance R_{LT} , small airways flow resistance FR_s , compressible airways resistance FR_c , tracheal flow resistance FR_T , vocal folds flow resistance FR_{VF} , upper airways flow resistance FR_U , and expiratory flow resistance FR_E ; **(b)** Anatomical correspondence of the respiratory, phonatory, and articulatory subsystems, illustrating the mapping between the lumped-element representation and the underlying human airways anatomy.

which accounts for pressure losses arising from the viscoelastic deformation of the parenchymal tissue. Following this initial resistance, the airflow passes into the small airways region, which physiologically corresponds to the bronchioles. These airways are characterized by their relatively small diameters, which cause significant viscous energy dissipation in this section. As the airflow passes this region, velocity gradients near the airway walls give rise to shear stresses that result in additional pressure losses. In the current framework, these pressure losses are incorporated through the small airways flow resistance FR_s .

Above the lung chamber lies the compressible airways, which represent the peripheral airways together with the two bronchi. This segment undergoes changes in luminal cross-sectional area in response to transmural pressure. In the present framework, this compartment is modeled using two identical piston–cylinder assemblies arranged in parallel, reflecting the bilateral structure of the bronchial tree. Each piston is governed by a spring–mass–damper formulation, in which the spring stiffness K_C represents the elastic behavior of the airway walls, the damping coefficient C_{1C} accounts for viscous dissipation within the airway tissue, and the lumped mass M_C represents the inertial contribution of the airway walls. Dynamic variations in airways volume are captured through the axial motion of the pistons. Since these airways are embedded within the intrapleural space, their lateral surfaces are subjected to the same intrapleural pressure P_{pl} that drives the lung motion. The pressure loss associated with the airflow through this region is incorporated into the lumped-element framework through the compressible airways flow resistance FR_c , which accounts for the energy dissipation arising from the airway deformation. Downstream of the compressible airways, airflow passes through the trachea, which is modeled as a rigid conduit. Pressure losses in this segment are incorporated through the tracheal flow resistance FR_T .

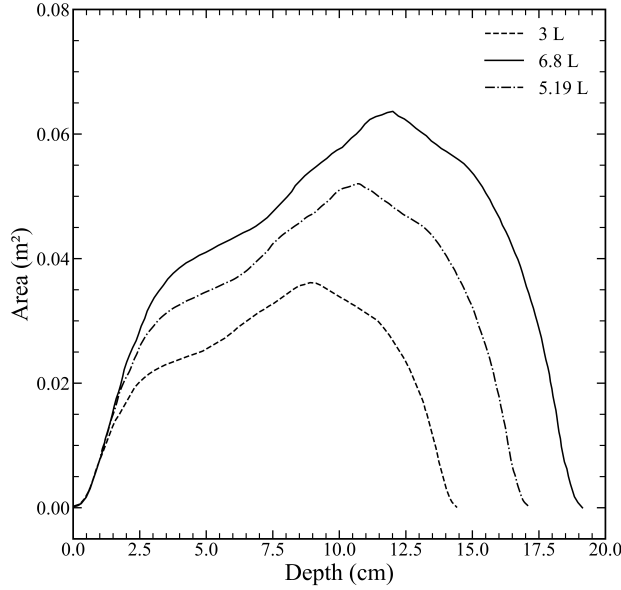
Past the trachea, the airflow enters the glottal region, constituting the phonatory subsystem. In the lumped-element representation, the vocal folds are modeled as a single effective mass–spring–damper system. The spring stiffness K_{VF} represents the elastic properties of the vocal fold tissues, while the damping coefficient C_{VF} accounts for the energy dissipation arising from the tissue viscosity, internal friction, and collision losses during the vocal folds contact. The aerodynamic interaction between the airflow and the oscillating vocal folds generates a time-varying transglottal pressure drop. This pressure loss is incorporated into the model through the vocal fold flow resistance FR_{VF} , which represents the instantaneous aerodynamic impedance of the glottis.

Downstream of the phonatory subsystem, the airflow enters the articulatory subsystem, which corresponds to the upper airways extending from the supraglottal region to the oral cavity. This segment includes the pharyngeal and oral passages that shape the airflow prior to the sound radiation. Aerodynamic losses within the upper airways are represented by the upper airways flow resistance FR_U , which accounts for pressure losses arising from viscous effects, the flow separation, and the geometric complexity of the supraglottal tract. Finally, the airflow is expelled from the oral cavity into the ambient environment. During sustained phonation, this outflow does not occur as a steady, freely diffusing stream. Instead, it must be actively driven through the lips. This behavior becomes evident when placing a hand in front of the mouth and perceiving discrete puffs of air during phonation. Such tactile sensations indicate that the pressure at the lips transiently exceeds atmospheric pressure, resulting in a jet-like expulsion of the airflow during each phonatory cycle. The additional aerodynamic loading associated with the driving airflow through the oral outlet against the atmospheric pressure is represented in the model by the expiratory flow resistance FR_E . Together, the resistive elements form the network that governs the pressure–flow relationship throughout the respiratory–phonatory–articulatory subsystems. The mathematical expressions for these resistances and their implementation are provided in the supplementary materials under the

section compressible airways spring model and compliance.

Lung Geometry and Kinematics

In the present modeling framework, the two lungs are combined into a single equivalent piston-cylinder representation, characterized by an effective cross-sectional area α_L and diameter D_L . The total moving lung mass is taken as $M_L = 1.2$ kg [7], and the total lung capacity (TLC), defined as the maximum volume of air contained in the lungs at full inspiration, is taken to be 5.19 L in the present study. The average lung cross-sectional area α_L was obtained by interpolating between experimentally measured lung area-depth profiles at total lung capacities of 3.0 L and 6.8 L, reported by Mogensen *et al.* [8]. Since direct area-depth measurement for a total lung capacity of 5.19 L was not available, an interpolation procedure was adopted to construct an area-depth profile for this lung volume (see Fig. S3).



Supplementary Figure S3. Lung cross-sectional area A as a function of the physical depth. Experimentally measured area-depth profiles for a total lung capacity of 3.0 L and 6.8 L, reported by Mogensen *et al.* [8], are shown together with the interpolated profile at 5.19 L, produced in the present study.

To ensure a geometric consistency between the two datasets, the axial coordinate was expressed in terms of a normalized depth variable ξ , defined as

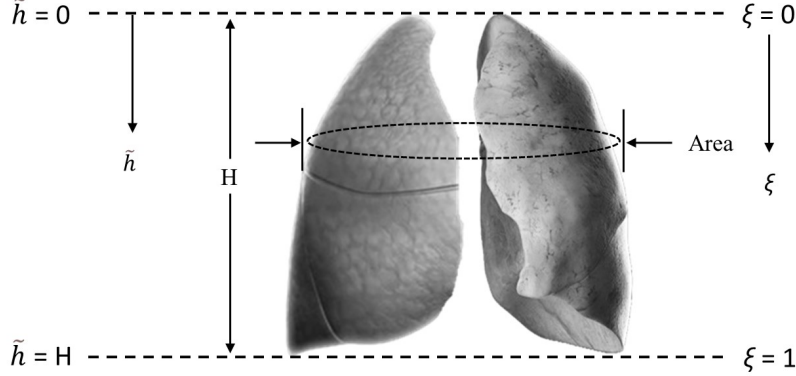
$$\xi = \frac{\tilde{h}}{H}, \quad \xi \in [0, 1], \quad (\text{S5})$$

where $\tilde{h}$ denotes the physical depth measured from the lung apex and H is the total lung height at the corresponding total lung capacity, as illustrated in Fig. S4. Under this normalization, $\xi = 0$ corresponds to the apex and $\xi = 1$ to the lung base. This transformation maps area-depth profiles for both total lung capacities onto a common scale. At each normalized depth ξ , the lung cross-sectional area A corresponding to the intermediate lung volume was obtained by a linear

interpolation in the lung volume,

$$A_{5.19}(\xi) = A_{3.0}(\xi) + \frac{5.19 - 3.0}{6.8 - 3.0} [A_{6.8}(\xi) - A_{3.0}(\xi)], \quad (\text{S6})$$

where $A_{3.0}(\xi)$ and $A_{6.8}(\xi)$ denote the experimentally measured cross-sectional area distributions at total lung capacities of 3.0 L and 6.8 L, respectively, and $A_{5.19}(\xi)$ is the interpolated distribution at a total lung capacity of 5.19 L. This procedure produces a smooth area–depth profile for 5.19 L that lies between the two experimental configurations, as shown in Fig. S3.



Supplementary Figure S4. Schematic illustration of the axial depth coordinate and cross-sectional area definition used for the lung geometry interpolation. The physical axial coordinate $\tilde{h}$ is measured from the lung apex ($\tilde{h} = 0$) to the lung base ($\tilde{h} = H$), where H denotes the total lung height at a given lung volume. The axial coordinate is normalized using the dimensionless variable $\xi = \tilde{h}/H$, such that $\xi = 0$ corresponds to the apex and $\xi = 1$ to the base. At each normalized depth ξ , a transverse lung cross-sectional area is defined, shown by the dashed *Area*.

From the interpolated area distribution, an equivalent constant cross-sectional area α_L was computed by averaging over the lung height according to

$$\alpha_L = \frac{1}{H} \int_0^H A(\tilde{h}) d\tilde{h} = \int_0^1 A(\xi) d\xi \quad (\text{S7})$$

Based on this formulation, the average cross-sectional area was obtained as $\alpha_L = 3.3873 \times 10^{-2} \text{ m}^2$. The corresponding effective lung diameter is therefore

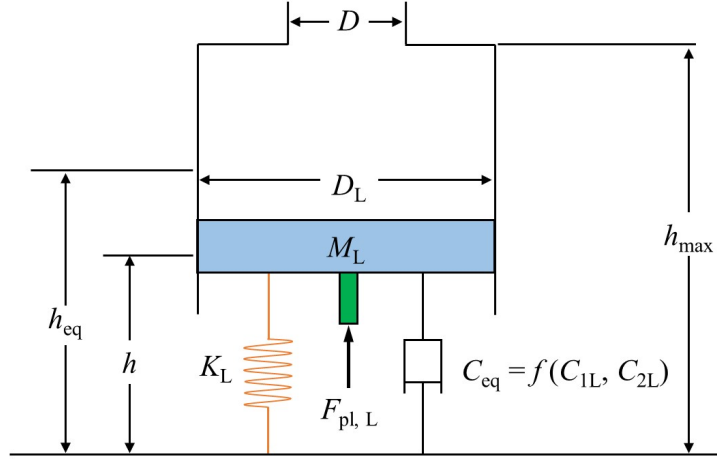
$$D_L = 2\sqrt{\frac{\alpha_L}{\pi}} = 0.2076 \text{ m (20.76 cm)}. \quad (\text{S8})$$

Using this value of α_L , the instantaneous lung volume V_L is expressed as

$$V_L = \frac{\pi D_L^2}{4} (h_{\max} - h) = \alpha_L (h_{\max} - h), \quad (\text{S9})$$

where h denotes the instantaneous axial position of the lung piston measured from the reference, as shown in Fig. S5 and $h_{\max}$ represents the maximum allowable piston travel. For a total lung capacity of 5.19 L and a cross-sectional area $\alpha_L = 3.3873 \times 10^{-2} \text{ m}^2$, the maximum allowable piston travel can be calculated as

$$h_{\max} = \frac{\text{TLC}}{\alpha_L} = 0.153 \text{ m (15.3 cm)}. \quad (\text{S10})$$



Supplementary Figure S5. Lumped-element representation of the human lungs. The left and right lungs are combined into a single equivalent piston-cylinder system with lumped mass M_L . The axial piston position h governs the instantaneous lung volume. The spring stiffness K_L represents the elastic recoil of the lung parenchyma, while the damping elements (C_{1L} , C_{2L}) capture linear and nonlinear dissipative effects. The piston is driven by the intrapleural force $F_{pl,L}$; and the geometric parameters D_L , h_{eq} , and h_{max} denote the effective lung diameter, equilibrium piston position, and maximum allowable piston excursion, respectively.

Lung Spring Model and Compliance

The lung elastic recoil pressure $P_{el,L}$ quantifies the inward-directed mechanical pressure generated by the lung parenchyma at a given lung volume. This pressure arises from the intrinsic elasticity of the lung tissues. Physiologically, lung elastic recoil is governed by the balance between the inward recoil of the parenchymal tissue, driven primarily by elastin and collagen fibers, and the outward recoil imposed by the thoracic cage. Variations in $P_{el,L}$ therefore directly regulate lung volume by modifying the net mechanical load acting on the lung surface. An increase in the elastic recoil pressure enhances the inward-directed force on the lungs, leading to a reduction in the lung volume, whereas a decrease in elastic recoil pressure allows the lungs to expand. The sensitivity of lung volume to changes in the elastic recoil pressure is characterized by the lung compliance, which is defined as

$$C_L = \left| \frac{\Delta V_L}{\Delta P_{el,L}} \right|, \quad (\text{S11})$$

where ΔV_L denotes the change in the lung volume produced by a corresponding change $\Delta P_{el,L}$ in the elastic recoil pressure. A larger value of C_L indicates more distensible lungs that undergo substantial volume changes for small pressure variations, whereas a smaller value corresponds to stiffer lungs requiring higher pressures to achieve the same volume change. In the present lumped-element framework, the lung compliance is mapped onto a nonlinear spring that generates an elastic recoil force $F_{el,L}$, as shown in Fig. S5. This force acts on the lung piston and serves as the mechanical analog of the elastic recoil pressure, with the relationship $P_{el,L} = F_{el,L}/\alpha_L$, where α_L is the effective lung cross-sectional area. In direct analogy with the physiological response of the lung volume to the elastic recoil pressure, the axial displacement of the piston responds to the applied elastic recoil force. For such a spring element, an incremental change in the applied force produces a corresponding incremental displacement. Accordingly, the effective stiffness K_L of the

lung spring is defined through

$$\frac{1}{K_L} = \left| \frac{\Delta h}{\Delta F_{el,L}} \right|, \quad (\text{S12})$$

which represents the differential form of Hooke's law. Here, $\Delta F_{el,L}$ denotes the change in the elastic recoil force acting on the piston, and Δh is the resulting incremental piston displacement. Since the lung volume in the piston-cylinder model is given by $V_L = \alpha_L(h_{\max} - h)$, the differentiation yields $\Delta V_L = -\alpha_L \Delta h$. Combining this relationship with equation (S12) and the definition of lung compliance in equation (S11) leads to

$$C_L = \frac{\alpha_L^2}{K_L}, \quad (\text{S13})$$

which establishes a direct connection between the physiological lung compliance and the stiffness of the mechanical spring used in the model. To describe the nonlinear elastic behavior of lung tissues, the static elastic recoil pressure is modeled following the framework introduced by Le Rolle *et al.* [9]. In this formulation, the elastic recoil pressure is expressed as an exponential function of the lung volume,

$$P_{el,L}(V_L) = A_L e^{\bar{k}_L V_L} - B_L, \quad (\text{S14})$$

where A_L , B_L , and $\bar{k}_L$ are physiological parameters characterizing the intrinsic elastic response of the lung tissues. The values $A_L = 19.61$ Pa, $B_L = 49.03$ Pa, and $\bar{k}_L = 1000.0 \text{ m}^{-3}$ are adopted from [10]. This exponential form reflects a progressive tissue stiffening at higher lung volumes, where collagen fibers increasingly straighten and restrict further expansion. Differentiating equation (S14) with respect to the lung volume and taking the inverse provides the nonlinear lung compliance,

$$C_L(P_{el,L}) = \frac{1}{\bar{k}_L (P_{el,L} + B_L)}, \quad (\text{S15})$$

which explicitly captures the volume-dependent reduction in the compliance at elevated lung pressures. Substituting equation (S15) into equation (S13) yields the corresponding nonlinear stiffness of the lung spring,

$$K_L = \alpha_L^2 \bar{k}_L (P_{el,L} + B_L) = \alpha_L^2 \bar{k}_L A_L e^{\bar{k}_L V_L}, \quad (\text{S16})$$

which governs the elastic response of the lung piston within the proposed unified mechanical framework.

Lung Damping Model and Tissue Dissipation

The dissipative behavior of the lung in the present piston-cylinder representation is modeled through a nonlinear damping law that accounts for the tissue viscosity and the progressive increase in the viscous resistance associated with lung tissues stiffening during deformation. As the lungs expand and recoil, the internal friction within the parenchymal tissues and supporting collagen network converts mechanical energy into heat, giving rise to a velocity-dependent energy dissipation. In its most general form, the damping force acting on the lung piston may be expressed as an infinite series expansion in terms of the piston velocity,

$$F_{\text{damp},L}(\dot{h}) = a_{1L} \dot{h} + a_{2L} \dot{h}^2 + a_{3L} \dot{h}^3 + \dots, \quad (\text{S17})$$

where $\dot{h}$ denotes the piston velocity and the coefficients a_{1L} , a_{2L} , a_{3L} , $\dots$ quantify successive orders of damping. The linear term $a_{1L} \dot{h}$ represents classical viscous losses associated with tissue viscosity,

while the quadratic term $a_{2L}\dot{h}^2$ captures an enhanced viscoelastic resistance arising from the progressive collagen fiber recruitment and strain-dependent stiffening as lung deformation increases. Higher-order terms correspond to additional nonlinear dissipative mechanisms linked to geometric nonlinearity, tissue rearrangement, and the microstructural strain stiffening. Identification of all coefficients in the full expansion of equation (S17) would require detailed in vivo characterization of lung tissues dissipation across a wide range of deformation rates, which is not available in the literature. Thus, we truncate the series and retain only the dominant contributions governing the lung energy dissipation during sustained phonation. This leads to the reduced damping representation of

$$F_{\text{damp,L}}(\dot{h}) \approx a_{1L}\dot{h} + a_{2L}\dot{h}^2, \quad (\text{S18})$$

which preserves the essential linear and nonlinear viscous effects. To ensure that the nonlinear damping contribution always opposes the direction of motion and that the effective damping remains positive for any nonzero velocity, the quadratic term is reformulated in a sign-preserving manner. The final damping expression implemented in the model is therefore given as

$$F_{\text{damp,L}}(\dot{h}) = -C_{1L}\dot{h} - C_{2L}\frac{\dot{h}^3}{|\dot{h}|}, \quad (\text{S19})$$

where C_{1L} and C_{2L} denote the linear and nonlinear viscous damping coefficients, respectively, as shown in Fig. S5. The velocity ratio $\dot{h}^3/|\dot{h}|$ is equivalent to $\dot{h}^2 \text{sign}(\dot{h})$, which ensures that the nonlinear term consistently resists the piston motion and remains positive-definite for all nonzero velocities. The combined effect of the linear and nonlinear contributions may be expressed through a single velocity-dependent effective damping coefficient,

$$C_{\text{eq}} = C_{1L} + C_{2L}|\dot{h}|, \quad (\text{S20})$$

which provides a compact representation of the total dissipative behavior of the lungs.

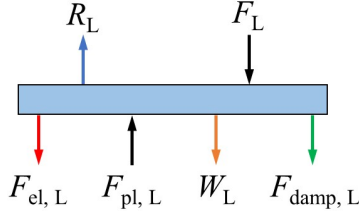
Governing Equation of the Lung Piston-Cylinder Model

As illustrated in Fig. S6, multiple forces act on the lung piston in the piston-cylinder model as it moves within the lung piston-cylinder system. The downward gravitational force is given by the piston weight, $W_L = M_L g$, where M_L is the lumped lung mass and g is the gravitational acceleration. The elastic recoil force generated by the lung tissue is denoted by $F_{\text{el,L}}$ and is modeled as

$$F_{\text{el,L}}(t) = -K_L(t)(h(t) - h_{\text{eq}}), \quad (\text{S21})$$

where h_{eq} denotes the equilibrium piston position. Mathematically, this equilibrium position corresponds to the piston location at which the elastic recoil force is absent. Physiologically, h_{eq} represents the functional residual capacity (FRC) of the lungs, at which the inward elastic recoil of the lung tissues is balanced by the outward elastic recoil of the chest wall. At this resting configuration, no muscular effort is required to maintain the lung volume. The dissipative force arises from the tissue viscosity and is captured by the damping force ($F_{\text{damp,L}}$). In addition, the piston experiences an upward intrapleural force $F_{\text{pl,L}} = \alpha_L P_{\text{pl}}$, where P_{pl} is the intrapleural pressure acting on the lung surface and α_L is the effective lung cross-sectional area. This pressure is generated primarily by diaphragmatic and thoracic motion and serves as the primary driving mechanism for the lung deformation during breathing. Conversely, the internal lung pressure P_L exerts a downward force $F_L = \alpha_L P_L$, which represents the resistance imposed by the compressed air within the

lungs. Finally, the supportive force R_L accounts for the mechanical support provided by the rib cage, diaphragm, and surrounding thoracic structures that guide the axial motion of the piston.



Supplementary Figure S6. Free-body diagram of the lung piston in the lumped piston-cylinder representation. The diagram illustrates all forces acting on the equivalent lung piston along the axial direction. Downward forces include the elastic recoil force of the lung tissues $F_{el,L}$, which resists deformation from the equilibrium position, the gravitational force $W_L = M_L g$ associated with the lumped lung mass, the internal lung pressure force $F_L = \alpha_L P_L$, and the viscous damping force $F_{damp,L}$, which accounts for linear and nonlinear tissue dissipation during the lung motion. Upward forces include the intrapleural force $F_{pl,L} = \alpha_L P_{pl}$, generated by the intrapleural pressure acting on the lung surface, and the supportive force R_L , representing structural support from the rib cage, diaphragm, and surrounding anatomical constraints.

Applying Newton's second law of motion and summing all forces acting on the piston in Fig. S6 yields the governing equation of motion

$$M_L \ddot{h}(t) = F_{el,L}(t) + F_{pl,L}(t) + F_{damp,L}(t) + W_L + R_L(t) + F_L(t). \quad (S22)$$

Substituting the explicit expressions for each force gives

$$\begin{aligned} M_L \ddot{h}(t) = & -K_L(t) (h(t) - h_{eq}) + (P_{pl}(t) - P_L(t)) \frac{\pi D_L^2}{4} \\ & - C_{1L} \dot{h}(t) - C_{2L}(t) \frac{\dot{h}(t)^3}{|\dot{h}(t)|} - M_L g + R_L(t). \end{aligned} \quad (S23)$$

Expressing the dynamics in terms of the lung volume using the kinematic relation $V_L(t) = \alpha_L (h_{max} - h(t))$, the governing equation can be recast directly in the volume form as

$$\begin{aligned} -\frac{M_L}{\alpha_L} \ddot{V}_L(t) = & K_L(t) \left(\frac{V_L(t)}{\alpha_L} + h_{eq} - h_{max} \right) + \alpha_L (P_{pl}(t) - P_L(t)) \\ & - M_L g + \frac{C_{1L}(t)}{\alpha_L} \dot{V}_L(t) + \frac{C_{2L}(t)}{\alpha_L^2} \frac{\dot{V}_L(t)^3}{|\dot{V}_L(t)|} + R_L, \end{aligned} \quad (S24)$$

which constitutes the general governing equation of the lung piston-cylinder system in the present lumped-element framework.

The governing equation derived above is general and can accommodate a range of respiratory and phonatory conditions. In the present study, the model is evaluated under physiological respiratory patterns for which time-resolved reference data are available in the literature. Two representative breathing conditions are considered for our study. They are quiet breathing, corresponding to a relaxed baseline state with low tidal volume and minimal muscular effort, and regular breathing, representing a moderate respiratory demand with larger tidal volume. Marconi and De Lazzari [10]

reported the time-resolved pressure and flow waveforms for both cases. Substituting the corresponding waveforms into our general model gives

$$-\frac{M_L}{\alpha_L} \ddot{V}_L^r(t) = K_L^r(t) \left(\frac{V_L^r(t)}{\alpha_L} + h_{\text{eq}} - h_{\text{max}} \right) + \alpha_L (P_{\text{pl}}^r(t) - P_L^r(t)) - M_L g + \frac{C_{1L}(t)}{\alpha_L} \dot{V}_L^r(t) + \frac{C_{2L}(t)}{\alpha_L^2} \frac{\dot{V}_L^r(t)^3}{|\dot{V}_L^r(t)|} + R_L^r \quad (\text{S25})$$

and

$$-\frac{M_L}{\alpha_L} \ddot{V}_L^q(t) = K_L^q(t) \left(\frac{V_L^q(t)}{\alpha_L} + h_{\text{eq}} - h_{\text{max}} \right) + \alpha_L (P_{\text{pl}}^q(t) - P_L^q(t)) - M_L g + \frac{C_{1L}(t)}{\alpha_L} \dot{V}_L^q(t) + \frac{C_{2L}(t)}{\alpha_L^2} \frac{\dot{V}_L^q(t)^3}{|\dot{V}_L^q(t)|} + R_L^q. \quad (\text{S26})$$

Here, the superscripts q and r denote quiet and regular breathing, respectively. Equations (S25) and (S26) contain five unknowns, C_{1L} , C_{2L} , R_L^r , R_L^q , and h_{eq} , while only two equations are available. The system is therefore under-determined. However, we can determine the equilibrium position h_{eq} analytically. The spring contribution in equations (S25)-(S26) has the form $K_L(t) \left(\frac{V_L(t)}{\alpha_L} + h_{\text{eq}} - h_{\text{max}} \right)$, the parameter h_{eq} represents the equilibrium spring position, and the elastic element should not impose a net bias over a breathing cycle. Hence,

$$\left\langle K_L(t) \left(\frac{V_L(t)}{\alpha_L} + h_{\text{eq}} - h_{\text{max}} \right) \right\rangle = 0 \quad (\text{S27})$$

where $\langle \cdot \rangle$ denotes a time average over one representative cycle. Solving equation (S27) gives

$$h_{\text{eq}} = h_{\text{max}} - \frac{1}{\alpha_L} \frac{\langle K_L(t) V_L(t) \rangle}{\langle K_L(t) \rangle}. \quad (\text{S28})$$

Defining the FRC as

$$\text{FRC} = \alpha_L (h_{\text{max}} - h_{\text{eq}}) \quad (\text{S29})$$

and substituting equation (S28) into equation (S29) provides the FRC as

$$\text{FRC} = \frac{\langle K_L(t) V_L(t) \rangle}{\langle K_L(t) \rangle} \quad (\text{S30})$$

Applying equation (S30) to the quiet- and regular-breathing waveforms gives $\text{FRC}^q = 2.79$ L and $\text{FRC}^r = 2.93$ L. Their mean value is taken as

$$\text{FRC} = \frac{\text{FRC}^q + \text{FRC}^r}{2} = 2.86 \text{ L} \quad (\text{S31})$$

corresponding to 55.13% of the total lung capacity (5.19 L). The relative difference between the two breathing-specific values is

$$\varepsilon_{\text{FRC}} = \frac{|\text{FRC}^r - \text{FRC}^q|}{\frac{1}{2}(\text{FRC}^r + \text{FRC}^q)} \times 100 = 4.68\% \quad (\text{S32})$$

Thus, we will use the average of the both functional residual capacities as a single resting operating point for subsequent simulations. Using $\alpha_L = 3.387300 \times 10^{-2} \text{ m}^2$, $h_{\text{max}} = \frac{V_{\text{TLCL}}}{\alpha_L} = 0.1532 \text{ m}$,

and $\text{FRC} = 2.86 \text{ L}$, equation (S29) gives $h_{\text{eq}} = h_{\text{max}} - \frac{\text{FRC}}{\alpha_L} = 0.06875 \text{ m}$. After calculating h_{eq} , there still remains four unknowns. The damping coefficients C_{1L} and C_{2L} are first estimated using particle swarm optimization, and the optimized values are then substituted into equations (S25) and (S26) to compute the supportive forces R_L^r and R_L^q for regular and quiet breathing. Details of the optimization procedure are provided in the supplementary materials under the section particle swarm optimization algorithm.

Particle Swarm Optimization Algorithm

The particle swarm optimization algorithm provides a population-based stochastic search method. Each particle represents a candidate solution that evolves through the search space by updating its position according to its own historical performance and the performance of neighboring particles. In the present work, particle swarm optimization is employed to identify the lung damping coefficients C_{1L} and C_{2L} , which govern the linear and nonlinear dissipative mechanisms of the respiratory subsystem. The optimization framework consists of two components. The first component imposes physiological and mechanical constraints that restrict the feasible region of the parameter space. These constraints include bounds on dissipative energy expenditure per breath and limits on the supportive forces to ensure a realistic behavior. The second component describes the particle swarm algorithm itself, which explores the constrained parameter space and identifies combinations of C_{1L} and C_{2L} that minimize the objective function while satisfying all imposed physiological requirements.

In the present framework, the lung damping parameters are calculated based on the mechanical energy dissipated over a single breathing cycle. The total dissipative energy associated with the lung dashpot is written as

$$E_d = C_{1L} \int \dot{h}(t)^2 dt + C_{2L} \int |\dot{h}(t)|^3 dt, \quad (\text{S33})$$

where $\dot{h}(t)$ denotes the instantaneous velocity of the lung piston, C_{1L} is the linear viscous damping coefficient, and C_{2L} is the nonlinear damping coefficient. The integrals are evaluated over one complete breathing cycle. In the lumped-element representation of the lung, the piston $V_L(t) = \alpha_L (h_{\text{max}} - h(t))$. Differentiating this relation with respect to time gives

$$\dot{h}(t) = -\frac{\dot{V}_L(t)}{\alpha_L} \quad (\text{S34})$$

Substituting this expression into equation (S33) allows the dissipative energy to be written in terms of the lung volume rate. Thus, the dissipation integrals becomes

$$I_1 = \int_0^{T_{\text{breath}}} \frac{\dot{V}_L(t)^2}{\alpha_L^2} dt \quad (\text{S35})$$

and

$$I_2 = \int_0^{T_{\text{breath}}} \frac{|\dot{V}_L(t)|^3}{\alpha_L^3} dt, \quad (\text{S36})$$

where T_{breath} denotes the breathing period. The dissipative energy per breath can be expressed in a compact linear form as

$$E_d = C_{1L} I_1 + C_{2L} I_2 \quad (\text{S37})$$

For a single breathing condition, the above relation may be written in matrix form as

$$\begin{bmatrix} I_1 & I_2 \end{bmatrix} \begin{bmatrix} C_{1L} \\ C_{2L} \end{bmatrix} = [E_d]. \quad (\text{S38})$$

In the present modeling work, time-resolved lung-volume waveforms $V_L(t)$ are available at a breathing rate of 12 breaths per minute for quiet breathing and regular breathing. From these waveforms, the corresponding dissipation integrals $I_1^{(q)}$, $I_2^{(q)}$, $I_1^{(r)}$, and $I_2^{(r)}$ can be computed directly. If the dissipative energy loss associated with quiet and regular breathing at 12 breaths per minute were known separately, the damping parameters could be obtained by solving

$$\begin{bmatrix} I_1^{(q)} & I_2^{(q)} \\ I_1^{(r)} & I_2^{(r)} \end{bmatrix} \begin{bmatrix} C_{1L} \\ C_{2L} \end{bmatrix} = \begin{bmatrix} E_{\text{diss}}^{(q)} \\ E_{\text{diss}}^{(r)} \end{bmatrix}. \quad (\text{S39})$$

However, the experimental study of Athanasiades *et al.* [11] reports the dissipative energy loss per breathing cycle without distinguishing between quiet and regular breathing. At 12 breaths per minute, the reported dissipative energy per breath is

$$E_{\text{diss}}^{12} = 7.5833 \times 10^{-2} \text{ J/cycle}. \quad (\text{S40})$$

Because the breathing mode associated with this value is not specified, the above system cannot be applied directly. To resolve this ambiguity, additional experimental information is used. The same study reports the dissipative energy loss at a lower breathing rate of 8 breaths per minute, given by

$$E_{\text{diss}}^8 = 9.25 \times 10^{-2} \text{ J/cycle}. \quad (\text{S41})$$

Although time-resolved lung-volume data are not available at 8 breaths per minute, the availability of waveform data at 12 breaths per minute enables an interpolation-based strategy to estimate the corresponding dynamics at 8 breaths per minute. For this, we will denote $V_L^{12}(t)$ as the lung-volume waveform at 12 breaths per minute, defined over one breathing period $T_{12} = 60/12$. For a target breathing rate f , the breathing period is $T(f) = 60/f$. We define a time-scaling factor

$$a = \frac{T(f)}{T_{12}} = \frac{12}{f}, \quad (\text{S42})$$

and a volume-scaling factor

$$s = \frac{V_T(f)}{V_T(12)}, \quad (\text{S43})$$

where $V_T(f)$ denotes the tidal volume at breathing rate f . The introduction of the time-scaling factor a adjusts the duration of the breathing cycle so that the waveform spans the correct period corresponding to the new breathing rate and the volume-scaling factor s ensures that the interpolated lung-volume waveform at the target breathing rate preserves the correct tidal volume. In general, the tidal volume is not constant across breathing rates. Experimental measurements show that slower breathing is accompanied by a larger tidal volume, whereas faster breathing is associated with a smaller tidal volume. Thus, to construct an approximate lung-volume waveform at a new breathing rate, we enforce the correct total volume change per breath in addition to the correct period. Using these scaling factors, the lung-volume waveform at rate f is approximated as

$$V_L^f(t') = s V_L^{12}\left(\frac{t'}{a}\right) \quad (\text{S44})$$

with $t' = at$. The differentiation of the above equation gives the corresponding lung-volume rate

$$\dot{V}_L^f(t') = \frac{s}{a} \dot{V}_L^{12} \left(\frac{t'}{a} \right). \quad (\text{S45})$$

Now, using this scaled waveform, the dissipation integrals at breathing rate f satisfy the scaling relations

$$I_1^{(f)} = \frac{s^2}{a} I_1^{(12)} \quad (\text{S46})$$

and

$$I_2^{(f)} = \frac{s^3}{a^2} I_2^{(12)}. \quad (\text{S47})$$

Applying these relations for $f = 8$ breaths per minute allows the dissipative energy expressions at 12 and 8 breaths per minute to be written as

$$E_{\text{diss}}^{12} = C_{1L} I_1^{12} + C_{2L} I_2^{12} \quad (\text{S48})$$

$$E_{\text{diss}}^8 = C_{1L} I_1^8 + C_{2L} I_2^8 \quad (\text{S49})$$

or equivalently in a matrix form as

$$\begin{bmatrix} I_1^{12} & I_2^{12} \\ I_1^8 & I_2^8 \end{bmatrix} \begin{bmatrix} C_{1L} \\ C_{2L} \end{bmatrix} = \begin{bmatrix} E_{\text{diss}}^{12} \\ E_{\text{diss}}^8 \end{bmatrix}. \quad (\text{S50})$$

Even with this interpolation strategy, it remains unclear which specific breathing mode (quiet or regular) the experimentally reported dissipative energy values correspond to. Thus, rather than forcing a single mode assumption, we identify a physically plausible range for (C_{1L}, C_{2L}) . Now, for each mode $m \in \{q, r\}$ and rate $f \in \{8, 12\}$, the dissipated energy per breath can be written as

$$E_{\text{dis}}^{(m,f)} = C_{1L} I_1^{(m,f)} + C_{2L} I_2^{(m,f)}, \quad (\text{S51})$$

where the dissipation integrals are computed from the mode- and rate-specific volume waveform as follows

$$I_1^{(m,f)} = \int_0^{T(f)} \frac{\dot{V}_L^{(m,f)}(t)^2}{\alpha_L^2} dt \quad (\text{S52})$$

$$I_2^{(m,f)} = \int_0^{T(f)} \frac{|\dot{V}_L^{(m,f)}(t)|^3}{\alpha_L^3} dt \quad (\text{S53})$$

and $T(f) = 60/f$.

Now, we enforce that the waveforms for quiet breathing at 12 breaths per minute and the interpolated quiet waveform at 8 breaths per minute reproduce the two rate-specific dissipative energy losses reported by Athanasiades *et al.* [11]. Hence,

$$\begin{bmatrix} I_1^{(q,12)} & I_2^{(q,12)} \\ I_1^{(q,8)} & I_2^{(q,8)} \end{bmatrix} \begin{bmatrix} C_{1L} \\ C_{2L} \end{bmatrix} = \begin{bmatrix} E_{\text{dis}}^{(12)} \\ E_{\text{dis}}^{(8)} \end{bmatrix}. \quad (\text{S54})$$

Solving equation (S54) gives a quiet-conditioned estimate of

$$\begin{bmatrix} \hat{C}_{1L}^{(q)} \\ \hat{C}_{2L}^{(q)} \end{bmatrix} = \begin{bmatrix} I_1^{(q,12)} & I_2^{(q,12)} \\ I_1^{(q,8)} & I_2^{(q,8)} \end{bmatrix}^{-1} \begin{bmatrix} E_{\text{dis}}^{(12)} \\ E_{\text{dis}}^{(8)} \end{bmatrix} \quad (\text{S55})$$

Similarly, for the regular breathing we apply the same strategy which gives

$$\begin{bmatrix} I_1^{(r,12)} & I_2^{(r,12)} \\ I_1^{(r,8)} & I_2^{(r,8)} \end{bmatrix} \begin{bmatrix} C_{1L} \\ C_{2L} \end{bmatrix} = \begin{bmatrix} E_{\text{dis}}^{(12)} \\ E_{\text{dis}}^{(8)} \end{bmatrix}, \quad (\text{S56})$$

which gives the regular-conditioned estimate

$$\begin{bmatrix} \hat{C}_{1L}^{(r)} \\ \hat{C}_{2L}^{(r)} \end{bmatrix} = \begin{bmatrix} I_1^{(r,12)} & I_2^{(r,12)} \\ I_1^{(r,8)} & I_2^{(r,8)} \end{bmatrix}^{-1} \begin{bmatrix} E_{\text{dis}}^{(12)} \\ E_{\text{dis}}^{(8)} \end{bmatrix}. \quad (\text{S57})$$

These two solutions in equations (S55)–(S57) are both compatible with the same rate-specific dissipation targets but arise from different waveform kinematics. Therefore, rather than selecting one solution and discarding the other, we interpret them as bracketing a plausible parameter region for C_{1L} and C_{2L} as follows

$$C_{1,\min} = \min(\hat{C}_{1L}^{(q)}, \hat{C}_{1L}^{(r)}) \quad C_{1,\max} = \max(\hat{C}_{1L}^{(q)}, \hat{C}_{1L}^{(r)}) \quad (\text{S58})$$

$$C_{2,\min} = \min(\hat{C}_{2L}^{(q)}, \hat{C}_{2L}^{(r)}) \quad C_{2,\max} = \max(\hat{C}_{2L}^{(q)}, \hat{C}_{2L}^{(r)}) \quad (\text{S59})$$

Although equations (S58) and (S59) are directly derived from experimental measurements, several sources of uncertainty are there that suggests a need for a controlled widening of the current parameter range. These include uncertainty in the reported dissipation values, the approximate nature of the waveform interpolation used to construct the 8 breaths per minute dynamics, and the possibility that different parameter combinations may result in nearly identical dissipative energies. Thus, to tackle these uncertainties, we expand the conservative intervals by $\pm 25\%$ and define the final search bounds

$$C_{1L,\min} = 0.75 C_{1,\min} \quad C_{1L,\max} = 1.25 C_{1,\max} \quad (\text{S60})$$

$$C_{2L,\min} = 0.75 C_{2,\min} \quad C_{2L,\max} = 1.25 C_{2,\max} \quad (\text{S61})$$

With the search region defined, the next step is to construct an objective function that enforces consistency across breathing rates and breathing modes. For any candidate parameter pair (C_{1L}, C_{2L}) , the predicted dissipated energy per breath in mode $m \in \{q, r\}$ at rate $f \in \{8, 12\}$ is computed from equation (S51) as

$$E_f^{(m)}(C_{1L}, C_{2L}) = C_{1L} I_1^{(m,f)} + C_{2L} I_2^{(m,f)}. \quad (\text{S62})$$

Then we define the experimental dissipation ratio

$$R_{\text{data}} = \frac{E_{\text{dis}}^{(12)}}{E_{\text{dis}}^{(8)}} \quad (\text{S63})$$

and the corresponding predicted ratios for each breathing mode

$$R_{\text{pred}}^{(m)}(C_{1L}, C_{2L}) = \frac{E_{12}^{(m)}(C_{1L}, C_{2L})}{E_8^{(m)}(C_{1L}, C_{2L})} \quad (\text{S64})$$

The assumption we used in this work is that the net energy loss of the respiratory subsystem should be comparable between the quiet and regular breathing at a given rate. Since the experimental

data do not label mode, we enforce that both breathing modes reproduce the observed cross-rate scaling. We therefore define the objective function of

$$J(C_{1L}, C_{2L}) = (R_{\text{pred}}^{(q)} - R_{\text{data}})^2 + (R_{\text{pred}}^{(r)} - R_{\text{data}})^2 \quad (\text{S65})$$

The ratio-based cost function in equation (S65) is adopted because it captures the frequency dependence of the dissipative energy. Moreover, by operating on ratios rather than absolute energy values, the formulation also reduces the sensitivity to uncertainty in the reported magnitudes of $E_{\text{dis}}^{(12)}$ and $E_{\text{dis}}^{(8)}$, since any uniform bias affecting both measurements partially cancels. In addition, this approach enforces a shared parameterization across quiet and regular breathing conditions, requiring a single pair of damping coefficients to reproduce the same cross-rate scaling for both quiet and regular breathing and thereby preventing overfitting to a specific breathing condition. Thus, in the optimization procedure, candidate pairs (C_{1L}, C_{2L}) are sampled within the expanded bounds given by equations (S60) and (S61), to minimize the objective function in equation (S65).

The next part of the optimization framework involves the application of particle swarm optimization algorithm. This approach will provide a population based stochastic search strategy for identifying the parameters that best reproduce the supportive forces during quiet and regular breathing. In this framework, each particle represents a candidate parameter vector, denoted by $\mathbf{x}_i^{(t)} \in \mathbb{R}^2$, where the dimensionality of the parameter space corresponds to the two optimization variables C_{1L} and C_{2L} , and j is the iteration index. The velocity of the particle $\mathbf{v}_i^{(j)}$ determines the direction and magnitude of its movement through the search domain. The evolution of each particle is influenced by its own historically best position ($\mathbf{pbest}_i$) and by the best position discovered by the swarm ($\mathbf{gbest}$). The velocity update rule combines inertia with stochastic cognitive and social components and is written as

$$v_{i,d}^{(j+1)} = \chi \left[w v_{i,d}^{(j)} + c_1 r_1 (pbest_{i,d} - x_{i,d}^{(j)}) + c_2 r_2 (gbest_d - x_{i,d}^{(j)}) \right] \quad (\text{S66})$$

where $\chi = 0.729$ is the constriction factor that ensures a stable convergence, $c_1 = c_2 = 2.05$ are the cognitive and social coefficients, and r_1 and r_2 are independent stochastic variables drawn from a uniform distribution between zero and one. The particle position is updated according to

$$x_{i,d}^{(j+1)} = x_{i,d}^{(j)} + v_{i,d}^{(j+1)}. \quad (\text{S67})$$

To ensure an effective balance between the global exploration during the early stages of the search and the local refinement as the algorithm approaches convergence, the inertia weight w decreases linearly with time according to

$$w = 0.9 - j \left(\frac{0.5}{T_{\text{max}}} \right), \quad (\text{S68})$$

where T_{max} is the maximum number of iterations. This schedule is known to enhance convergence properties by gradually reducing the influence of previous velocities. In the present study, the particle swarm consists of 4000 particles and is executed for 5000 iterations to minimize the objective function. At each iteration, every particle compares its current objective value to its historical best value and updates $\mathbf{pbest}_i$ if improvement is observed. The global best $\mathbf{gbest}$ is also updated whenever a particle discovers a superior solution. After the convergence, the optimal parameter vector is obtained as

$$\mathbf{x}^* = [C_{1L}^*, C_{2L}^*], \quad (\text{S69})$$

which represents the set of values that most accurately reproduces the supportive forces associated with quiet and regular breathing. To assess the robustness and reduce the influence of stochastic

variability inherent to swarm-based optimizations, the particle swarm procedure is repeated across multiple independent runs using different random seeds. The final calibrated values of (C_{1L}, C_{2L}) are obtained by averaging the optimal solutions across all trials, and the associated variability is quantified through the corresponding standard deviations. The resulting coefficients are $C_{1L} = 195.79 \pm 16.05\% \text{ kg s}^{-1}$ and $C_{2L} = 1291.01 \pm 16.05\% \text{ kg m}^{-2} \text{ s}^{-1}$.

With the identification of the lung damping coefficients C_{1L} and C_{2L} , the mechanical energy dissipated by the lung dashpot during a breathing cycle can now be evaluated directly from the model. For a given breathing mode $m \in \{q, r\}$ at 12 breaths per minute, the lung-related dissipative energy is computed as

$$E_L^{(m,12)} = C_{1L} I_1^{(m,12)} + C_{2L} I_2^{(m,12)}, \quad (\text{S70})$$

where $I_1^{(m,12)}$ and $I_2^{(m,12)}$ are the dissipation integrals obtained from the corresponding lung-volume waveform. This modeled lung dissipation represents only the contribution associated with tissue-level damping captured by C_{1L} and C_{2L} and is therefore not expected to exactly match the experimentally reported total dissipative energy $E_{\text{dis}}^{(12)}$, which reflects dissipative losses from both the lungs and the compressible airways. Thus, the remaining discrepancy between the experimentally reported dissipation and the lung dissipation is attributed to additional viscous losses occurring within the compressible airways. Accordingly, the damping energy dissipated by the compressible airways over one breathing cycle is defined as

$$E_{cd}^{(m,12)} = E_{\text{dis}}^{(12)} - (C_{1L} I_1^{(m,12)} + C_{2L} I_2^{(m,12)}). \quad (\text{S71})$$

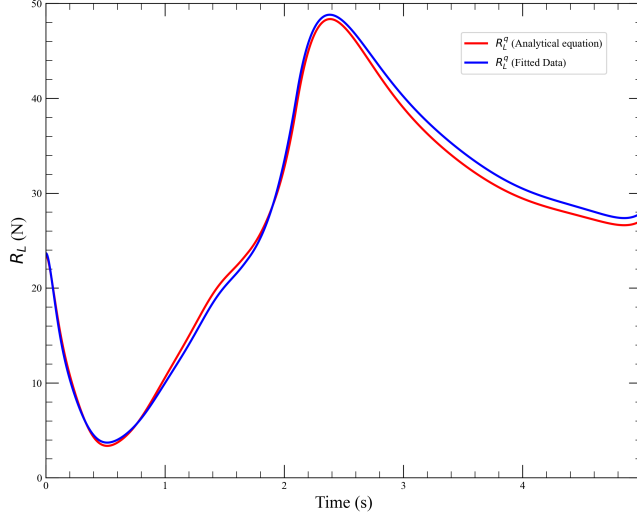
Applying this expression to the identified damping parameters gives an average mechanical energy dissipation by the compressible airways of approximately 0.015 J/cycle for regular breathing and 0.02 J/cycle for quiet breathing at 12 breaths per minute.

Modeling of Lung Supportive Forces

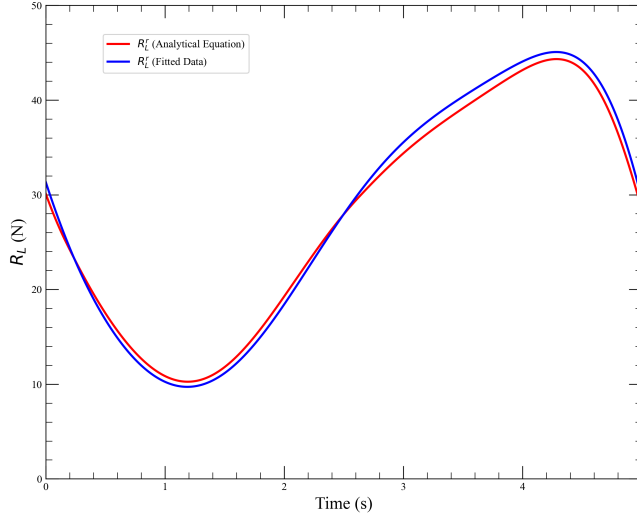
The particle swarm optimization algorithm provides optimal estimates of the lung damping coefficients C_{1L} and C_{2L} . Substituting these optimized parameters into equations S25 and S26 allows the lung supportive forces associated with quiet breathing, denoted by R_L^q , and regular breathing, denoted by R_L^r , to be computed directly from the force balance equations. The resulting supportive forces, shown in Figs. S7 and S8, reflect the mechanical response of the thoracic support system under the corresponding breathing conditions. A direct use of these expressions, however, provides supportive forces that are explicitly tied to the breathing pattern from which they are derived. Such dependence limits their applicability to phonatory conditions, where pleural pressure dynamics, airflow rates, and temporal characteristics differ substantially from those observed during quiet or regular breathing. To establish a formulation that remains valid across a broader range of respiratory tasks, including sustained phonation and nonperiodic breathing patterns, the supportive force must be expressed in a form that depends only on the instantaneous mechanical state of the lung rather than on the global breathing mode.

In the current framework, the mechanical state of the lung is characterized by $(V_L(t), \dot{V}_L(t), \ddot{V}_L(t))$, which captures the instantaneous lung volume, volumetric flow rate, and inertial loading, respectively. Thus, the lung supportive force is expressed in the general functional form

$$R_L(t) = f(V_L(t), \dot{V}_L(t), \ddot{V}_L(t)), \quad (\text{S72})$$



Supplementary Figure S7. Comparison between the lung supportive force obtained directly from the governing force balance equations and the supportive force reconstructed using the unified state-based formulation. The red curve represents the supportive force $R_L^q(t)$ computed analytically from the full lung piston-cylinder model under quiet breathing conditions, while the blue curve shows the corresponding supportive force reconstructed from the fitted linear relation $R_L(t) = \bar{A}_R \tilde{V}_L(t) + \bar{B}_R$, where $\tilde{V}_L(t) = V_L(t) + \beta_L \dot{V}_L(t)$. The close agreement between the two trajectories over the entire breathing cycle demonstrates that the averaged coefficients accurately capture the underlying mechanical behavior of the lung supportive force despite the simplification.



Supplementary Figure S8. Comparison between the lung supportive force computed from the full governing equations and the supportive force reconstructed using the unified state-based formulation under regular breathing conditions. The red curve denotes the analytically obtained supportive force $R_L^r(t)$ evaluated directly from the lung piston-cylinder force balance, while the blue curve represents the supportive force reconstructed from the fitted linear relation $R_L(t) = \bar{A}_R \tilde{V}_L(t) + \bar{B}_R$, where the effective lung volume is defined as $\tilde{V}_L(t) = V_L(t) + \beta_L \dot{V}_L(t)$. The close overlap of the two curves throughout the breathing cycle demonstrates that the averaged state-based formulation accurately captures the dominant mechanical features of the lung supportive force during regular breathing, including both the inspiratory loading phase and the expiratory unloading phase.

which implies that two respiratory conditions producing identical instantaneous lung volume, flow, and acceleration must generate identical supportive forces. To construct a unified representation of the supportive force, an effective lung volume $\tilde{V}_L(t)$ is introduced and defined as

$$\tilde{V}_L(t) = V_L(t) + \beta_L \dot{V}_L(t) + \gamma_L \ddot{V}_L(t), \quad (\text{S73})$$

where β_L and γ_L are scalar coefficients that quantify the relative contributions of flow-dependent and inertial effects to the effective mechanical state of the lung. In principle, a more general formulation could include higher-order temporal derivatives of lung volume to explicitly represent additional dynamic effects. However, identifying such contributions would require reliable measurements across multiple distinct breathing conditions. Since only quiet and regular breathing data are available in the present study, the representation is truncated at the second temporal derivative. The relative importance of the included terms is further assessed through the ratio $|\gamma_L/\beta_L|$, which provides insight into the hierarchical influence of inertial versus flow-related effects in shaping the lung supportive force.

Following this formulation, a consistent description of the lung supportive force across different breathing conditions is obtained when the trajectories of R_L^q and R_L^r intersect at identical values of the effective lung volume $\tilde{V}_L$. Each intersection corresponds to an instant at which quiet and regular breathing produce the same supportive force, despite arising from distinct respiratory dynamics. Intersection points between the supportive force curves were identified using cubic spline interpolation. At each intersection time t_i , the corresponding values of lung volume $V_L(t_i)$, volumetric flow rate $\dot{V}_L(t_i)$, and volumetric acceleration $\ddot{V}_L(t_i)$ were extracted for both breathing conditions. Using two such intersection instances, denoted by $(t_{\text{in1}}, t_{\text{in2}})$, a system of equations was formulated to determine the coefficients β_L and γ_L :

$$\begin{aligned} \beta_L \left(\dot{V}_L^q(t_{\text{in1}}) - \dot{V}_L^r(t_{\text{in1}}) \right) + \gamma_L \left(\ddot{V}_L^q(t_{\text{in1}}) - \ddot{V}_L^r(t_{\text{in1}}) \right) &= V_L^r(t_{\text{in1}}) - V_L^q(t_{\text{in1}}) \\ \beta_L \left(\dot{V}_L^q(t_{\text{in2}}) - \dot{V}_L^r(t_{\text{in2}}) \right) + \gamma_L \left(\ddot{V}_L^q(t_{\text{in2}}) - \ddot{V}_L^r(t_{\text{in2}}) \right) &= V_L^r(t_{\text{in2}}) - V_L^q(t_{\text{in2}}) \end{aligned} \quad (\text{S74})$$

Solving this system gives $\beta_L = -5.919 \text{ s}$ and $\gamma_L = 0.0164 \text{ s}^2$. These values indicate that the volumetric flow rate contributes strongly and negatively to the effective state variable, while the contribution from the volumetric acceleration is positive but comparatively small. The ratio $|\gamma_L/\beta_L| \approx 2.77 \times 10^{-3}$ demonstrates that the influence of the second temporal derivative is several orders of magnitude weaker than that of the first derivative. This pronounced disparity confirms that higher-order temporal effects contribute negligibly to the supportive force within the physiological range of breathing dynamics. Consequently, the acceleration term is omitted from our reduced order reconstruction without meaningful loss of accuracy. This allowed the supportive force to be described accurately using only the lung volume and its first temporal derivative. Under this simplification, the effective lung volume reduces to $\tilde{V}_L(t) = V_L(t) + \beta_L \dot{V}_L(t)$. Now, a unified representation of the lung supportive force is obtained by expressing $R_L(t)$ as a linear function of $\tilde{V}_L(t)$:

$$R_L(t) = A_R \tilde{V}_L(t) + B_R \quad (\text{S75})$$

The coefficients A_R and B_R are computed from the supportive forces associated with quiet and regular breathing as

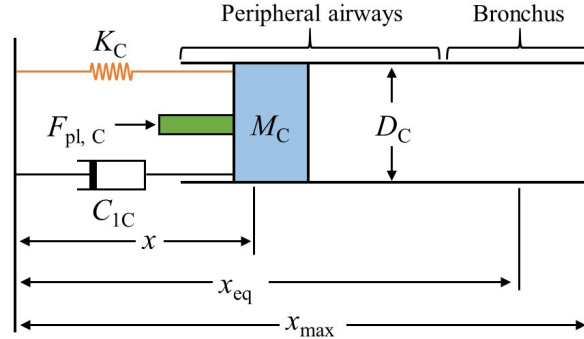
$$A_R = \frac{R_L^r - R_L^q}{\tilde{V}_L^r - \tilde{V}_L^q} \quad (\text{S76})$$

$$B_R = R_L^q - A_R \tilde{V}_L^q \quad (\text{S77})$$

The coefficients A_R and B_R vary in time because the relationship between the supportive force and effective lung volume evolves throughout the breathing cycle. To further simplify the model, average values of these coefficients, denoted by $\bar{A}_R$ and $\bar{B}_R$, were computed. The validity of this approximation was assessed by reconstructing the supportive force using $R_L(t) = \bar{A}_R \tilde{V}_L(t) + \bar{B}_R$ and comparing the results with the supportive forces obtained directly from quiet and regular breathing. As shown in Figs. S7 and S8, this averaged representation achieves a fit accuracy of approximately 94.2% for quiet breathing and 95.2% for regular breathing. These results demonstrate that despite the simplification, the averaged coefficients reproduce the supportive force trajectories with high fidelity.

Compressible Airways Geometry and Effective Mass

The compressible airways represent a pressure-sensitive, variable-volume segment whose geometry changes in response to the transmural pressure [12]. The complete geometric configuration is illustrated in Fig. S2, where each cylindrical assembly consists of an upper segment representing a bronchus and a lower segment representing one-half of the peripheral airways region. Each piston-cylinder unit is idealized as having a constant diameter D_C , as shown in Fig. S9. To maintain geometric and mechanical continuity with the proximal airways, this diameter is taken to be equal to the exit diameter of the lung piston-cylinder system, ($D_C = 17$ mm). This choice is consistent with established anatomical averages for the right and left bronchi and avoids introducing nonphysiological flow features associated with abrupt expansions or contractions at the airway junctions. The corresponding cross-sectional area is therefore $\alpha_C = \pi D_C^2/4 \approx 2.27 \times 10^{-4} \text{ m}^2$.



Supplementary Figure S9. Lumped-element representation of the compressible airways. The piston of effective mass M_C represents the deformable airway walls. The spring element with stiffness K_C models the elastic compliance of the airway tissues, while the dashpot with damping coefficient C_{1C} accounts for viscous energy dissipation associated with airway wall motion. The piston is driven by the intrapleural force $F_{pl,C}$, which induces volume changes. The instantaneous piston displacement is denoted by x , with x_{eq} indicating the equilibrium position and x_{max} the maximum allowable excursion corresponding to the fully expanded compressible airways volume. The effective airway diameter is denoted by D_C . The upper region corresponds to the bronchus, while the lower region represents the peripheral airways lacking cartilaginous support.

The total compressible airways volume consists of contributions from both the two bronchi and

the peripheral airways. A representative bronchial length of $L_B = 3$ cm is adopted for each bronchus [13], giving a combined bronchial volume of $V_B = 2\alpha_C L_B \approx 13.65$ mL. The remaining contribution arises from the peripheral airways, whose volume has been reported to vary between 0.015 L and 0.185 L [10]. As a result, the total compressible airways volume spans from 28.65 mL to 198.65 mL. Using the upper bound of this range, the corresponding maximum piston excursion is $x_{\max} = \frac{V_{C,\max}}{2\alpha_C} = 43.76$ cm. This effective maximum travel length represents the volume-equivalent axial displacement of the combined bronchi and peripheral airways structures within the lumped compressible airways model. During dynamic simulations, the instantaneous compressible airways volume is expressed as

$$V_C(t) = 2\alpha_C(x_{\max} - x(t)), \quad (\text{S78})$$

where $x(t)$ denotes the time-dependent piston displacement. To ensure consistency with lung mechanics at functional residual capacity, the equilibrium position of the compressible airways piston is defined such that the airway volume occupies the same fraction of its maximum volume as the lung volume occupies relative to the total lung capacity. This condition gives $x_{\text{eq}} = x_{\max} \left(\frac{h_{\text{eq}}}{h_{\max}} \right)$. The inertial response of the compressible airways is represented through an effective mass M_C , corresponding to the mass of the airway wall tissues associated with the two bronchi and the peripheral airways. Direct measurements of the mass of the peripheral airways tissue are not available. Therefore, a representative tissue density ρ_C is estimated by averaging reported densities of the primary structural constituents of the airways walls. Hyaline cartilage exhibits densities between 1.1 and 1.2 g mL⁻¹ [14], connective tissue typically ranges from 1.0 to 1.04 g mL⁻¹ [15], and the smooth muscle has a reported density of approximately 1.06 g mL⁻¹ [16]. Assuming comparable volumetric contributions from these tissue types, their mean value is adopted, giving $\rho_C \approx 1.10$ g mL⁻¹ = 1100 kg m⁻³. The total airway wall tissue volume is approximated as $V_{w,C} = 2\pi x_{\max} [(r_C + t_C)^2 - r_C^2]$, where the inner radius $r_C = 0.0085$ m and wall thickness $t_C = 0.000799$ m are taken from anatomical measurements reported by Bankier *et al.* [17]. Using these values gives an effective compressible airways tissue mass of $M_C = \rho_C V_{w,C} \approx 2.95 \times 10^{-3}$ kg.

Compressible Airways Spring Model and Compliance

The elastic behavior of the compressible airways is represented in the lumped-element framework by an effective spring with stiffness K_C . Consistent with the mechanical analogy adopted for the lung piston-cylinder system, the spring stiffness is related to the airway compliance through

$$K_C = \frac{2\alpha_C^2}{C_C}, \quad (\text{S79})$$

where α_C is the cross-sectional area of a single airway piston and C_C denotes the compliance of the compressible airways compartment. The factor of 2 accounts for the presence of two identical piston-cylinder units arranged in parallel to represent the combined response of the bilateral compressible airways system. The deformation of the compressible airways is governed by the transmural pressure P_{tm} , defined as the pressure difference between the airway lumen and the surrounding intrapleural space [10]. The static pressure-volume relationship of the compressible airways is described by the sigmoidal function

$$V_C(P_{tm}) = \frac{V_{C,\max}}{1 + \exp\left(-\frac{P_{tm}-A}{B}\right)}, \quad (\text{S80})$$

where $V_{C,\max} = 198.65$ mL denotes the maximum attainable compressible airways volume. The parameter $A = 431.493$ Pa specifies the transmural pressure at which the airway compliance reaches its maximum, while $B = 431.493$ Pa controls the steepness of the pressure–volume transition. Differentiation of equation (S80) with respect to P_{tm} gives the nonlinear, pressure-dependent compliance

$$C_C(V_C) = \frac{dV_C}{dP_{tm}} = \frac{V_{C,\max}}{B} \frac{\exp\left(-\frac{P_{tm}-A}{B}\right)}{\left[1 + \exp\left(-\frac{P_{tm}-A}{B}\right)\right]^2} = \frac{V_C}{B} \left(1 - \frac{V_C}{V_{C,\max}}\right), \quad (\text{S81})$$

which reaches its maximum near mid-range airway volumes and decreases as the airway approaches either collapse or full distension. Substitution of equation (S81) into equation (S79) gives the corresponding pressure-dependent stiffness of the compressible airways

$$K_C = \frac{2 \alpha_C^2 B V_{C,\max}}{V_C (V_{C,\max} - V_C)}, \quad (\text{S82})$$

which increases sharply as the airways volume approaches the limits of collapse or full distension.

Damping Behavior of the Compressible Airways

The dissipative behavior of the compressible airways is represented in the lumped-element framework by a single Newtonian dashpot, as illustrated in Fig. S9. This is different from the lung damping formulation, which incorporates both linear and nonlinear components to account for the tissue-level dissipation. This is because the compressible airways experience substantially smaller deformations than the lung parenchyma during respiration and phonation. As a result, the dominant mechanism of energy loss in this region arises from viscous friction rather than nonlinear tissue deformation or strain-dependent stiffening. Accordingly, a linear Newtonian damping representation is sufficient to capture the relevant physical behavior of the compressible airways. With this assumption, the damping force acting on each compressible airways piston is modeled as

$$F_{\text{damp},C}(\dot{x}) = -C_{1C} \dot{x}, \quad (\text{S83})$$

where C_{1C} denotes the viscous damping coefficient and $\dot{x}$ is the axial velocity of the compressible airways piston. The mechanical energy dissipated by this damping element over a single breathing cycle is obtained by integrating the instantaneous power loss $C_{1C} \dot{x}^2$ over the cycle duration. Because the compressible airways are represented by two identical piston–cylinder assemblies operating in parallel, energy is dissipated in both damping elements. The total dissipated energy per breathing cycle is therefore given by

$$E_{\text{cd}} = 2 C_{1C} \int_0^{T_{\text{breath}}} \dot{x}^2 dt, \quad (\text{S84})$$

where T_{breath} denotes the duration of one complete breathing cycle and E_{cd} represents the total damping energy loss per cycle. Using the kinematic relation $\dot{x} = -\dot{V}_C/(2\alpha_C)$, the dissipated energy can be written as

$$E_{\text{cd}} = \frac{C_{1C}}{2} \int_0^{T_{\text{breath}}} \left(\frac{\dot{V}_C}{\alpha_C}\right)^2 dt \quad (\text{S85})$$

The average mechanical energy dissipated by the compressible airways during one breathing cycle is provided in the supplementary materials under the section particle swarm optimization algorithm,

with values of 0.015 J/cycle for regular breathing and 0.02 J/cycle for quiet breathing. Equating these values with equation (S85) allows the damping coefficient to be computed for each breathing condition. For quiet and regular breathing, the corresponding damping coefficients are given by

$$C_{1C}^q = \frac{2E_{cd}^q}{\int_0^{T_{\text{breath},q}} \left(\frac{\dot{V}_C^q}{\alpha_C} \right)^2 dt} \quad (\text{S86})$$

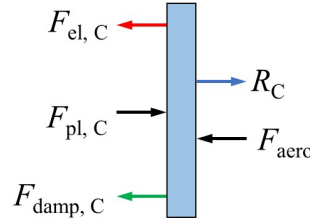
$$C_{1C}^r = \frac{2E_{cd}^r}{\int_0^{T_{\text{breath},r}} \left(\frac{\dot{V}_C^r}{\alpha_C} \right)^2 dt} \quad (\text{S87})$$

A single effective damping coefficient is adopted to represent the compressible airways across all respiratory conditions. This coefficient is defined as the arithmetic mean of the values obtained for quiet and regular breathing, $C_{1C} = (C_{1C}^q + C_{1C}^r)/2 = 7.37 \text{ N s m}^{-1}$.

Governing Equation of the Compressible Airways System

As illustrated in Fig. S10, several forces act on the piston of the compressible airways piston-cylinder system. The elastic restoring force $F_{\text{el},C}$ arises from the deformation of the airways wall and is modeled as

$$F_{\text{el},C}(t) = -K_C(t)(x(t) - x_{\text{eq}}). \quad (\text{S88})$$



Supplementary Figure S10. Free-body diagram of the piston representing the compressible airways. The piston is subjected to an elastic restoring force $F_{\text{el},C}$, a viscous damping force $F_{\text{damp},C}$, an intrapleural pressure force $F_{\text{pl},C}$ acting from the surrounding pleural space, and an aerodynamic pressure force F_{aero} arising from the airway lumen. The supportive force R_C represents structural support provided by the surrounding airway tree and connective tissues.

The viscous energy dissipation within the airway wall is represented by the damping force $F_{\text{damp},C}$, defined previously in equation (S83). The piston is further subjected to an intrapleural pressure force $F_{\text{pl},C} = \alpha_C P_{\text{pl}}(t)$ acting from the surrounding pleural cavity and an opposing aerodynamic pressure force $F_{\text{aero}} = \alpha_C P_{\text{aero}}(t)$ generated by the air contained within the airway lumen. In addition, the piston experiences a supportive force $R_C(t)$, which accounts for the mechanical support from the surrounding airway tree and connective tissues. Summing all forces acting on the piston and applying Newton's second law gives the governing equation of motion

$$M_C \ddot{x}(t) = -K_C(t)(x(t) - x_{\text{eq}}) + \alpha_C P_{\text{pl}}(t) - C_{1C} \dot{x}(t) - \alpha_C P_{\text{aero}}(t) + R_C(t) \quad (\text{S89})$$

The governing equation can be expressed in terms of compressible airways volume using the kinematic relation $V_C(t) = 2\alpha_C(x_{\max} - x(t))$, which leads to

$$\begin{aligned} -\frac{M_C}{2\alpha_C} \ddot{V}_C(t) &= K_C(t) \left(\frac{V_C(t)}{2\alpha_C} + x_{\text{eq}} - x_{\max} \right) + \alpha_C (P_{\text{pl}}(t) - P_{\text{aero}}(t)) \\ &+ \frac{C_{1C}}{2\alpha_C} \dot{V}_C(t) + R_C(t) \end{aligned} \quad (\text{S90})$$

Substituting the time-resolved pressure and volume waveforms corresponding to regular and quiet breathing into equation (S90) produces the following system

$$\begin{aligned} -\frac{M_C}{2\alpha_C} \ddot{V}_C^r(t) &= K_C^r(t) \left(\frac{V_C^r(t)}{2\alpha_C} + x_{\text{eq}} - x_{\max} \right) + \alpha_C (P_{\text{pl}}^r(t) - P_{\text{aero}}^r(t)) \\ &+ \frac{C_{1C}}{2\alpha_C} \dot{V}_C^r(t) + R_C^r(t) \end{aligned} \quad (\text{S91})$$

$$\begin{aligned} -\frac{M_C}{2\alpha_C} \ddot{V}_C^q(t) &= K_C^q(t) \left(\frac{V_C^q(t)}{2\alpha_C} + x_{\text{eq}} - x_{\max} \right) + \alpha_C (P_{\text{pl}}^q(t) - P_{\text{aero}}^q(t)) \\ &+ \frac{C_{1C}}{2\alpha_C} \dot{V}_C^q(t) + R_C^q(t) \end{aligned} \quad (\text{S92})$$

All parameters appearing in equations (S91) and (S92) have been defined in the preceding sections. These governing equations therefore provide the supportive forces $R_C^r(t)$ and $R_C^q(t)$ associated with regular and quiet breathing directly from the mechanical balance of the compressible airways system.

Modeling of Compressible Airways Supportive Forces

Following the same methodology developed for the lung supportive forces, the compressible airways supportive force is generalized in terms of the instantaneous mechanical state of the airways rather than the specific breathing pattern from which it is derived. For this, an effective compressible airways volume is introduced as a linear combination of the instantaneous volume and its temporal derivatives as follows

$$\tilde{V}_C(t) = V_C(t) + \beta_C \dot{V}_C(t) + \gamma_C \ddot{V}_C(t), \quad (\text{S93})$$

where β_C and γ_C are scalar coefficients that quantify the influence of flow rate and inertial acceleration on the effective mechanical state of the compressible airways.

The coefficients β_C and γ_C are identified by enforcing the condition that quiet and regular breathing must produce identical supportive forces whenever they share the same effective compressible airways volume. This condition is satisfied at the intersection points of the supportive-force trajectories $R_C^q(t)$ and $R_C^r(t)$. Intersection times are identified using a cubic spline interpolation of the two force signals. At each intersection time t_i , the corresponding values of $V_C(t_i)$, $\dot{V}_C(t_i)$, and $\ddot{V}_C(t_i)$ are extracted for both breathing conditions. Using two such intersection points $t_{i,1}$ and $t_{i,2}$, the following linear system is constructed

$$\begin{aligned} \beta_C (\dot{V}_C^q(t_{i,1}) - \dot{V}_C^r(t_{i,1})) + \gamma_C (\ddot{V}_C^q(t_{i,1}) - \ddot{V}_C^r(t_{i,1})) &= V_C^r(t_{i,1}) - V_C^q(t_{i,1}) \\ \beta_C (\dot{V}_C^q(t_{i,2}) - \dot{V}_C^r(t_{i,2})) + \gamma_C (\ddot{V}_C^q(t_{i,2}) - \ddot{V}_C^r(t_{i,2})) &= V_C^r(t_{i,2}) - V_C^q(t_{i,2}) \end{aligned} \quad (\text{S94})$$

Solving this system gives $\beta_C = 14.762\text{ s}$ and $\gamma_C = 0.014\text{ s}^2$. These values indicate that the compressible airways supportive force is strongly influenced by the airflow rate, while inertial effects play a minor role. The ratio $|\gamma_C/\beta_C| \approx 9.5 \times 10^{-4}$ confirms that the contribution of the second temporal derivative is nearly three orders of magnitude smaller than that of the first derivative. As a result, the acceleration term can be neglected without noticeable loss of accuracy, leading to the reduced effective volume definition

$$\tilde{V}_C(t) = V_C(t) + \beta_C \dot{V}_C(t) \quad (\text{S95})$$

With β_C determined, the unified supportive force law is written as

$$R_C(t) = A_C \tilde{V}_C(t) + B_C \quad (\text{S96})$$

The coefficients A_C and B_C are obtained from

$$A_C = \frac{R_C^r - R_C^q}{\tilde{V}_C^r - \tilde{V}_C^q} \quad (\text{S97})$$

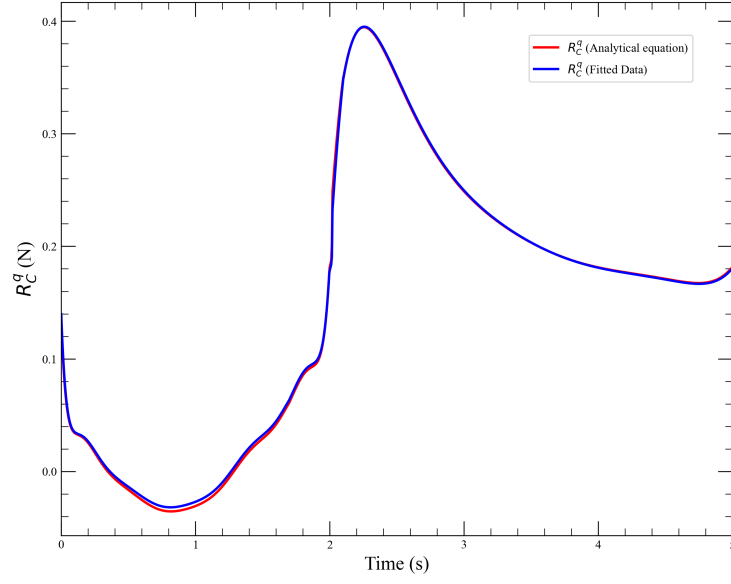
$$B_C = R_C^q - A_C \tilde{V}_C^q \quad (\text{S98})$$

Although the coefficients A_C and B_C are time-dependent quantities that vary over the breathing cycle, we replace these time-varying coefficients by their cycle-averaged values, denoted by $\bar{A}_C = -1076.888\text{ N m}^{-3}$ and $\bar{B}_C = 0.219\text{ N}$. This averaging strategy mirrors the approach adopted for the lung supportive forces. Using these averaged coefficients, the compressible airways supportive force is approximated as $R_C(t) \approx \bar{A}_C \tilde{V}_C(t) + \bar{B}_C$, which provides a linear state-based description of the supportive force in terms of the effective compressible airways volume. The predictive accuracy of this approximation is evaluated by comparing the reconstructed supportive force with the forces obtained directly from the governing equations under quiet and regular breathing conditions. As shown in Figs. S11 and S12, the reconstructed force achieves a fit accuracy of 98.7% for quiet breathing and 97.9% for regular breathing.

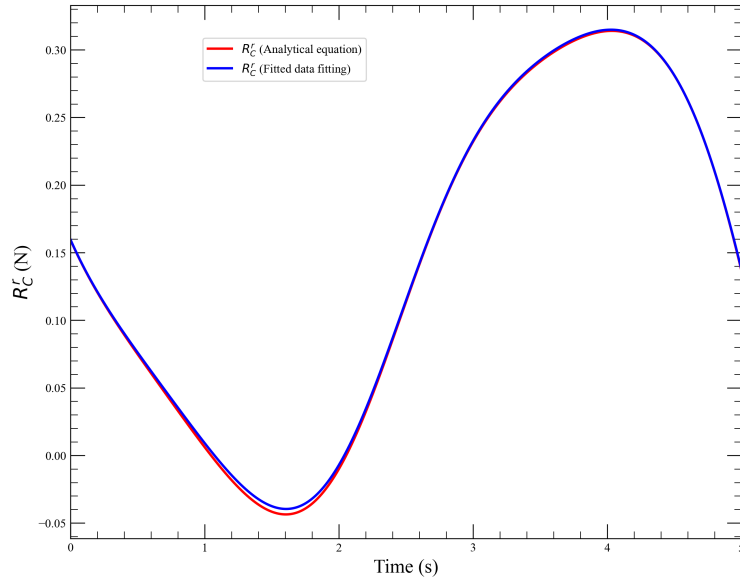
Flow Resistances

The flow resistance quantifies the pressure drop required to drive a unit volumetric airflow through the airway network. In the present lumped-element formulation, seven distinct resistive components are incorporated to represent pressure losses occurring along different anatomical regions of the respiratory tract, as was illustrated in Fig. S2. These components are the lung tissue resistance R_{LT} , the small airways resistance FR_S , the compressible airways resistance FR_C , the tracheal resistance FR_T , the vocal folds resistance FR_{VF} , the upper airways resistance FR_U , and the expiratory resistance FR_E . Together, these elements account for viscous dissipation, geometric constrictions, and deformation-induced losses encountered by the airflow between the lungs and the atmosphere.

The lung tissue resistance represents energy losses associated with the deformation of the lung parenchyma during volume change. This resistance is assumed to be constant and is taken as $1.96 \times 10^4\text{ Pa s m}^{-3}$ as reported by [10]. This resistance captures the energy loss due to the tissue deformation and is independent of the airway geometry. The resistive contribution of the small



Supplementary Figure S11. Time-resolved compressible airways supportive force during quiet breathing. The red curve corresponds to the supportive force computed directly from the governing equation of the compressible airways piston–cylinder system, while the blue curve shows the force reconstructed using the unified state-based formulation. The close agreement demonstrates that the averaged linear representation accurately captures the compressible airways supportive force.



Supplementary Figure S12. Time-resolved compressible airways supportive force during regular breathing. The analytical solution obtained from the governing equation is shown in red, and the force reconstructed from the unified state-based model is shown in blue. The strong overlap between the two curves confirms that the reduced-order formulation accurately reproduces the supportive force dynamics.

airways reflects the flow limitation imposed by bronchioles. This volume-dependent behavior is modeled using an exponential relationship of the form

$$FR_S = R_{s,d} \exp \left[-K_S \frac{V_L - RV}{TLC - RV} \right] + R_{s,m}, \quad (\text{S99})$$

where $R_{s,d} = 215.75 \text{ kPa s m}^{-3}$ characterizes the dynamic component of small airways resistance that decreases with the lung inflation, $K_S = 10.9$ governs the sensitivity of this resistance to the lung volume, and $R_{s,m} = 1.96 \text{ kPa s m}^{-3}$ represents the minimum resistance attained at high lung volumes. The residual volume is taken as $RV = 23 \times 10^{-6} \text{ m}^3$. This formulation captures the physiological opening of small airways as the lung volume increases.

In addition to this empirical description, viscous losses within the bronchi are evaluated using Poiseuille's law. The resistance of a single bronchus of length L_B and diameter D_C is given by

$$FR_B = \frac{128\mu L_B}{\pi(D_C)^4} \quad (\text{S100})$$

Since the compressible airways representation consists of two identical bronchus, arranged in parallel, the effective resistance becomes one half of the single-path resistance, giving $R_{B,\text{eff}} = \frac{64\mu L_B}{\pi(D_C)^4}$.

The compressible airways resistance incorporates both geometric constriction effects of the peripheral airways and viscous losses within the two bronchi. Thus, it is modeled as

$$FR_C = \bar{k}_C \left(\frac{V_{C,\text{max}}}{V_C} \right)^2 + \frac{64\mu L_B}{\pi(D_C)^4}, \quad (\text{S101})$$

where $\bar{k}_C = 20.59 \text{ kPa s m}^{-3}$ denotes the minimum resistance corresponding to the maximal airway expansion of the peripheral airways. Next, is tracheal flow resistance, which is also evaluated using Poiseuille's law by modeling the trachea as a rigid cylindrical conduit supporting the laminar airflow. In the present study, the trachea is represented as a straight tube with an average length of $L = 11.8 \text{ cm}$ and a mean internal diameter of $D = 17 \text{ mm}$. The dynamic viscosity of air at body temperature is taken as $\mu = 1.89 \times 10^{-5} \text{ Pa s}$. Substituting these physiological parameters into Poiseuille's formulation gives the tracheal flow resistance

$$FR_T = \frac{8\mu L}{\pi r^4} = 1.15 \text{ kPa s m}^{-3} \quad (\text{S102})$$

This resistance accounts for viscous dissipation along the tracheal segment and remains constant under the assumption of laminar flow and negligible tracheal deformation. At the downstream of the trachea, an additional and highly dynamic resistive element arises during sustained phonation due to the periodic opening and closing of the glottis. Such a behavior imposes a time-varying aerodynamic impedance on the airflow, which is represented by the glottal flow resistance FR_{VF} . The instantaneous geometry of the glottis is quantified by the glottal area waveform $A_g(t)$, extracted from high-speed videoendoscopy data using the segmentation framework described earlier. Since the vocal folds form a rapidly varying constriction, the associated resistance is defined as

$$FR_{VF}(t) = \frac{P_{\text{sub}}(t) - P_{\text{sup}}(t)}{Q_g(t)}, \quad (\text{S103})$$

where P_{sub} and P_{sup} denote the spatially averaged subglottal and supraglottal pressures and $Q_g(t)$ is the instantaneous glottal flow rate. Unlike the resistive elements associated with respiration,

which vary slowly with the lung volume or transmural pressure, the glottal resistance oscillates at the fundamental frequency of vocal folds vibration and can change substantially within a single vibratory cycle. When the glottis is widely open, the resistance approaches a minimum value, whereas during near closure the resistance increases sharply and becomes infinity when the vocal folds are fully closed, reflecting the temporary interruption of airflow. The detailed computational procedure for evaluating $FR_{VF}(t)$ is described in the supplementary materials under the section phonation dynamics within the lumped-element architecture.

The upper airways flow resistance represents the cumulative pressure losses contributed by the anatomical structures extending from the supraglottal region to the oral cavity. These losses arise from a combination of geometric curvature, gradual and abrupt area changes, flow separation, and local turbulence within the pharyngeal and oral passages. In previous work, Marconi and De Lazzari [10] modeled the combined resistance of the two bronchi, trachea, and upper airways using a nonlinear flow-dependent formulation of the form

$$FR_{U,\text{combined}} = A_U + K_U |F|, \quad (\text{S104})$$

where A_U represents the minimal resistance at low flow rates and K_U characterizes the increase in resistance with airflow magnitude F . In the present model, resistances offered by the trachea and the two bronchi are treated separately. Thus, the upper airways resistance is reformulated to represent only the supraglottal-to-oral segment. The resulting modified upper airways resistance is expressed as

$$FR_U = A_{U,\text{mod}} + K_U |F|, \quad (\text{S105})$$

where $A_{U,\text{mod}} = 31.04 \text{ kPa s m}^{-3}$ denotes the minimal upper airways resistance after removal of the tracheal component and $K_U = 45.11 \text{ MPa s}^2 \text{ m}^{-6}$ characterizes the nonlinear increase in upper airways resistance with the airflow magnitude. The final resistive component introduced during phonation occurs at the exit of the oral cavity and is referred to as the expiratory flow resistance FR_E . This resistance quantifies the additional pressure required to discharge airflow from the lips into the surrounding environment. Unlike the upstream airway segments, where pressure losses are primarily associated with viscous friction along compliant or rigid walls, the expiratory resistance arises from a rapid geometric transition at the mouth opening. As air exits the oral cavity, it undergoes a sudden expansion from the finite lip aperture into the effectively unbounded external domain. This abrupt expansion prevents the airflow from remaining attached to the boundaries, causing flow separation at the lip edges and the formation of a free jet. The kinetic energy carried by this jet is partially dissipated through turbulent mixing with the surrounding quiescent air, leading to irreversible mechanical energy losses. These losses manifest as a pressure drop across the lip opening. A direct physical manifestation of this process is the perceptible air puff felt when placing a hand in front of the mouth during phonation or forced exhalation. This sensation reflects the momentum flux of the emerging jet, which indicates a positive gauge pressure at the lip exit. These processes are described by the classical head-loss relation for a sudden expansion, in which a flow passes from a conduit of cross-sectional area A_1 and mean velocity V_1 into a downstream region of area A_2 and mean velocity V_2 . The associated pressure drop can be written as

$$\Delta P = \frac{1}{2} \rho_a \left(V_1^2 - V_2^2 \right) \left[1 - \left(\frac{A_1}{A_2} \right)^2 \right], \quad (\text{S106})$$

where ρ_a is the air density. In the phonatory setting, the upstream section corresponds to the lip opening and the downstream region is the external environment, which during sustained phonation

can be treated as effectively unbounded, corresponding to the limiting case $A_2 \rightarrow \infty$. In this limit, $A_1/A_2 \rightarrow 0$ and the downstream mean velocity approaches $V_2 \rightarrow 0$, so equation (S106) reduces to

$$\Delta P = \frac{1}{2} \rho_a V_1^2 \quad (\text{S107})$$

showing that the required pressure surplus at the lips is set by the kinetic energy per unit volume of the emerging jet. Using continuity at the lip opening, the mean exit velocity is

$$V_1(t) = \frac{Q_g(t)}{A_l}, \quad (\text{S108})$$

where $Q_g(t)$ is the glottal flow rate and A_l is the lip opening area. Substituting equation (S108) into equation (S107) gives

$$\Delta P(t) = \frac{1}{2} \rho_a \left(\frac{Q_g(t)}{A_l} \right)^2 \quad (\text{S109})$$

Based on this formulation, the expiratory flow resistance can be defined as

$$FR_E(t) = \frac{\Delta P(t)}{Q_g(t)} = \frac{1}{2} \rho_a \frac{|Q_g(t)|}{A_l^2}, \quad (\text{S110})$$

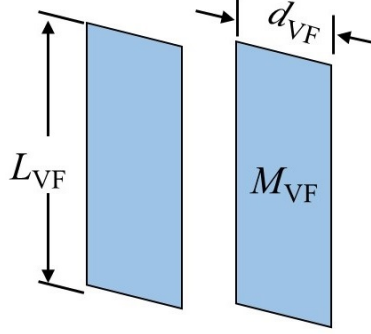
where the absolute value ensures that $FR_E(t)$ remains nonnegative and that the associated pressure loss always opposes the direction of flow.

Phonation Dynamics within the Lumped-Element Architecture

The unified lumped-element framework incorporates a mechanical representation of the vocal folds that is driven by subglottal airflow. Since the glottis forms the narrowest constriction in the phonatory airway, it governs the instantaneous impedance to the airflow and plays a dominant role in shaping the aerodynamic loading. In this study, phonatory dynamics are represented using a single-mass vocal fold model, illustrated schematically in Fig. S13. In this formulation, each vocal fold is idealized as a three-dimensional rectangular body whose distributed tissue mechanics are reduced to a single mass, which is governed by a linear spring and a nonlinear damping element. Despite its reduced order, such a single mass model is capable of capturing the essential features of vocal folds vibration, including the self-oscillation, asymmetric loading during opening and closing phases, and the sharp increase in mechanical resistance near collision. The geometric, inertial, and constitutive parameters employed here are adopted directly from the study of Bin Ali *et al.*[18]. Accordingly, the superior–inferior length of each vocal fold is set to $L_{VF} = 1.60$ cm, the medial–lateral thickness is taken as $d_{VF} = 0.32$ cm, and the effective mass of each fold is specified as $M_{VF} = 0.120$ g. The elastic response of the tissue is modeled using a linear stiffness constant $K_{VF} = 396.6550$ N m^{−1} and the dissipative characteristics of the vocal folds are modeled by the following nonlinear damping formulation

$$C_{VF}(x) = C_{\min} + C_f \tanh(x_{c,VF} - x_{VF}) \quad (\text{S111})$$

Here, $C_{\min} = 0$ denotes the minimum viscous resistance when vocal folds have the maximum distance from each other, and $C_f = 7.5362 \times 10^3$ controls the magnitude of damping amplification as the folds approach collision. The variable x_{VF} represents the instantaneous medial displacement



Supplementary Figure S13. Schematic representation of the single-mass vocal fold model in the present modeling framework. Each vocal fold is represented as a rigid rectangular body with effective mass M_{VF} , superior-inferior length L_{VF} , and medial-lateral thickness d_{VF} . The two folds are positioned symmetrically about the glottal midline.

of each vocal fold measured from the midline, while $x_{c,VF}$ denotes the critical displacement at which the initial contact between the two folds occurs. This definition follows the modeling convention that $x_{c,VF}$ marks the transition between the open and closed phases of the glottal cycle. When $x_{VF} > x_{c,VF}$, the vocal folds remain separated and the glottis is open. When $x_{VF} = x_{c,VF}$, the folds are just in contact, and when $x_{VF} < x_{c,VF}$, a compressive interaction between the folds takes place. The change in damping is modeled using the positive portion of the hyperbolic tangent function, which provides a smooth yet rapidly increasing damping profile that reflects the sharp rise in tissue resistance near the contact without introducing numerical discontinuities.

During sustained phonation, the periodic opening and closing of the vocal folds produce a strongly time-varying pressure difference between the subglottal and supraglottal regions. This transglottal pressure difference arises from the combined effects of the airflow acceleration through the glottal constriction and viscous losses associated with the narrowing of the glottal channel as the folds approach contact. Within the present lumped-element formulation, the instantaneous pressure drop across the glottis is expressed as

$$P_{\text{sub}}(t) - P_{\text{sup}}(t) = \frac{k_t \rho_a}{2} \frac{|Q_g(t)| Q_g(t)}{A_g(t)^2} + \gamma \frac{Q_g(t)}{A_g(t)^3}, \quad (\text{S112})$$

where P_{sub} and P_{sup} denote the spatially averaged subglottal and supraglottal pressures, respectively, $Q_g(t)$ is the glottal volume flow rate, and $A_g(t)$ is the instantaneous glottal area extracted from high-speed videoendoscopy data. The coefficient $k_t = 1$ represents the transglottal pressure coefficient, and ρ_a is the air density. The first term in equation (S112) represents the inertial component of the pressure drop and dominates when the vocal folds are widely separated and the airflow accelerates rapidly through the glottis. This quadratic dependence on the flow reflects the conversion of pressure energy into the kinetic energy of the jet formed within the glottal constriction. The second term captures the viscous dissipation within the glottal channel and becomes dominant as the glottal area decreases during the closing phase of vibration. This term rises sharply near the closure and accounts for the rapid increase in the flow resistance that accompanies narrowing of the glottis. To complete the aerodynamic coupling between the vocal folds and the surrounding airway system, the glottal flow rate $Q_g(t)$ is computed using the nonlinear formulation proposed

by Lucero and Schoentgen [19] as follows

$$Q_g(t) = \frac{2 A_g(t)^3 \delta_p(t)}{\frac{\rho_a c A_g(t)^3}{A^*} + \gamma + \sqrt{\left(\frac{\rho_a c A_g(t)^3}{A^*} + \gamma\right)^2 + 2 k_t \rho_a A_g(t)^4 |\delta_p(t)|}} \quad (\text{S113})$$

Here, $\delta_p(t)$ denotes the effective driving pressure acting across the glottis and is defined as $\delta_p(t) = (1 + r_s) P_{\text{sub}}^+(t) - (1 + r_e) P_{\text{sup}}^-(t)$, where P_{sub}^+ and P_{sup}^- represent the forward-traveling pressure waves in the subglottal and supraglottal tracts, respectively, and r_s and r_e are the corresponding acoustic reflection coefficients. The parameter $c \approx 35000$ cm/s denotes the speed of sound in air. The effective acoustic coupling area A^* is defined through $\frac{1}{A^*} = \frac{1}{A_s} + \frac{1}{A_e}$, where A_s and A_e denote the cross-sectional areas of the subglottal and supraglottal airways, respectively.

In the present study, the effective driving pressure δ_p is selected based on both the experimental validation and physiological considerations. For benchmarking against the excised-larynx measurements reported by Lehoux *et al.* [20], δ_p was set to 2.72 kPa, which corresponds to the upper range of steady subglottal pressure levels reported for sustained phonation in their anechoic configuration. For general phonation simulations, however, the mean subglottal pressure typically lies within the range of approximately 5–10 cmH₂O. Accordingly, a target mean subglottal pressure of 7.5 cmH₂O is adopted following Hoffman *et al.* [21]. Under these conditions, $\delta_p(t)$ is determined iteratively. The procedure begins with an initial estimate corresponding to 7.5 cmH₂O, after which the resulting subglottal pressure waveform $P_{\text{sub}}(t)$ is computed and its cycle-averaged value is evaluated. If the computed mean subglottal pressure is lower than the target value, δ_p is increased; if it exceeds the target, δ_p is reduced. This iterative adjustment is repeated until the relative error between the simulated and target mean subglottal pressures falls below 0.01%. Based on this iterative procedure, the effective driving pressure converges to $\delta_p = 8.82$ cmH₂O.

Numerical Method

From the governing equations of the lung and the compressible airways given by equations (S24) and (S78), they can be combined into a single coupled dynamic constraint that governs the simultaneous evolution of lung volume and compressible airways volume during phonation. Eliminating the pleural pressure and rearranging the inertial elastic dissipative and supportive force contributions gives the following composite governing equation

$$\begin{aligned} \frac{M_L}{\alpha_L^2} \ddot{V}_L(t) - \frac{M_C}{2\alpha_C^2} \ddot{V}_C(t) + F R_s \dot{V}_L(t) + \frac{C_{1L}}{\alpha_L^2} \dot{V}_L(t) - \frac{C_{1C}}{2\alpha_C^2} \dot{V}_C(t) + \frac{C_{2L}}{\alpha_L^3} \frac{\dot{V}_L(t)^3}{|\dot{V}_L(t)|} \\ + \frac{K_L(t)}{\alpha_L} \left(\frac{V_L(t)}{\alpha_L} + h_{\text{eq}} - h_{\text{max}} \right) - \frac{K_C(t)}{\alpha_C} \left(\frac{V_C(t)}{2\alpha_C} + x_{\text{eq}} - x_{\text{max}} \right) \\ - \frac{M_L g}{\alpha_L} + \frac{R_L}{\alpha_L} - \frac{R_C}{\alpha_C} = 0 \end{aligned} \quad (\text{S114})$$

Equation (S114) represents a strongly nonlinear second order differential algebraic system that couples lung mechanics, compressible airways mechanics, tissue viscoelasticity, gravity, and flow dependent resistive losses. The unknowns $V_L(t)$ and $V_C(t)$ are not independent but are additionally constrained by volumetric continuity across the glottis. During phonation, the total volumetric balance is governed by $\dot{V}_L(t) + \dot{V}_C(t) + \dot{V}_{\text{VF}}(t) = -Q_g(t)$ or equivalently $\dot{V}_L(t) + \dot{V}_C(t) = -\tilde{Q}_g(t)$,

where $\tilde{Q}_g(t) = Q_g(t) + \dot{V}_{VF}(t)$ denotes the adjusted glottal flow rate that accounts for the instantaneous volumetric change of the vocal folds. This kinematic constraint introduces an additional coupling between the lung and airways accelerations through the relation $\ddot{V}_C(t) = -\ddot{V}_L(t) - \ddot{\tilde{Q}}_g(t)$. Together equation (S114) and the continuity constraint form a closed dynamical system in which the lung acceleration $\ddot{V}_L(t)$ becomes the primary dynamic variable. Due to the presence of nonlinear stiffness terms, exponential volume dependent resistances, cubic velocity dependent damping, and externally prescribed glottal flow, the system does not have a closed form analytical solution. Thus, a direct numerical time-integration strategy is required.

In the present work, the coupled lung–airways dynamics are solved using a fourth order Runge–Kutta (RK4) time integration scheme. The RK4 method is particularly well suited for this problem because it provides high order accuracy for smooth but strongly nonlinear systems while maintaining numerical stability under the high temporal resolution. To numerically integrate the coupled lung–compressible airways dynamics defined by equation (S114) together with the volumetric continuity constraint, the governing equations are first transformed into an explicit first order state-space representation suitable for time marching. The primary state variables are chosen as the lung volume and its first derivative together with the compressible airways volume and its first derivative. Accordingly, the state vector is defined as

$$\mathbf{y}(t) = \begin{bmatrix} V_L(t) \\ \dot{V}_L(t) \\ V_C(t) \\ \dot{V}_C(t) \end{bmatrix} \quad (\text{S115})$$

The time derivatives of the state variables follow directly from kinematics and the governing equations. The first and third components satisfy the identities $\frac{dV_L}{dt} = \dot{V}_L$ and $\frac{dV_C}{dt} = \dot{V}_C$, while the second and fourth components require evaluation of the dynamic force balance. Using the continuity constraint $\dot{V}_L(t) + \dot{V}_C(t) = -\tilde{Q}_g(t)$, we get $\dot{V}_C(t) = -\tilde{Q}_g(t) - \dot{V}_L(t)$ and $\ddot{V}_C(t) = -\ddot{V}_L(t) - \ddot{\tilde{Q}}_g(t)$. Substitution of this expression into equation (S114) produces a single scalar equation that is solved explicitly for the lung acceleration $\ddot{V}_L(t)$. All remaining terms are treated as known functions of the current state $\mathbf{y}(t)$ and time t , including nonlinear elastic forces, velocity dependent damping, gravity, supportive forces, and the glottal flow rate. Once $\ddot{V}_L(t)$ is obtained, the airway acceleration $\ddot{V}_C(t)$ follows directly from the constraint equation above. The resulting system can therefore be written compactly as $\frac{d\mathbf{y}}{dt} = \mathbf{f}(t, \mathbf{y}(t))$, where the vector valued function $\mathbf{f}$ contains both the kinematic relations and the dynamically evaluated accelerations. This formulation is well suited for explicit Runge-Kutta integration. Time is discretized uniformly with a constant step size $\Delta t = \frac{1}{4000}$ s, which matches the temporal resolution of the high-speed videoendoscopy data. At each discrete time level $t_n = n\Delta t$, the fourth order Runge-Kutta method advances the solution according to $\mathbf{y}_{n+1} = \mathbf{y}_n + \frac{\Delta t}{6}(\mathbf{k}_1 + 2\mathbf{k}_2 + 2\mathbf{k}_3 + \mathbf{k}_4)$ where the intermediate slope evaluations are given by $\mathbf{k}_1 = \mathbf{f}(t_n, \mathbf{y}_n)$ $\mathbf{k}_2 = \mathbf{f}(t_n + \frac{\Delta t}{2}, \mathbf{y}_n + \frac{\Delta t}{2}\mathbf{k}_1)$ $\mathbf{k}_3 = \mathbf{f}(t_n + \frac{\Delta t}{2}, \mathbf{y}_n + \frac{\Delta t}{2}\mathbf{k}_2)$ $\mathbf{k}_4 = \mathbf{f}(t_n + \Delta t, \mathbf{y}_n + \Delta t\mathbf{k}_3)$.

At each intermediate stage the adjusted glottal flow rate $\tilde{Q}_g(t)$ and its time derivative are evaluated. After each full time step, the continuity constraint is explicitly enforced at the velocity level to prevent the accumulation of numerical drift between the lung and compressible airways volumes. All nonlinear terms, resistances, stiffnesses, damping contributions, and supportive forces are recomputed at each Runge-Kutta substep using the intermediate states $\mathbf{y}_n + \frac{\Delta t}{2}\mathbf{k}_i$.

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