Respiratory Laryngeal Coordination in Airflow Conservation and Reduction of Respiratory Effort of Phonation

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Summary: Objective. This study evaluates the need of airflow conservation and the effect of glottal resistance on respiratory effort of phonation under different phonation conditions.
Methods. A computational model of the pressure–volume–flow relationship of the respiratory system is developed.
Results. Simulations show that increasing the glottal resistance reduces the glottal airflow and allows phonation to be sustained for a longer breath group duration. For a given breath group duration, the reduced airflow also allows phonation to be sustained within a narrow range of lung volumes, thus lowering the overall respiratory effort.
Conclusions. This study shows that for breath group durations and subglottal pressures typical of normal conversational speech, airflow conservation or maintaining "effortless" respiratory support does not provide a stricter requirement on the glottal resistance than that required for initiating phonation. However, the need for airflow conservation and respiratory effort reduction becomes relevant when the target subglottal pressure and breath group duration increase as in prolonged speech or singing or in conditions of weakened pulmonary function. In those conditions, the glottal resistance is expected to increase proportionally with increasing subglottal pressure to conserve airflow consumption and reduce respiratory effort.
Key Words: Airflow conservation–Respiratory–laryngeal coordination–Glottal resistance–Respiratory effort of phonation–Respiratory model.

INTRODUCTION

Experiments with human subjects have shown that as one increases vocal intensity, the subglottal pressure often increases significantly, whereas the mean airflow remains relatively constant.[1–4] In some conditions, the mean glottal flow even decreases slightly with increasing intensity.[1] Although this relatively constant airflow results from laryngeal adjustments, which may be required to facilitate vocal loudness increase, one may wonder if the possible need for conservation of airflow and respiratory effort may also contribute to some degree. Due to the finite vital capacity of the lungs, it is possible that airflow has to be maintained at a certain level so that speech can continue as long as required or until an appropriate prosodic boundary before one takes the next breath. Such demand for airflow conservation is particularly important in singing, which often requires loud phonation for a prolonged time.[5] On the other hand, because the elastic recoil force of the lungs and thus respiratory effort depend critically on the lung volume, maintaining a low airflow would allow phonation to be sustained at an optimal lung volume range for a longer time, thus reducing overall respiratory effort of speech.

The goal of this study is to evaluate if there is such need of airflow conservation, and the effect of glottal resistance on respiratory effort of phonation under different phonation conditions (breath group duration and target subglottal pressure or vocal intensity). It is hypothesized that airflow conservation may not be a concern for normal speech, but may pose a physiological constraint for phonation conditions requiring higher subglottal pressure for a longer time. For this purpose, a muscular–aerodynamic model of the respiratory system is developed in this study to investigate airflow consumption and respiratory effort required to maintain a target subglottal pressure for breath group durations typical of normal speech and singing at different glottal resistance conditions. This respiratory model may also be combined with a self-oscillating phonation model, which may find applications in natural speech synthesis.

MODEL

The mechanics of respiratory system has been well described in previous studies (eg, Hixon[6]). Figure 1 shows a sketch of the respiratory model of this study. The lungs are subject to the lung pressure P_{alv} and a net expiratory muscular force P_{exp} (negative sign indicates that the force is inspiratory). In addition to these external forces, the elastic recoils of the lungs and thorax also generate a relaxation pressure P_{rlx} acting on the lungs (positive values indicate a pressure directed toward the lungs). As in previous studies (eg, Venegas et al[7]), the relaxation pressure P_{rlx} and lung volume V_{lung} are related by a sigmoid function (Figure 1B):

$$V_{lung} = RV + \frac{VC}{1 + ae^{-P_{rlx}/d}}; \quad P_{rlx} = -d \ln\left(\frac{TLC - V_{lung}}{a(V_{lung} - RV)}\right), \quad (1)$$

where RV is the lung residual volume, VC is the lung vital capacity, total lung capacity $(TLC) = RV + VC$ is the total lung capacity, and a and d are two model coefficients. These two coefficients are determined by considering the following conditions when the lung volume equals the functional residual capacity (FRC):

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$$P_{rlx}\big|_{V_{lung}=FRC}=0;\quad \left.\frac{dV_{lung}}{dP_{rlx}}\right|_{V_{lung}=FRC}=E$$

$$a=\frac{TLC-FRC}{FRC-RV};\quad d=\frac{(FRC-RV)(TLC-FRC)}{VC\cdot E},\qquad (2)$$

where E is the respiratory system compliance at the FRC. Assume a quasi-steady respiratory process, the alveolar pressure is

$$P_{alv}=P_{rlx}+P_{exp}.\qquad (3)$$

The lung volume changes because of two factors: changes in the P_{exp}, which compresses or enlarges the lungs according to Boyle's law, and airflow Q out of the lungs:

$$\frac{dV_{lung}}{dt}=-Q-\frac{V_{lung}}{P_{atm}+P_{alv}}\frac{dP_{alv}}{dt},\qquad (4)$$

where P_{atm} is the atmospheric pressure. Equation 4 can be rewritten in a format for numerical time integration with a time step of Δt:

$$V_{lung}\left(\frac{P_{atm}+P_{alv}}{P_{atm}+P_{alv,0}}\right)=V_{lung,0}-Q\Delta t.\qquad (5)$$

When the glottal resistance is specified as R_g, the subglottal pressure P_s can be calculated from the P_{alv} as:

$$P_s=P_{alv}\frac{R_g}{R_{law}+R_g}=P_{alv}-QR_{law},\qquad (6)$$

where R_{law} is the flow resistance of the lower airway. The second expression in Equation 6 can be used to couple the respiratory model (Equations 1, 3, and 4) to a self-oscillating phonation model, eg, the two-mass model or a continuum model of phonation using a one-dimensional flow description.[8]

In this study, the following model parameter values are used, as adopted from Hoit and Hixon[9]; TLC = 7 L, RV = 2 L, FRC = 3.5 L. The respiratory system compliance is set to 0.001 L/Pa.[10] The glottal resistance during normal phonation is in the range of 1–9 Pa·s/mL. This value is expected to be even lower in pathological conditions such as vocal fold paralysis. In this study, the glottal resistance with values in the range of 0.2–9 Pa·s/mL is considered.

For each simulation condition, the lung volume is initially set at the FRC, at which the relaxation pressure is zero. The simulation starts with an inspiration period of 0.5 seconds followed by expiration of a certain duration of interest. In the inspiration phase, the glottal resistance is set at 0.1 Pa·s/mL, simulating open glottis breathing conditions, and the inspiratory muscle pressure is increased sinusoidally from zero to the desired peak value P_{ins}. In the expiration phase, the expiratory muscular pressure is either set at zero (Figure 2) or varied to maintain a target subglottal pressure (Figures 3–7).

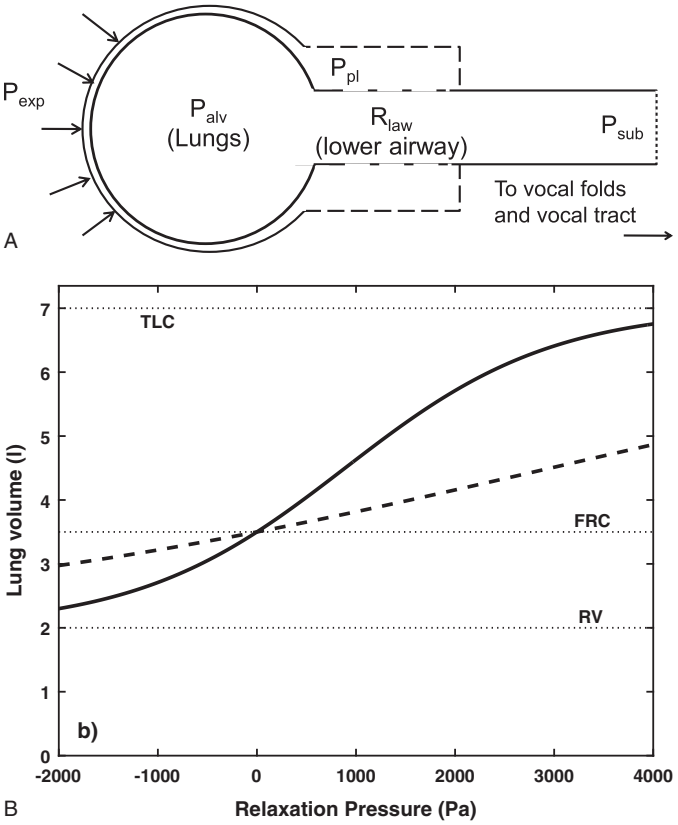

FIGURE 1. **A**. A sketch of the respiratory model. P_{exp}, the net expiratory muscle pressure; P_{alv}, the alveolar pressure; P_{pl}, the intrapleural pressure; P_{sub}, the subglottal pressure; R_{law}, the lower airway resistance. **B**. The lung volume–relaxation pressure curve used in this study for normal lung compliance (solid line) and reduced lung compliance (dashed line). FRC, functional residual capacity; RV, lung residual volume; TLC, total lung capacity.

RESULTS

General model behavior

Figure 2 shows the subglottal pressure and lung volume as a function of time for different conditions of the glottal resistance (1, 4, and 9 Pa·s/mL) and peak inspiratory muscle pressure in the inspiratory phase (–0.6, –1.5, and –2.4 kPa). For all conditions shown, no expiratory muscle pressure is imposed in the expiratory phase. Without any expiratory muscle pressure, the subglottal pressure is determined by the relaxation pressure, which decreases with decreasing lung volume as airflow rushes out of the lungs. This rate of decline in the subglottal pressure decreases with increasing glottal resistance, which reduces the glottal airflow and thus the rate of decrease in the lung volume. This increases the duration of the expiratory phase before inspiration is required. Alternatively, the duration of the expiratory phase can also be increased by increasing the inspiratory muscle pressure in the preceding inspiration phase to start expiration at a higher lung volume, which, however, has a much smaller effect on the rate of decline of the subglottal pressure.

The subglottal pressure is often maintained at a desirable value during phonation. Considering a typical target subglottal pressure of 800 Pa, Figure 3 shows the net respiratory muscle pressure required to maintain this target subglottal pressure, and the corresponding change in the lung volume with time. For a glottal resistance of 1 Pa·s/mL and an inspiratory muscle pressure of –600 Pa, the target subglottal pressure can only be maintained