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).

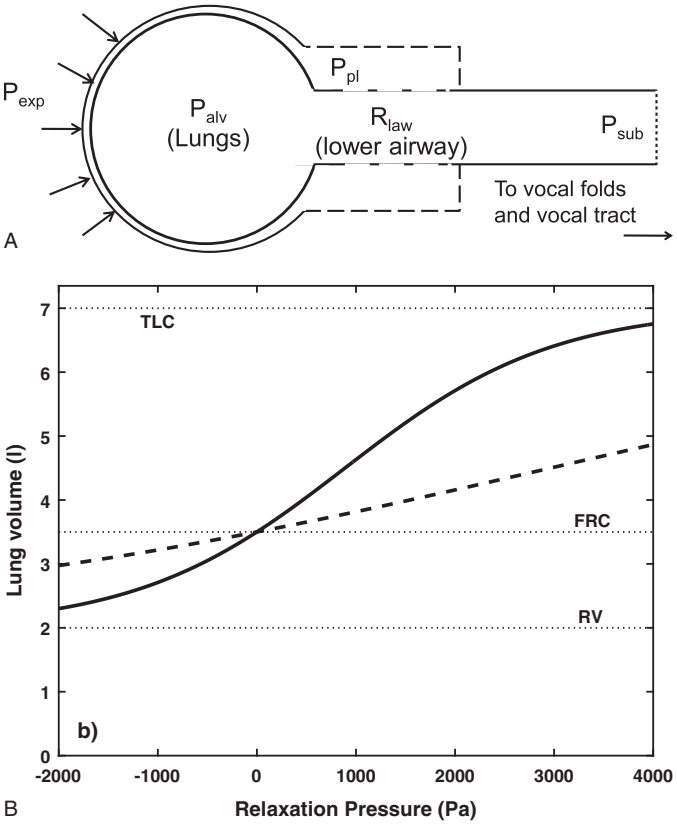

FIGURE 1. **A**. A sketch of the respiratory model. P_{exp}, the net expiratory muscle pressure; P_{alv}, the alveolar pressure; P_{pl}, the intrapleural pressure; P_{sub}, the subglottal pressure; R_{law}, the lower airway resistance. **B**. The lung volume–relaxation pressure curve used in this study 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

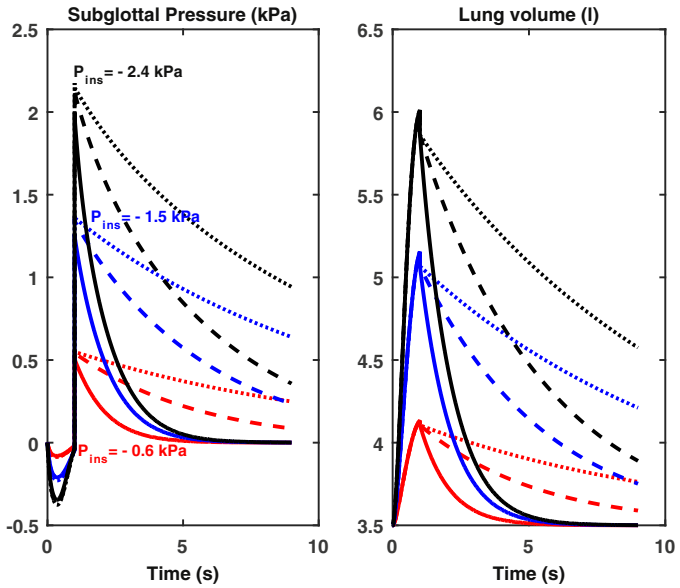

FIGURE 2. The subglottal pressure (left) and lung volume (right) as a function of time following a 0.5-s inspiratory phase. No respiratory muscle pressure is applied in the expiratory phase (P_{exp} = 0 Pa). ——, Rg = 1 Pa·s/mL; – – –, Rg = 4 Pa·s/mL; - - -, Rg = 9 Pa·s/mL. Curves in red, blue, and black colors indicate conditions with a P_{ins} of –0.6 kPa, –1.5 kPa, and –2.4 kPa, respectively.

for 2.5 seconds, beyond which the required expiratory muscle pressure increases abruptly. To maintain the target subglottal pressure longer, either the glottal resistance has to be increased or a deep inspiration is required (at the cost of a higher inspiration muscle pressure).

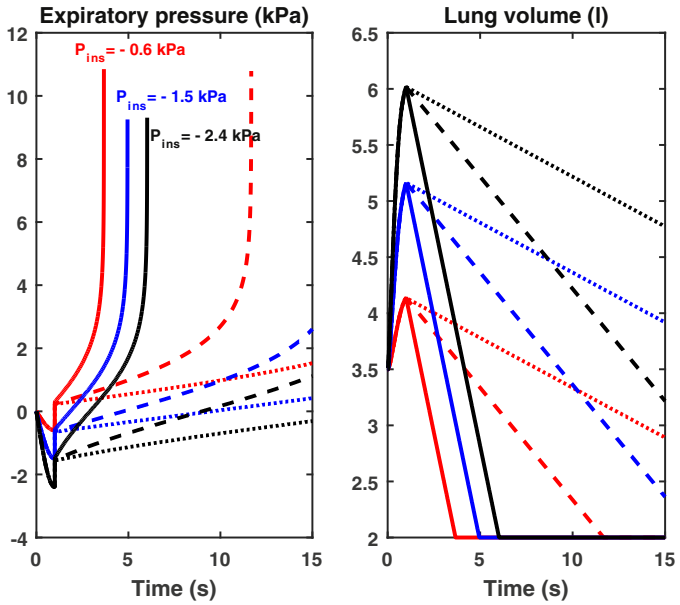

FIGURE 3. The expiratory muscle pressure required to maintain a target subglottal pressure (left) and the corresponding lung volume (right) as a function of time following a 0.5-s inspiratory phase. ——, Rg = 1 Pa·s/mL; – – –, Rg = 4 Pa·s/mL; - - -, Rg = 9 Pa·s/mL. Curves in red, blue, and black colors indicate conditions with a P_{ins} of –0.6 kPa, –1.5 kPa, and –2.4 kPa, respectively.

Normal speech

In conversational speech, the average breath group duration is about 4 seconds.[11] Figure 4 shows the peak expiratory muscular pressure required to maintain a given target subglottal pressure for 4 seconds and the termination lung volume (LVT) at the end of this 4-second period (quantified as a percentage of the VC), as a function of the glottal resistance and the target subglottal pressure. Three conditions of different inspiratory efforts (with a peak inspiratory muscle pressure of –0.6, –1.5, and –2.4 kPa) are considered, which correspond to an initial expiratory lung volume of about 40%, 60%, and 80% of the VC. Figure 4 shows that for a given target subglottal pressure, there is a minimum glottal resistance (about 0.5–3 Pa·s/mL) that is required to maintain the target subglottal pressure for 4 seconds. This minimum glottal resistance can be as small as 0.5 Pa·s/mL and increases with increasing target subglottal pressure and decreasing initial lung volume. In the following discussions, this minimum glottal resistance as a function of the target subglottal pressure is referred to as the airflow constraint.

It is known that to initiate and sustain phonation, the subglottal pressure has to exceed a threshold pressure or the phonation threshold pressure. To understand if airflow conservation imposes a stricter constraint than the phonation threshold pressure, Figure 4 also shows the phonation threshold pressure as a function of the glottal resistance, which is obtained from the numerical simulations in Zhang[8] using a three-dimensional continuum model of the vocal folds. Because the phonation threshold pressure depends on the geometry and material properties of the vocal folds and the glottal gap, the phonation threshold pressure–glottal resistance curve is expected to vary across different voice conditions and subjects. The curve shown in Figure 4 only serves to illustrate a typical relation between the phonation threshold pressure and the glottal resistance, which we consider sufficient for the qualitative discussion below.

The typical range of the subglottal pressure in normal speech is between 200 and 800 Pa. In this range, Figures 4 and 5A show that the phonation threshold pressure imposes a stricter constraint on the glottal resistance than that required by the airflow constraint. Thus, airflow conservation does not seem to be a concern for normal speech production. Indeed, the glottal resistance during normal speech production often falls in the range of 2–8 Pa·s/mL (eg, Holmberg et al[3]), which is much higher than the minimum glottal resistance required by airflow conservation for subglottal pressure between 200 and 800 Pa.

Figure 4 also shows that although the target subglottal pressure can be maintained for 4 seconds with this minimum glottal resistance, it results in an LVT very close to the RV and thus results in a very high expiratory muscular pressure. Increasing glottal resistance allows the 4 seconds to finish at a higher LVT and reduces the overall expiratory muscular pressure. Alternatively, a lower expiratory muscular pressure and a higher LVT can also be achieved by taking a deeper breath in the inspiratory phase, but again at the cost of a higher inspiratory muscle pressure.

Human speech is often considered "effortless" in the sense that speech often does not require noticeable effort in the respiratory or laryngeal systems. Certainly, "effortless" is highly

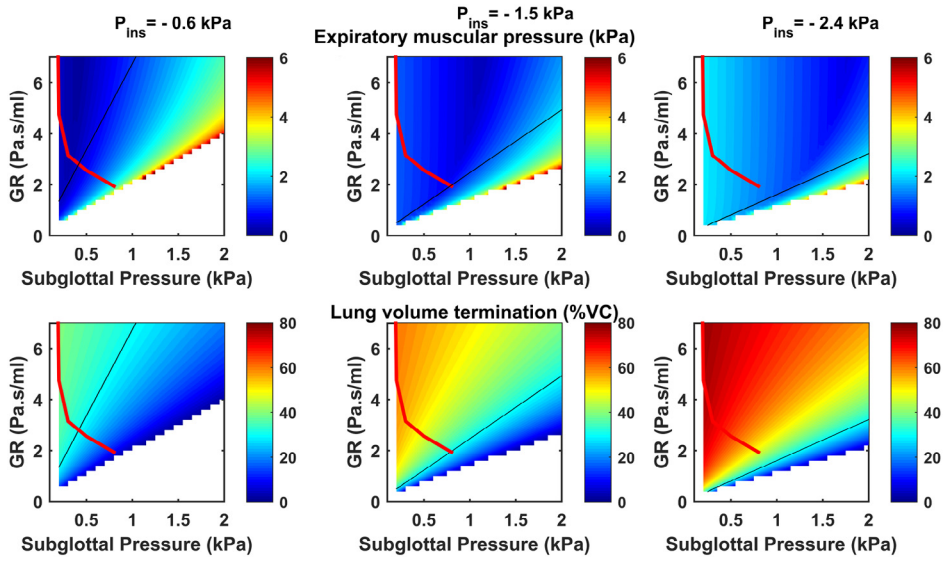

FIGURE 4. Normal speech conditions. The peak expiratory muscular pressure required to maintain a target subglottal pressure for a breath group duration of 4 seconds (top row) and the termination lung volume (LVT) at the end of the 4-second period as a percentage of the VC (bottom row), as a function of the glottal resistance (GR) and the target subglottal pressure. Three inspiratory muscular pressures are considered: P_{ins} = –0.6 kPa (left), –1.5 kPa (middle), and –2.4 kPa (right). The red thick lines indicate phonation threshold pressure, and the thin lines indicate conditions with LVT = 30%VC.

subjective and difficult to quantify. However, Hoit and Hixon[9] have shown that a breath group in speech often ends with an LVT at around 30% of the VC. This is understandable because at a very low LVT, the required expiratory muscle pressure increases, whereas the physiological maximum expiratory muscle decreases (Lausted et al)[12]; thus, one has to work extra hard to maintain the target subglottal pressure. If we impose the 4-s breath group to end at an LVT at 30% of the VC (Figure 5A), this will require a higher glottal resistance. However, the required glottal resistances are still lower than that required by the phonation threshold pressure constraint, except for a target subglottal pressure of about >800 Pa.

In summary, the conservation of airflow and respiratory effort is generally not a concern in normal conversational speech unless for very loud voice production, in which case the respiratory effort can be reduced by either increasing the glottal resistance or taking a deeper breath in the inspiratory phase.

Singing or prolonged phonation

Figure 6 shows the results similar to Figure 4, but for a breath group duration of 6.5 seconds, which has been reported to be the average breath group duration in classical singing.[13] It is very likely that singing may involve even longer breath group durations. Singing generally also requires a much higher subglottal pressure. A subglottal pressure of up to 1500 or 2000 Pa is common. In these conditions, airflow conservation imposes a stricter constraint on the glottal resistance than the requirement for initiating phonation, particularly if one wants to avoid an LVT close to the RV. As a result, an increase in the subglottal pressure has to be accompanied by a simultaneous increase in the glottal resistance to conserve airflow. Thus, one of the goals of singing training is to improve the singer's ability to maintain glottal closure against high subglottal pressures. Indeed, Sabol et al[5] showed that a significant result of singing training is that singers are capable of producing the same sound pressure level using less airflow after training.

Laryngeal–respiratory compensation in pathological conditions

In the conditions of weakened pulmonary function, the requirement for the glottal resistance may increase to raise LVT and avoid excessively high respiratory effort. Figure 7 shows a condition with reduced lung compliance, as for example in pulmonary fibrosis. Due to the reduced lung compliance (0.0003 L/Pa), the relaxation pressure is small only for a reduced range of the lung volume close to the FRC (Figure 1B). This leads to increased expiratory effort if the same range of lung volume is used for phonation. For example, for conditions ending at an LVT of 10% of the VC, the maximum required expiratory muscle pressure is as high as 8 kPa (compared with 2 kPa in Figure 4), close to the physiological limit at this lung volume. The reduced lung compliance also indicates that a much higher inspiratory effort is required to start expiration at a higher initial lung volume. Comparing Figures 4 and 7 shows that the same increase in the inspiratory muscle pressure causes a noticeable increase in the LVT for conditions of very high glottal resistance (in which case, LVT is similar to the initial lung volume) in Figure 4, but the change was much reduced in Figure 7. In this case, to avoid excessively high expiratory effort, one can only compensate with increased laryngeal effort to increase the glottal resistance (Figure 5C).

Similarly, in cases of vocal fold paresis in which one is unable to maintain sufficient glottal closure, the high phonation threshold pressure will lead to significantly increased airflow consumption. In this case, the only compensation option is to increase inspiratory effort to start speech at a high initial lung volume.

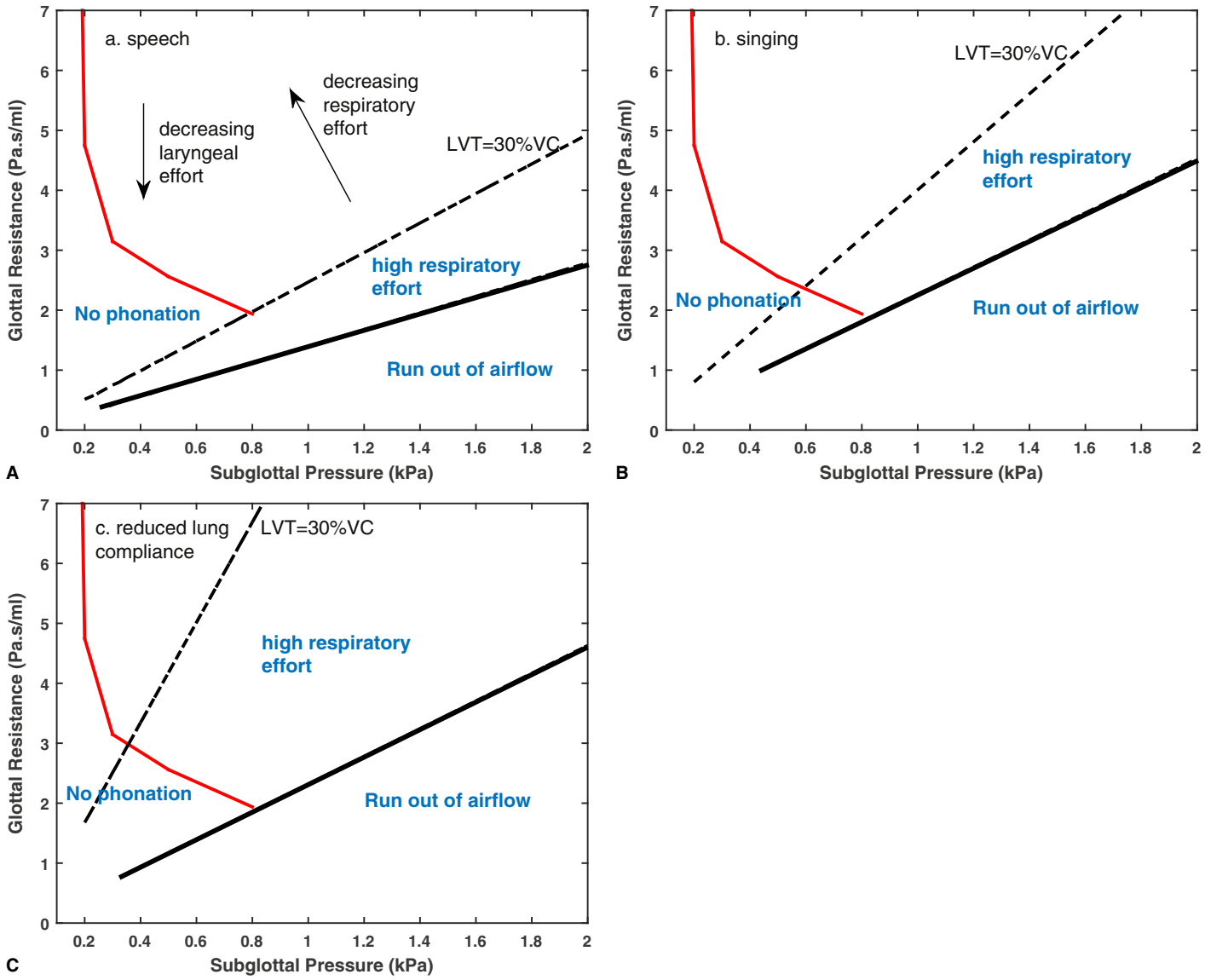

FIGURE 5. Constraints due to phonation initiation (red line), conservation of airflow (thick line) and respiratory effort (dashed line for "effortless" speech) in the glottal resistance (GR)–target subglottal pressure (Ps) space. These constraints define different regimes in the GR–Ps space. Phonation is not possible in regions to the left of the phonation initiation line. For regions below the airflow constraint, one would run out of airflow and would not be able to finish the desired breath group duration. In regions between the thick line and the dashed line, phonation is possible for the desired breath group duration but the respiratory effort is likely high. (**A**) Normal speech; (**B**) prolonged speech or singing; (**C**) speech with reduced lung compliance. LVT, termination lung volume; VC, lung vital capacity.

DISCUSSIONS

As the lung volume approaches the RV, the physiological maximum expiratory muscle pressure decreases rapidly, whereas the expiratory muscle pressure required to maintain a given target subglottal pressure increases rapidly. Thus, in order to maintain a low expiratory effort, it is necessary to maintain an LVT well above the RV. This study shows that as one increases the subglottal pressure to increase vocal intensity, an LVT well above the RV can be maintained by either increasing the glottal resistance, which reduces glottal airflow or the rate of lung volume decrease, or increasing the initial lung volume (thus increasing the supply of air). Whereas the former requires increased laryngeal effort, the latter comes at the cost of increased inspiratory effort. Increasing the glottal resistance is advantageous because for a given breath group duration, it reduces airflow and thus allows phonation to be sustained within a narrow range of lung volumes, thus lowering the overall respiratory effort. In normal speech, a coordinated and balanced respiratory–laryngeal effort is expected. When weakness in one system occurs due to either pathological conditions or aging, it will likely be compensated by hyperfunction of the other system.

The original goal of this study is to evaluate the need for airflow conservation during speech production. This study shows that for breath group durations and subglottal pressures typical of normal conversational speech, airflow conservation or the maintenance of "effortless" respiratory support does not provide a

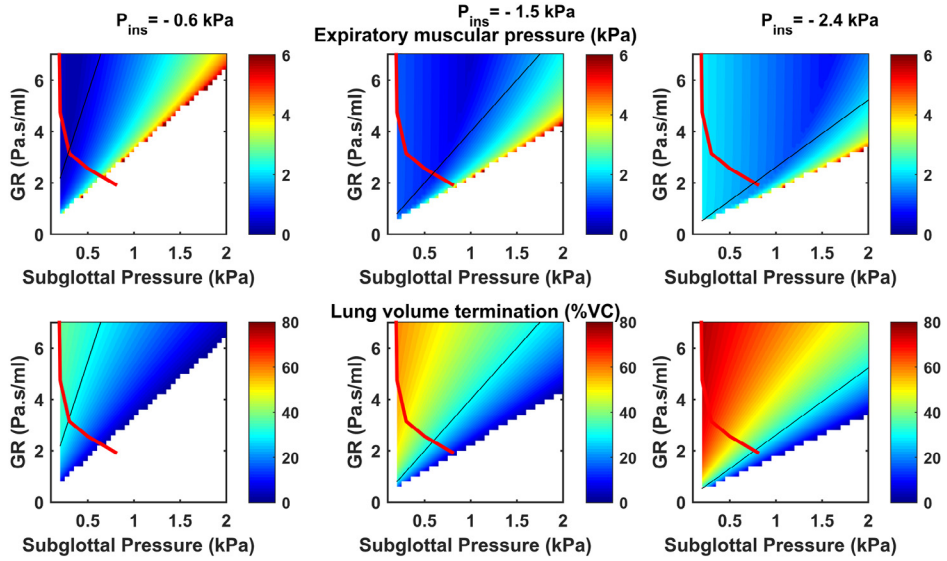

FIGURE 6. Prolonged speech or singing. The peak expiratory muscular pressure required to maintain a target subglottal pressure for a breath group duration of 6.5 seconds (top row) and the termination lung volume (LVT) at the end of the 6.5-second period as a percentage of the VC (bottom row), as a function of the glottal resistance (GR) and the target subglottal pressure. Three inspiratory muscular pressures are considered: P_{ins} = –0.6 kPa (left), –1.5 kPa (middle), and –2.4 kPa (right). The red thick lines indicate phonation threshold pressure, and the thin lines indicate conditions with LVT = 30%VC.

stricter requirement on the glottal resistance than that required for initiating phonation, except for very high subglottal pressures (800 Pa in this study). 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. In those conditions, the glottal resistance is expected to increase proportionally with increased subglottal pressure to conserve airflow consumption and reduce respiratory effort.

In humans, the glottal resistance in normal speech conditions varies in about 2–8 Pa·s/mL, which is higher than that required by either airflow conservation or phonation initiation. This indicates that the laryngeal adjustments accompanying vocal intensity increase are activated for reasons other than conservation

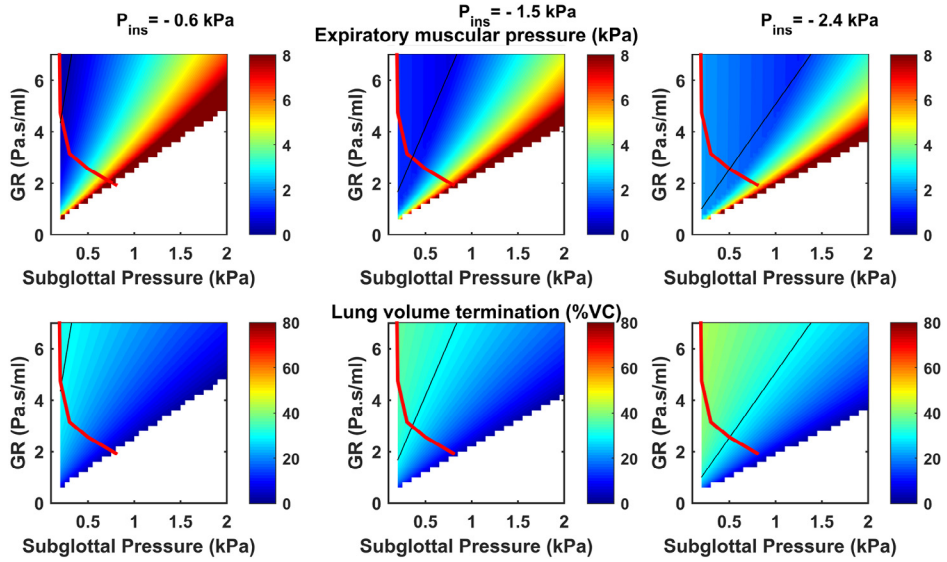

FIGURE 7. Speech under reduced lung compliance. The peak expiratory muscular pressure required to maintain a target subglottal pressure for a breath group duration of 4 seconds (top row) and the termination lung volume (LVT) at the end of the 4 seconds as a percentage of the VC (bottom row), as a function of the glottal resistance (GR) and the target subglottal pressure. Three inspiratory muscular pressures are considered: P_{ins} = –0.6 kPa (left), –1.5 kPa (middle), and –2.4 kPa (right). The red thick lines indicate phonation threshold pressure, and the thin lines indicate conditions with LVT = 30%VC.

of airflow and respiratory effort or phonation initiation. It is likely that the laryngeal adjustments, which increase the glottal resistance, are activated to facilitate the production of high-order harmonics and reduce noise production, both of which may contribute to a perceived increase in vocal intensity.

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