# The Impact of Glottal Configuration on Speech Breathing

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**Summary: Objective.** The purpose of this study was to examine whether changes in respiratory patterns occurred in response to volitional changes in glottal configuration.

**Methods.** Twelve vocally healthy participants read a passage while wearing the Inductotrace respiratory inductive plethysmograph, which measures the excursions of the rib cage and abdomen. Participants read the passage 5 times in a typical speaking voice (*baseline* phase), 10 times in an experimental voice, which was similar to a breathy vocal quality (*experimental* phase), and 5 times again in a typical speaking voice (*return* phase). Kinematic estimates of lung volume (LV) initiation, LV termination, and LV excursion were collected for each speech breath.

**Results.** Participants spoke with larger LV excursions during the *experimental* phase, characterized by increased LV initiation and decreased LV termination compared with the *baseline* phase.

**Conclusion.** In response to volitional changes in glottal configuration, healthy individuals spoke with increased LV excursion. They both responded to changes (decreasing LV termination) and planned for more efficient future utterances (increasing LV initiation) during the *experimental* phase. This study demonstrated that respiratory patterns change in response to changes in glottal configuration; future work will examine these patterns in individuals with voice disorders.

**Key Words:** Voice–Respiratory–Glottal insufficiency–Voice disorder–Functional vocal changes.

## INTRODUCTION

There are well-documented interactions between the laryngeal and respiratory systems during speech breathing. In vocally healthy speakers, changes in the respiratory system have been shown to result in changes in the laryngeal system. Speech produced at high lung volumes (LVs) has been associated with longer voice onset times,[1] increased subglottal pressure,[2] increased sound pressure level,[3,4] increased fundamental frequency,[3,4] and increased glottal leakage.[2] In contrast, speech produced at low LVs has been associated with a more adducted vocal state compared with speech produced at high LVs.[5] Using whispered speech rather than phonation, some studies have also examined the converse relationship in vocally healthy speakers, ie, the effect of an altered laryngeal system on the respiratory system.[6,7] Compared with phonation, speech breathing during whispered productions was characterized by increased air expended per syllable[6,7] and terminating breath groups at low LVs.[6]

Individuals with voice disorders and/or dysphonia who have differences in their glottal configuration due to structural or functional differences in their laryngeal system provide a unique opportunity to examine the respiratory system. Previous research examining respiratory kinematic measures have reported the LV at speech initiation (henceforth LV initiation), the LV at speech termination (henceforth LV termination), and the total volume of air expelled during a speech breath (henceforth LV excursion) in individuals with structural differences (ie, vocal lesions) have demonstrated different, although sometimes contradictory, patterns compared with vocally healthy individuals. Individuals with vocal lesions may present with a breathy vocal quality as the lesions prevent the vocal folds from fully adducting, resulting in glottal insufficiency during phonation. Previous studies have demonstrated that these individuals speak with both decreased LV initiations and terminations,[8] only decreased LV initiations,[9] only decreased LV terminations,[10] and larger LV excursions, characterized by both increased LV initiations and decreased LV terminations.[11,12] Overall, although there are documented differences in the respiratory patterns of individuals with vocal lesions, these studies were unable to determine whether the observed respiratory changes were due to compensation for the structural changes to the vocal folds (ie, the presence of vocal lesions) or whether the changes in the respiratory patterns were a precipitating factor for later vocal changes.

Respiratory differences have also been noted in individuals with dysphonia but without known structural differences in their vocal folds. Similar to individuals with vocal lesions, individuals with dysphonia may present with a breathy vocal quality; however, there are no structural differences in their vocal folds. Individuals with high voice use who self-report vocal difficulties have been shown to speak with decreased LV terminations[13–15] and decreased LV initiations[13] compared with their counterparts without vocal difficulties. These studies suggest that individuals with dysphonia, without vocal lesions, may also demonstrate respiratory differences when compared with individuals without dysphonia.

In addition to the previously mentioned respiratory kinematic measures, respiratory patterns during speech can also be evaluated by independently examining the movement of the two respiratory subcomponents, the rib cage and the abdomen.[16] Hixon and colleagues[17] proposed that although the total volume change

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is measured by evaluating both subcomponents, the relative contribution of each subcomponent can also be measured. As their relative contribution depends on factors such as body position, speaking task, and individual preferences in breathing patterns,[18,19] examining the subcomponents individually may have additional merit. Individuals with voice disorders have been clinically noted to have inefficient "clavicular" breathing, and successful therapeutic intervention has been shown to change these breathing patterns to more efficient ones (eg, Koufman and Blalock[20]).

Taken together, these studies demonstrate that there are differences in the respiratory patterns of individuals with voice disorders and/or vocal difficulties compared with vocally healthy adults. However, these studies do not indicate whether there is a directionality in the relationship between glottal configuration and respiratory patterns, which is important information needed to improve therapeutic interventions for individuals with voice disorders. Specifically, does the glottal configuration and/or vocal pathology cause disordered respiratory patterns, do the disordered respiratory patterns cause disordered glottal configuration and/or vocal pathology, or is it a combination of both? Therefore, the current study examined one of the above questions: do the respiratory patterns of individuals with healthy voices change following volitional changes to their glottal configuration? In this study, we compared respiratory patterns from vocally healthy adults speaking in a typical speaking voice to respiratory patterns used when producing a voice with altered glottal configuration, resulting in a quality similar to a breathy voice. Participants first produced a typical voice for a period of time, then produced the voice with glottal insufficiency, followed again by use of a typical voice. This experimental paradigm allowed us to examine changes in speech breathing patterns as a function of time, to see whether there were any adaptations or compensations due to the altered glottal configuration assumed during periods of glottal insufficiency. As opposed to a whispered voice, in which there is the absence of full adduction of the vocal folds, examination of a breathy voice has more ecological validity for generalizing information to the phonation patterns of individuals with voice disorders, as it allows the examination of phonation with glottal insufficiency. We hypothesized that respiratory patterns would change as a result of these volitional changes to glottal configuration.

## METHODS

### Participants

Twelve healthy adults (M = 22.9 years, standard deviation [SD] = 3.9 years; 6 female, 6 male) participated in a single experimental session. Participants did not report any prior history of voice, speech, language, hearing, or breathing disorders and were recruited from the Boston University graduate and undergraduate populations. Prior to starting the experimental session, all participants provided relevant background information via a structured interview with the experimenter. Four participants (two female, two male) had more than 3 years of vocal training and/or played a wind instrument after middle-school age. All participants completed written consent, in compliance with the Boston University Institutional Review Board.

### Equipment and signals collected

Acoustic data were recorded in Reaper (Cockos Incorporated, San Francisco, California 2016) throughout the study via a headset microphone (model WH20; Shure, Niles, Illinois), placed 7 cm from the mouth at a 45-degree angle, down and to the right, from the center of the mouth. The microphone signal was preamplified by an RME Quadmic II (RME, Haimhausen, Germany) and sampled at 44,100 Hz with 16-bit resolution using a MOTU ultralite mk3 hybrid (model UltraLite3Hy; MOTU, Cambridge, Massachusetts). Vital capacity was calculated using the Phonatory Aerodynamic System (PAS; KayPentax, Lincoln Park, New Jersey) for eight participants following the experimental setup described below. Participants maximally exhaled after producing a maximum inhale, and the PAS software was used to calculate vital capacity.

Signals from the Inductotrace respiratory inductive plethysmograph system (Ambulatory Monitoring, INC, Ardsley, New York) were acquired using NI-DAQ instrumentation (NI USB-6212; National Instruments, Austin, Texas) at a sampling rate of 10,000 Hz. The Inductotrace (Ambulatory Monitoring, INC.) system measures excursions of the two respiratory subcomponents: rib cage and abdomen. Changes in respiratory excursions were sensed via rib cage and abdomen coils placed on the participant (see Figure 1). Equipment setup was consistent with the manufacturer's recommendations (Ambulatory Monitoring, INC.). Briefly, the rib cage coil was placed below the axilla, and the abdominal coil was placed below the lowest rib.

### General procedures and speech tasks

#### *Calibration*

Calibration of the Inductotrace (Ambulatory Monitoring, INC.) respiratory inductive plethysmograph system for each participant

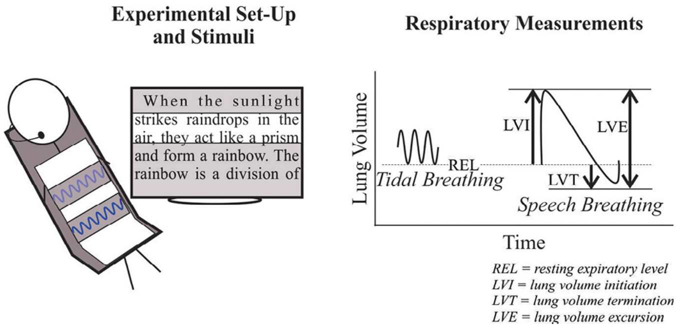

**FIGURE 1.** Schematic of experimental setup and stimuli (*left*): following calibration, participants sat in a chair reclined to 120 degrees. The rib cage coil (light blue) was placed below the axilla, and the abdominal coil was placed below the lowest rib (dark blue). During the experimental task, the rainbow passage was displayed on a screen; a white bar moved down the passage to control reading rate. Schematic of respiratory measurements (*right*): changes in lung volume (LV) are depicted. The resting expiratory level (REL), that is LV termination (LVT) during tidal breathing, is indicated by the horizontal dotted line. LV initiation (LVI) of a speech breath is the maximum inhalation minus REL. LV termination of a speech breath is the minimum LV spoken to minus REL. LV excursion (LVE) is the difference between the maximum and the minimum LV of a single speech breath.

was accomplished by recording the signals from the two coils during breaths in and out of a 0.8-L spirobag while the participants wore a noseclip to prevent nasal air leakage. Calibration was completed in two postures: 1) standing, and 2) sitting on a chair reclined at an angle 30 degrees from upright (ie, 120 degrees reclined), similar to a supine body position. The resulting signals were calibrated using the least squares method (Ambulatory Monitoring, INC.), via a custom MATLAB script,[21] resulting in a correction factor for each coil. Following completion of the experiment, voltage signals from the rib cage and abdomen coils were converted into liters using the previously obtained correction factor.

### Experimental task

After the Inductotrace (Ambulatory Monitoring, INC.) was calibrated, each participant sat on a chair that was reclined at a 120-degree angle for the remainder of the experiment. The angle of the chair positioned the participant in a comfortable, relatively supine posture that he or she was able to maintain for the entire experiment, thereby reducing the opportunity for artifact from unnecessary movement due to discomfort or repositioning, similar to methodologies discussed in previous work.[8] In order to determine a baseline of respiratory kinematics during quiet breathing, LV measures were collected during 1–2 minutes of tidal breathing. Thereafter, each participant performed 20 ordered trials of speech production during which they read the entire "Rainbow Passage,"[22] which was presented to them on a computer screen: 5 trials in his or her typical speaking voice (*baseline* phase), 10 trials in the experimental voice, similar to a breathy vocal quality (*experimental* phase), and then 5 trials in his or her typical speaking voice (*return* phase). A passage was chosen to allow for examination of phrase-length speech in order to capture potential speech breathing changes as a function of time. As all participants were vocally healthy speakers with no aberrant speech breathing patterns, typical speaking voice was defined as the participants' everyday speaking voice. The experimenter present (C.M. or E.H.M.) provided training and instructions to each participant on how to produce a voice with glottal insufficiency prior to the start of the experiment; the participant was instructed not to speak between trials. During each trial, the rate of speech produced was loosely controlled using a bar scrolling through the passage on the computer screen (see Figure 1); it took participants an average of 109.0 seconds (SD = 5.1 seconds) to reach the end of the passage. One potential compensatory method for reading with glottal insufficiency might be to increase speech rate in order to finish the passage quickly; therefore, rate was controlled during this study in order to more effectively examine potential respiratory differences between the use of a typical voice and the use of the experimental voice. All participants were successfully able to produce the experimental voice, as determined auditorily by the experimenter present (C.M. or E.H.M.).

## Data and statistical analysis

All statistical analyses were conducted in Minitab.[23] As there were small variations in the exact time point for completion of reading the passage, for each of the 20 readings of the "Rainbow Passage,"[22] the first 100 seconds of the total 130 seconds were analyzed to avoid analysis of any breathing after the passage, which could be categorically different from speech breathing. This resulted in 20 trials for each participant, each consisting of 100 seconds of the participant reading the entire "Rainbow Passage."[22] For all analysis of variance (ANOVA) calculations, effect sizes were estimated for the factors with a squared partial curvilinear correlation ($\eta_p^2$). In analyses with significant main effects of phase, Tukey *post hoc* analyses were conducted to further assess differences among the phases with a corrected alpha level of 0.05. Cohen's *d* effect sizes were calculated to assess the magnitude of statistically significant differences, designated as either small (0.2–0.3), medium (~0.5), or large (>0.8) effect sizes.[24] All trials were coded by the following phases: *baseline* (trials 1–5), *experimental* (trials 6–15), and *return* (trials 16–20).

### Evaluation of the experimental protocol

*Measurement and variables.* Percent vital capacity (%VC) analysis was conducted for the baseline phase: vital capacity results from the PAS system were obtained from eight participants while they were seated in the experimental chair that was reclined at a 120-degree angle. LV excursion, LV initiation, and LV termination measures during the *baseline* phase were calculated as %VC. This provided information on whether respiratory kinematic measures taken during this experiment were consistent with previous research. Results from LV excursion converted into %VC indicated the average volume of air used during speech breathing compared with the vital capacity of the individual. Results from LV termination and LV initiation conversion into %VC indicated the volume away from the resting expiratory level (REL), expressed in relation to the vital capacity of the individual.

- *LV excursion (%VC):*
$$\frac{\text{Maximum inhalation} - \text{maximum exhalation}}{\textit{Vital capacity}} \times 100$$
- *LV initiation (%VC):* $\dfrac{\text{Maximum inhalation} - \text{REL}}{\textit{Vital capacity}} \times 100$
- *LV termination (%VC):*
$$\frac{\text{Maximum exhalation} - \text{REL}}{\textit{Vital capacity}} \times 100$$

To confirm that participants were effectively changing their vocal quality during the *experimental* phase, smoothed cepstral peak prominence (CPPS) measures were calculated in Praat.[25] CPPS has been previously shown to correlate with the perception of breathiness[26] during readings of the "Rainbow Passage."[22] A Praat script was used to calculate CPPS on the voiced segments in each trial.[27]

*Analysis.* A repeated measures one-way ANOVA examined potential CPPS differences between the three phases (*baseline*, *experimental*, *return*).

### *Respiratory kinematics*

*Measurement and variables.* Respiratory kinematic measures were defined as the following, unless otherwise indicated. The signals from the rib cage and abdomen coils were converted to *liters* via application of the correction factors obtained during the calibration process. The calibrated rib cage and abdomen signals were summed, resulting in a single measure (in liters). A custom MATLAB script was used to identify the maximum inhalation and exhalation for each breath. As participants have individual differences in the volume of their REL (ie, LV termination during tidal breathing), some measures are reported relative to average REL (see Figure 1).

- *LV excursion:* maximum inhalation – maximum exhalation
- *LV initiation:* maximum inhalation – REL
- *LV termination:* maximum exhalation – REL

LV excursion for each subcomponent (LV $\text{excursion}_{\text{sub}}$) was also calculated. Following the calibration of the rib cage and abdomen signals, a custom MATLAB script identified the maximum inhalations and exhalations for each breath in each signal. LV $\text{excursion}_{\text{sub}}$ was calculated separately for each respiratory subcomponent, defined as the maximum excursion minus the minimum excursion.

*Analysis.* Each respiratory kinematic measure of interest (LV excursion, LV initiation, and LV termination) was averaged across each trial. Three repeated measures one-way ANOVAs examined whether there were significant differences in phase (*baseline*, *experimental*, *return*) within each respiratory kinematic measurement: LV excursion, LV initiation, and LV termination. An additional repeated measures two-way ANOVA examined whether LV $\text{excursion}_{\text{sub}}$ differed between the phases (*baseline, experimental, return*) within the two respiratory subcomponents measured (rib cage, abdomen).

### *Breaths taken in each phase*

*Measurement and variables.* For each participant, the number of breaths they took during the first 100 seconds of reading the "Rainbow Passage"[22] was calculated for each of the 20 trials. A breath was defined as an LV initiation peak identified in the summed rib cage and abdomen signal.

*Analysis.* The average number of breaths in each phase was computed for each participant by averaging across trials within each phase. A repeated measures one-way ANOVA examined potential differences in the average number of breaths between the three phases (*baseline*, *experimental*, *return*).

### *Descriptive and correlational analysis*

Descriptive analysis of paradoxical movement, that is, when the derivatives of the two respiratory subcomponents were opposite in sign, was conducted. Correlational analyses examined whether there were any significant linear relationships between the change in 1) number of breaths, 2) CPPS, 3) LV initiation, or 4) LV termination from the *baseline* phase to the *experimental* phase. In addition, although this experiment was not designed to determine the influence of musical experience on any of the variables, our participants were easily divided into two groups: 1) *musical*, defined as having singing and/or wind instrument experience for more than 3 years after middle school (N = 4), and 2) *nonmusical*, defined as having singing and/or wind instrument experience for less than 3 years after middle-school (N = 8). Thus, before collapsing data from these two groups of uneven sizes, we first examined any potential influence of significant musical and/or singing experience by qualitatively examining the change in LV excursion from the *baseline* phase to the *experimental* phase between the two groups. We hypothesized that if there were differences between the two groups, participants in the *musical* group would have a larger LV excursion than participants in the *nonmusical* group. Due to the small and unequal sizes of these two groups, statistical analyses were not conducted; however, summary statistics are reported.

## RESULTS

### Evaluation of the experimental protocol

%VC was calculated for the *baseline* phase for the eight participants who completed the PAS protocol. On average, participants used 12.5 %VC during their speech breaths (range, 7.6–20.5 %VC). Participants initiated their speech at 10.7 %VC above REL (range, 3.7–19.7 %VC above REL) and terminated their speech at 1.8 %VC below REL (range, 8.3 %VC below REL to 4.0 %VC above REL).

To examine whether participants changed their voice quality during the *experimental* phase, CPPS was calculated. A repeated measures one-way ANOVA revealed a significant main effect of phase ($F$ [2,226] = 476.6, $P < 0.01$, effect size $\eta_p^2 = 0.81$). Tukey *post hoc* analyses indicated that CPPS values during the *experimental* phase ($M = 8.8$) were significantly lower than both the *baseline* phase ($M = 13.2$) and the *return* phase ($M = 12.7$; both $p_{adj} < 0.01$). These differences had large effect sizes (Cohen's $d$ values of 2.84 and 2.38, respectively). CPPS values during the *return* phase were significantly lower than the CPPS values during the *baseline* phase with a small effect size ($p_{adj} = 0.024$, Cohen's $d = 0.35$).

### Respiratory kinematics

A repeated measures one-way ANOVA examining LV excursion revealed a significant main effect of phase ($F$ [2,226] = 147.0, $P < 0.01$, effect size $\eta_p^2 = 0.57$). Tukey *post hoc* analyses revealed that LV excursion values during the *experimental* phase ($M = 0.84$ L) were significantly higher than LV excursion values in both the *baseline* phase ($M = 0.54$ L) and the *return* phase ($M = 0.55$ L; both $p_{adj} < 0.01$). These differences had large effect sizes (Cohen's $d$ values of 1.46 and 1.33, respectively). LV excursion during the *return* phase did not significantly differ from LV excursion during the *baseline* phase ($p_{adj} = 0.80$).

A repeated measures one-way ANOVA examining LV initiation revealed a significant main effect of phase ($F$ [2,226] = 7.9, $P < 0.01$, effect size $\eta_p^2 = 0.06$). Tukey *post hoc* analyses indicated that LV initiation values during the *experimental* phase ($M = 0.54$ L above REL) were significantly higher than LV initiation values during the *baseline* phase ($M = 0.46$ above REL; $p_{adj} < 0.01$). This difference had a small effect size (Cohen's *d* value 0.41). LV initiation during the *return* phase ($M = 0.50$ L above REL) did not significantly differ from LV initiation during the *experimental* phase ($p_{adj} = 0.22$) or from LV initiation during the *baseline* phase ($p_{adj} = 0.12$).

A repeated measures one-way ANOVA examining LV termination revealed a significant main effect of phase ($F$ [2,226] = 161.4, $P < 0.01$, effect size $\eta_p^2 = 0.59$). Tukey *post hoc* analyses revealed that LV termination values during the *experimental* phase ($M = 0.32$ L below REL) were significantly lower than LV termination values in both the *baseline* phase ($M = 0.08$ L below REL) and the *return* phase ($M = 0.06$ L below REL; both $p_{adj} < 0.01$). These differences had large effect sizes (Cohen's *d* values of 1.21 and 1.34, respectively). The LV termination during the *return* phase did not significantly differ from the LV termination during the *baseline* phase ($p_{adj} = 0.41$; Figure 2).

A repeated measures two-way ANOVA on the LV excursion of the two respiratory subcomponents (rib cage, abdomen) revealed significant main effects of phase ($F$ [2,463] = 65.3, $P < 0.01$, effect size $\eta_p^2 = 0.22$) and respiratory subcomponent ($F$ [1,462] = 46.2, $P < 0.01$, effect size $\eta_p^2 = 0.11$); however, there was no significant interaction between phase and respiratory subcomponent ($P = 0.56$). Abdominal values were numerically larger than rib cage values in all phases (see Figure 3).

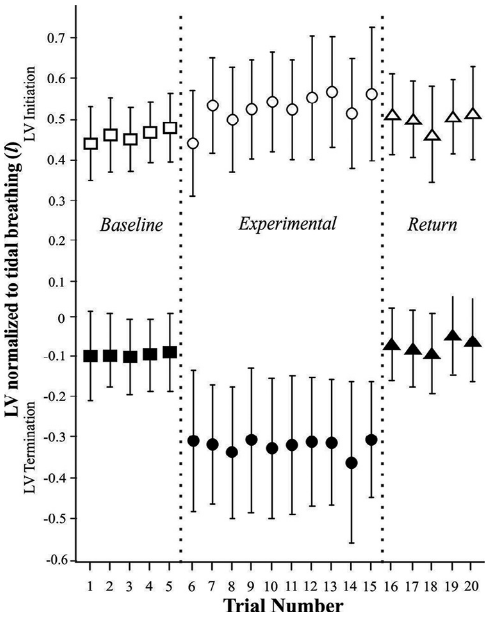

**FIGURE 2.** Average mean and 95% confidence intervals across subjects for lung volume (LV) initiation (open) and LV termination (solid), in the *baseline* (square), *experimental* (circle), and *return* (triangle) phases. Average values are normalized to the resting expiratory level (REL), with a value of 0 indicating REL measured during tidal breathing. LV termination values were significantly lower during the *experimental* phase than both the *baseline* and *return* phases. LV initiation values were significantly higher during the *experimental* phase than the *baseline* phase. LV termination during the *return* phase was not significantly different from that during the *baseline* phase. LV initiation was not significantly different during the *return* phase than either the *experimental* or the *baseline* phases.

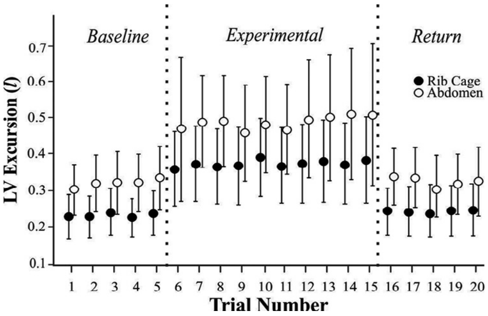

**FIGURE 3.** Lung volume (LV) excursion increased during the *experimental* phase. Average mean and 95% confidence intervals across subjects for LV excursion and the two respiratory subcomponents: rib cage (solid circles) and abdomen (open circles).

### Breaths taken in each phase

A repeated measures one-way ANOVA examining the number of breaths revealed a significant main effect of phase ($F$ [2,226] = 42.7, $P < 0.01$, effect size $\eta_p^2 = 0.27$). Tukey *post hoc* analyses revealed that the number of breaths was significantly higher during the *experimental* phase ($M = 30.4$ breaths) than both the *baseline* phase ($M = 25.5$ breaths) and the *return* phase ($M = 26.3$ breaths; both $p_{adj} < 0.01$). These differences had medium effect sizes (Cohen's *d* values of 0.63 and 0.54, respectively). The number of breaths during the *return* phase did not significantly differ from the number of breaths during the *baseline* phase ($p_{adj} = 0.64$).

### Descriptive and correlational analysis

The breathing of participants was classified as paradoxical an average of 0.76% of the time during the *baseline* phase, 1.16% during the *experimental* phase, and 1.09% during the *return* phase. Correlational analysis revealed there were no significant linear relationships in the change in the number of breaths, CPPS, LV excursion, LV termination, or LV inspiration from the *baseline* to the *experimental* phases (all $P > 0.05$). *Post hoc* analysis of whether participants' musical experience was related to the change in LV excursion (in liters) was examined. LV excursion changes from the *baseline* phase to the *experimental* phase indicated that the musical group (M = 0.2 L, SD = 0.2 liters) and the non-musical group (M = 0.4 L, SD = 0.1) were not clearly different. Therefore, the two groups were not separated for any of the above analyses.

## DISCUSSION

The current study demonstrated that vocally healthy individuals spoke with larger LV excursions while using a speaking voice produced with glottal insufficiency compared with a typical speaking voice. These larger excursions were primarily characterized by decreased LV terminations, in addition to increased LV initiations. Results from this study suggest that volitional changes in glottal configuration are accompanied by changes in respiratory patterns.

### Comparison with previous literature and evaluation of the experimental protocol

The experimental protocol required participants to maintain a voice produced with glottal insufficiency. An examination of CPPS values (*baseline* phase [M = 13.18], *experimental* phase [M = 8.76], and *return* phase [M = 12.68]) revealed values that were consistent with previous research. For example, one study demonstrated lower CPPS values in breathy voices (M = 10.8), and higher CPPS values (M = 14.4) in nonbreathy voices.[28] During the entirety of the experimental task, participants were required to remain in a relatively supine position to prevent additional movement and potential artifact. Due to the 120-degree angle of the chair, LVs derived from abdomen volumes during LV excursion$_{sub}$ were overall larger in all phases than the rib cage LV excursion$_{sub}$, which is consistent with a supine body posture.[18,19] However, this body position did not appear to hinder participants from producing typical speech breathing patterns in the current study. During the *baseline* phase, participants had an average LV excursion of 12.5 %VC. This is consistent with previous literature examining speech breathing during reading, showing averages of 10–23 %VC during speech breathing.[12,29] During these reading tasks examined in previous research, individuals initiated speech at 6–28 %VC relative to REL and terminated their speech at 8 %VC above REL to 11 %VC below REL. These values are consistent with the current study, which indicated average LV initiation values of 10.7 %VC above REL and average LV termination values of 1.8 %VC below REL. Overall, this suggests that the experimental setup of a relatively supine body posture to control additional movement did not prohibit individuals from achieving typical vital capacity and respiratory kinematic measures in the current protocol.

### Compensation for glottal insufficiency

During volitional production of the experimental speaking voice, we inferred that participants in the current study assumed a glottal configuration that resulted in glottal insufficiency. Therefore, the differences in breathing patterns seen in this study may be attributed to an attempt to compensate for this insufficiency. Participants increased the number of breaths they used, which has been proposed as a method of combatting glottal insufficiency.[10] An increase in the number of breaths was also noted in a previous study in a group of individuals with vocal nodules.[9] Authors cited the increase in number of breaths as the reason they did not observe the expected changes in LV terminations.[9] Although participants in the current study did increase the number of breaths they took, it may not have been enough to combat a decrease in LV terminations. One reason participants may not have increased the number of breaths to a larger degree is related to the linguistic context of the task in which they read the entire the “Rainbow Passage.”[22] Previous work has suggested that healthy adults will change the location of their inspiratory pauses only when there is a physiological need to pause.[30] Therefore, participants in the current study may not have wanted to further increase the number of breaths, as it may have substantially changed the linguistic content of the passage. Additionally, this experiment controlled reading rate in order to have comparable speaking rates between the different phases. Therefore, this control may have further impacted the number of breaths participants could take throughout the passage due to the necessity to maintain a consistent pace.

Another potential method of compensating for glottal insufficiency is to increase LV excursions either by increasing LV initiations or decreasing LV terminations. Participants in this study spoke using an increased LV initiation during the *experimental* phase compared with the *baseline* phase, taking advantage of the higher recoil pressures present at high LVs[15] as well as a larger inspiratory reserve. In addition, some evidence (eg, Hoit et al,[1] Iwarsson et al,[2] and Milstein[5]) suggests that speaking at higher LVs results in an abducted vocal state, potentially due to tracheal pull on the larynx.[31] This abducted vocal state would be beneficial for the current study design and may have helped participates produce the *experimental* voice. In addition to using an increased LV initiation, participants spoke to a lower LV termination during the *experimental* phase compared with both the *baseline* and *return* phases. Participants may have noted that, due to the self-perturbation of their glottal configuration (ie, glottal insufficiency), continued use of their previous speech breathing patterns would result in a deviation from the linguistic structure of the passage. Therefore, participants may have attempted to compensate by speaking with a lower LV before initiating the subsequent breath.

### Contribution of respiratory subcomponents

LV excursion of the entire respiratory system was shown to increase during the *experimental* phase; however, further analysis of the respiratory subcomponents revealed that there was no significant interaction between phase and respiratory subcomponent. The lack of a statistically significant interaction between respiratory subcomponent and phase demonstrated that the participants did not rely on either their rib cage or abdominal movement to change their breathing patterns during the *experimental* phase; rather, they changed both respiratory subcomponents relatively uniformly. Additionally, examination of paradoxical breathing revealed that participants used paradoxical breathing strategies for a very small percentage of time, with little variation as a function of phase. This small percentage of paradoxical breathing is consistent with previous work that indicates that utilization of paradoxical breathing in healthy individuals is not anomalous.[17,18]

Individuals with voice disorders have noted inefficient breathing strategies, often categorized as an overreliance on rib cage movement during breathing, resulting in increased breathiness and lower vocal intensity (eg, Stemple et al[32]). However, results from the current study do not show a relationship between producing the experimental voice and overreliance on rib cage movement. Further investigation is necessary to examine whether individuals with voice disorders have a similar lack of interaction between respiratory subcomponents during tasks in which they volitionally change glottal configuration.

### Adaptation of respiratory kinematics

The current study examined changes in speech breathing patterns after self-perturbation (eg, glottal insufficiency) was applied during the *experimental* phase. This is a similar paradigm to speech motor control studies that show compensation for changes in voice and articulatory features of auditory feedback within a single experimental session. Vocally healthy adults will compensate quickly to changes in voice[33,34] and articulation[35] in an attempt to correct discrepancies between their heard auditory feedback and expected auditory feedback.[36–38] The differences in LV termination noted in the current study, beginning at the first trial of the *experimental* phase, may be indicative of a similar rapid response, resulting in changes in LV termination in an attempt to compensate for unexpected discrepancies. That is, if participants had continued to use their previous speech breathing patterns during the *experimental* phase, it may have led to the production of a passage with an atypical linguistic structure. To rectify this discrepancy, participants may have compensated by speaking to a lower LV termination before taking the next breath. When the self-perturbation was removed during the *return* phase, participants immediately returned to their typical LV termination patterns. This suggests the LV termination changes can occur in response to a self-perturbation; however, as soon as the self-perturbation is removed, participants return to their previous speech breathing patterns.

Although LV termination changes can occur immediately in response to a self-perturbation, changes in LV initiation prior to an utterance require additional planning of the respiratory movements before initiating the speech production. In order to have effective and smooth speech breathing, individuals may need a stored motor plan for their LV initiation target based on what they are planning to produce. This would allow people to, relatively seamlessly, inhale to an appropriate volume before speaking, similar to how stored motor programs allow the generation of fluid motor movements in speech.[36–38] Therefore, participants may have noted the self-perturbation in the first trial of the *experimental* phase and compensated by decreasing their LV termination while also using this information to update their stored motor programs for LV initiation. This would result in a change in their LV initiation targets for subsequent trials. Examination of the *return* phase after the self-perturbation was removed during LV initiation also showed similarities to previous studies. In the current study, participants did not completely return to baseline as they did with LV termination. Specifically, although there were no significant differences between the *return* and *baseline* phases, there were also no significant differences between the *return* and *experimental* phases. The general shape of the average LV initiation across trial (see Figure 2) indicates that the increase noted during the *experimental* phase was relatively stable throughout the phase, ie, not continuing to rise in subsequent trials. This is similar to previous work in speech and voice showing that once perturbation is removed, there is no immediate return to baseline as the stored motor targets need to be updated again after the removal of the perturbation.[39–41]

Although shown here in the respiratory motor domain, this idea is consistent with previous work suggesting that individuals will adapt their voice[39] and articulation[40,41] in response to sustained auditory perturbation (ie, adaptive response), proposed to demonstrate the ability of the feedback system to update the feedforward system. This similarity to previous work suggests that the speech breathing control in the respiratory motor domain may have similar feedback and feedforward regulation to the voice and speech motor domains. However, it is relevant to note that in these previous experiments, consistent perturbation was surreptitiously applied over multiple repetitions of single words, with adaptation occurring just a few minutes following the onset of perturbation (eg, Villacorta et al[40]; Purcell and Munhall[41]). This differs from the current study, in which the participants were in control of the perturbation (ie, self-perturbation). Additionally, the current study involved changes in both the auditory and somatosensory systems, as participants could both hear and feel the changes in their glottal configuration. This additional sensory information may have affected the manner and magnitude to which participants responded to the changes in their speaking voices.

## CONCLUSIONS

Results from this study indicated that when healthy individuals volitionally spoke with increased glottal insufficiency, they used increased LV excursions, characterized by increased LV initiation and decreased LV termination. Participants also spoke with an increased number of breaths during the *experimental* phase of the paradigm, potentially as an attempt to compensate for the glottal insufficiency. These data suggest that individuals can control their respiratory system to respond immediately to self-perturbation by decreasing their LV termination as well as update their stored motor programs for LV initiation to make future productions more efficient. Future studies are necessary to extend this work to individuals with voice disorders.

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## REFERENCES

1. Hoit JD, Solomon NP, Hixon TJ. Effect of lung volume on voice onset time (VOT). *J Speech Lang Hear Res*. 1993;516–520.
2. Iwarsson J, Thomasson M, Sundberg J. Effects of lung volume on the glottal voice source. *J Voice*. 1998;424–433.
3. Dromey C, Ramig LO. The effect of lung volume on selected phonatory and articulatory variables. *J Speech Lang Hear Res*. 1998;491–502.
4. Watson PJ, Ciccia AH, Weismer G. The relation of lung volume initiation to selected acoustic properties of speech. *J Acoust Soc Am*. 2003;2812–2819.
5. Milstein CF. *Laryngeal function associated with changes in lung volume during voice and speech production in normal speaking women*. (Ph.D.), University of Arizona, Tucson, Arizona, 1999.
6. Stathopoulos ET, Hoit JD, Hixon TJ, et al. Respiratory and laryngeal function during whispering. *J Speech Lang Hear Res*. 1991;761–767.
7. Binazzi B, Lanini B, Bianchi R, et al. Breathing pattern and kinematics in normal subjects during speech, singing and loud whispering. *Acta Physiol*. 2006;233–246.
8. Sperry E, Hillman R, Perkell J. The use of inductance plethysmography to assess respiratory function in a patient with vocal nodules. *J Med Speech Lang Pathol*. 1994;137–145.
9. Iwarsson J, Sundberg J. Breathing behaviors during speech in healthy females and patients with vocal fold nodules. *Logoped Phoniatr Vocol*. 1999;154–169.
10. Schaeffer N, Cavallo SA, Wall M, et al. Speech breathing behavior in normal and moderately to severely dysphonic subjects during connected speech. *J Med Speech Lang Pathol*. 2002;1–19.

11. Sapienza CM, Stathopoulos ET. Respiratory and laryngeal measures of children and women with bilateral vocal fold nodules. *J Speech Lang Hear Res*. 1994;1229–1243.
12. Sapienza CM, Stathopoulos ET, Brown W. Speech breathing during reading in women with vocal nodules. *J Voice*. 1997;195–201.
13. Lowell SY, Barkmeier-Kraemer JM, Hoit JD, et al. Respiratory and laryngeal function during spontaneous speaking in teachers with voice disorders. *J Speech Lang Hear Res*. 2008;333–349.
14. Reich A, McHenry M. Respiratory volumes in cheerleaders with a history of dysphonic episodes. *Folia Phoniatr Logop*. 1987;71–77.
15. Hixon TJ, Putnam AH. Voice abnormalities in relation to respiratory kinematics. In: Hixon TJ, ed. *Respiratory Function in Speech and Song*. San Diego, California: College-Hill; 1987:259–280.
16. Konno K, Mead J. Measurement of the separate volume changes of rib cage and abdomen during breathing. *J Appl Physiol*. 1967;407–422.
17. Hixon TJ, Mead J, Goldman MD. Dynamics of the chest wall during speech production: function of the thorax, rib cage, diaphragm, and abdomen. *J Speech Lang Hear Res*. 1976;297–356.
18. Hixon TJ, Goldman MD, Mead J. Kinematics of the chest wall during speech production: volume displacements of the rib cage, abdomen, and lung. *J Speech Lang Hear Res*. 1973;78–115.
19. Hoit JD. Influence of body position on breathing and its implications for the evaluation and treatment of speech and voice disorders. *J Voice*. 1995;341–347.
20. Koufman JA, Blalock PD. Vocal fatigue and dysphonia in the professional voice user: Bogart-Bacall syndrome. *Laryngoscope*. 1988;493–498.
21. Matlab. *Matlab R2013a (Version 8.1.0.604)*. Natick, MA: The Mathworks, Inc.; 2013.
22. Fairbanks G. The rainbow passage. *Voice and Articulation Drillbook*. 2nd ed. New York, NY: Harper & Row New York; 1960.
23. Minitab. *Minitab Statistical Software (Version 16.2.3)*. State College, PA: Minitab Inc.; 2012.
24. Witte RS, Witte JS. *Statistics*. 9th ed. Hoboken, NJ: Wiley publishing; 2010.
25. Boersma P, Weenink D. Praat: doing phonetic by computer (Version 5.3.82). 2014. Available at: http://www.praat.org/. Accessed January 1, 2014.
26. Hillenbrand J, Houde RA. Acoustic correlates of breathy vocal quality: dysphonic voices and continuous speech. *J Speech Lang Hear Res*. 1996;311–321.
27. Maryn Y, Weenink D. Objective dysphonia measures in the program Praat: smoothed cepstral peak prominence and acoustic voice quality index. *J Voice*. 2015;35–43.
28. Barsties V Latoszek B, Maryn Y, Gerrits E, et al. The Acoustic Breathiness Index (ABI): a multivariate acoustic model for breathiness. *J Voice*. 2017;511.e11–511.e27.
29. Winkworth AL, Davis PJ, Ellis E, et al. Variability and consistency in speech breathing during reading: lung volumes, speech intensity, and linguistic factors. *J Speech Lang Hear Res*. 1994;535–556.
30. Grosjean F, Collins M. Breathing, pausing and reading. *Phonetica*. 1979;98–114.
31. Zenker W. Questions regarding the function of external laryngeal muscles. In: Brewer D, ed. *Research Potentials in Voice Physiology*. New York: State University of New York; 1964:20–40.
32. Stemple JC, Glaze LE, Gerdeman BK. *Clinical Voice Pathology: Theory and Management*. 4th ed. San Diego, California: Plural Publishing; 2000.
33. Burnett TA, Senner JE, Larson CR. Voice F0 responses to pitch-shifted auditory feedback: a preliminary study. *J Voice*. 1997;202–211.
34. Burnett TA, Freedland MB, Larson CR, et al. Voice F0 responses to manipulations in pitch feedback. *J Acoust Soc Am*. 1998;3153–3161.
35. Tourville JA, Reilly KJ, Guenther FH. Neural mechanisms underlying auditory feedback control of speech. *Neuroimage*. 2008;1429–1443.
36. Guenther FH. Cortical interactions underlying the production of speech sounds. *J Commun Disord*. 2006;350–365.
37. Guenther FH, Ghosh SS, Tourville JA. Neural modeling and imaging of the cortical interactions underlying syllable production. *Brain Lang*. 2006;280–301.
38. Tourville JA, Guenther FH. The DIVA model: a neural theory of speech acquisition and production. *Lang Cogn Process*. 2011;952–981.
39. Jones JA, Munhall KG. Perceptual calibration of F0 production: evidence from feedback perturbation. *J Acoust Soc Am*. 2000;1246–1251.
40. Villacorta VM, Perkell JS, Guenther FH. Sensorimotor adaptation to feedback perturbations of vowel acoustics and its relation to perception. *J Acoust Soc Am*. 2007;2306–2319.
41. Purcell DW, Munhall KG. Adaptive control of vowel formant frequency: evidence from real-time formant manipulation. *J Acoust Soc Am*. 2006;966–977.