Research Article

Phonation Demonstrates Goal Dependence Under Unique Vocal Intensity and Aerobic Workload Conditions

Aaron Ziegler,^a Jessie VanSwearingen,^b John M. Jakicic,^c and Katherine Verdolini Abbott^d

Purpose: This study investigated whether metabolic respiratory requirements (treadmill workload) affected glottal valving in phonation, based on aerodynamic measures, when a sound pressure level (vocal SPL) is dictated as a target goal. Consistent with a theory of action, we hypothesized that adjustments in glottal valving as measured by laryngeal airway resistance would be dependent upon vocal SPL level, even as workload increased, and loud vocal SPL would interfere more with respiratory homeostasis than spontaneous vocal SPL.

Method: Thirty-two women enrolled who were ages 18–35 years. A repeated-measures design was used with random assignment of workload and vocal SPL conditions. Aerodynamic and acoustic data were collected during phonation, as were gas volume and concentration data. Analyses were performed with generalized estimating equations.

Results: Laryngeal airway resistance at a low workload significantly increased when vocal SPL changed from spontaneous to loud. At a loud vocal SPL, laryngeal airway

resistance decreased when workload changed from rest to either low or high. Regarding the respiratory system response, minute ventilation increased at a loud vocal SPL when workload changed from rest to either low or high. End-tidal CO₂ increased under low and high workloads relative to rest at loud and spontaneous vocal SPLs.

Conclusions: Mostly consistent with a theory of action, in which motor control is goal dependent (i.e., vocal SPL targets), speakers can achieve a loud vocal SPL despite increases in workload requirements. In contrast, laryngeal airway resistance stays relatively low when vocal SPL occurs spontaneously, suggesting glottal adjustments are made to improve gas exchange as metabolic respiratory requirements become prioritized. Metabolic respiratory requirements appear to be overcome by the overlay of motor control for voicing when a loud vocal SPL is targeted. The implication of goal-dependent phonation for clinicians is that real-world conditions (i.e., loud vocal SPL) matter in vocal testing and voice therapy.

The respiratory and laryngeal systems work together during voice production to regulate vocal intensity (vocal sound pressure level [SPL]; Finnegan, Luschei, & Hoffman, 2000; Hixon, Goldman, & Mead, 1973; Hixon, Mead, & Goldman, 1976; Stathopoulos & Sapienza, 1993). The two systems also interact during respiration to control blood gas concentrations and maintain homeostasis

(Brancatisano, Collett, & Engel, 1983; Brancatisano, Dodd, & Engel, 1991; Butler, 2007; England & Bartlett, 1982).

The laryngeal system, specifically the vocal folds, adducts in voice production as the respiratory system increases lung pressure to drive air through the glottis and entrain the vocal folds into self-sustained vibration (Titze, 1988, 2002). On the other hand, the vocal folds abduct to decrease airflow resistance and accommodate greater volumes of exhaled air as ventilation increases to maintain respiratory homeostasis. Thus, opposing laryngeal configurations and different physiologic strategies by the respiratory and laryngeal systems exist when breathing as during phonation—with closed vocal folds—versus breathing for purposes of metabolic homeostasis—with open vocal folds.

It is unknown whether the same physiologic strategies apply during conditions of increased ventilation (high respiratory drive) when the vocal folds abduct for increased ventilation (hyperpnea) while simultaneously approximating

^aDepartment of Otolaryngology-Head & Neck Surgery, Oregon Health & Science University, Portland

^bDepartment of Physical Therapy, University of Pittsburgh, PA

^cDepartment of Health & Physical Activity, University of Pittsburgh, PA

^dUniversity of Delaware, Dover

Correspondence to Aaron Ziegler: wellnessgroupsjp@gmail.com

Editor-in-Chief: Julie Liss

Editor: Jack Jiang

Received April 5, 2018

Revision received October 16, 2018

Accepted March 11, 2019

https://doi.org/10.1044/2019_JSLHR-S-18-0126

Disclosure: The authors have declared that no competing interests existed at the time of publication.

for voice production, for example, in those who provide vocal instruction during aerobic exercise and similar occupations. Specifically, does intentional communication with preferred vocal SPL targets in high-respiratory drive conditions result in vocal fold adjustments that allow for increases in airflow as metabolic needs increase, favoring those needs while sacrificing vocal output (Bailey & Hoit, 2002; Bunn & Mead, 1971; Doust & Patrick, 1981; Hoit, Lansing, & Perona, 2007; Otis & Clark, 1968)? This question is important because of the implications for glottal valving and the risk for vocal injury with high impact stress from tight vocal fold adduction and high subglottal pressures as often occurs with loud vocal SPL (Grillo & Verdolini, 2008; Titze, 2000; Verdolini, Druker, Palmer, & Samawi, 1998). On the other hand, the risk for speaking-related dyspnea is relevant for patients with pulmonary conditions who experience shortness of breath when talking (Lee, Friesen, Lambert, & Loudon, 1998) due to the obstructive effect of approximated vocal folds on respiration with voicing (Russell, Cerny, & Stathopoulos, 1998).

A range of studies have investigated the synergistic relationship between the mechanically coupled respiratory and laryngeal systems during voice production using laryngeal airway resistance (R_{law}). R_{law} is the ratio of subglottal pressure (P_s) to translaryngeal airflow (U) during phonation (Baken & Orlikoff, 2000). A noninvasive technique exists for estimating R_{law} during the production of /pa/ consonant-vowel combinations (Holmberg, Hillman, & Perkell, 1988; Holmberg, Hillman, Perkell, & Gress, 1994; Leeper & Graves, 1984; Smitheran & Hixon, 1981). In broad terms, R_{law} increases as vocal SPL increases in resting breathing conditions (Holmberg et al., 1988; Leeper & Graves, 1984; Wilson & Leeper, 1992). Increases in R_{law} with greater vocal SPL come primarily from higher P_s . Breathier voice quality causes R_{law} to decrease, which is primarily from an increase in U (Holmberg, 1980; Holmberg, Hillman, & Perkell, 1989). Airway resistance provided by the larynx during phonation has an obstructive effect on air expulsion as reflected in significantly higher CO_2 partial pressures in voicing than in silent breathing (Gillespie, Slivka, Atwood, & Verdolini Abbott, 2015). A related measure, phonation threshold pressure (i.e., the smallest subglottal pressure required for self-sustained vocal fold vibration), significantly increases after cycling (Sandage, Connor, & Pascoe, 2013). Likely due to increased respiratory rate with exercise (McArdle, Katch, & Katch, 2010), such changes occur in the setting of a meaningful decrease in pharyngeal temperature that can negatively impact muscle contractive properties of the vocal folds (Sandage et al., 2013). Changes in phonation threshold pressure may also reflect an increase in vocal fold viscosity from vocal fold tissue desiccation as occurs with oral breathing (Sivasankar & Erickson, 2009).

In addition to changes in R_{law} , other voice changes may be relevant to our concerns about vocal motor control under elevated levels of respiratory drive. As compared to normal respiratory drive conditions, voice is perceived as louder, breathier, and higher in pitch under high-respiratory drive conditions (Bailey & Hoit, 2002; Otis & Clark, 1968)

that result in increases in ventilation from resting levels to tightly match the body's metabolic needs (i.e., O_2 and CO_2 partial pressures) to exercise demands (Butler, 2007; McArdle et al., 2010). Researchers have argued that metabolic needs begin to override linguistic demands as the intensity of high respiratory drive increases, as evidenced by changes in speech phrasing (Bailey & Hoit, 2002; Baker, Hipp, & Alessio, 2008; Otis & Clark, 1968) and difficulty with speaking tasks (Rotstein, Meckel, & Inbar, 2004). It has been reported that individuals provide a greater number of statements about increased concentration (i.e., mental effort) to breathe at higher intensities of high respiratory drive (Hoit et al., 2007) that may reflect a vulnerability of behavioral vocal motor control to the needs of automatic respiratory motor control. Taken together, findings to date suggest the voice reacts to—rather than acts in—conditions of high respiratory drive when constraints on vocal output are not in place (e.g., vocal SPL).

Action theory, which provides a framework for understanding possible competition between respiration and phonation, states that physiology of activity depends on functional goals (Reed, 1982). Under different initial conditions (e.g., different treadmill workloads), context-dependent tuning of coordinative structures such as the respiratory-laryngeal system takes place in attainment of an action goal (e.g., specified vocal SPL). The effect of a perturbation to a system (e.g., increased ventilation with exercise) should be negligible in terms of goal accomplishment, while other components adjust (Fowler, Rubin, Remez, & Turvey, 1980). This motor equivalence is possible because of heterarchical neural control that allows for goal attainment under varying, even extreme, initial conditions (e.g., high treadmill workload; Jürgens, 2009).

The intersection of learned phonation mediated by higher cortical pathways and automatic breathing at the medullary reticular formation in the brainstem sets the stage for competition between phonatory and respiratory functions (Blitzer, Brin, & Ramig, 2009). Higher brain centers during special acts of breathing (e.g., voice) transmit signals to the medulla that influence respiratory activity. The integration of information from cortical areas permits an interruption of the normal rhythmic breathing pattern generation. If this dual control system does in fact exist, final integration of neuronal signals would occur at the level of the motor neuron. The timing and strength of the final signal reaching respiratory muscle motor neurons would ultimately depend on brainstem (i.e., medullary) and higher cortical inputs as well as other input via networks of interneurons (Butler, 2007). However, up to this date, research that has addressed voice under conditions of high respiratory drive has only done so under conditions in which the voice was free to vary, paying too little attention to glottal valving under real-world conditions that require loud vocal SPL.

Objective

The objective of this study was to investigate whether metabolic respiratory requirements affect glottal valving in

phonation, based on aerodynamic measures, when a specific vocal SPL is dictated as a target goal and coupled with increased respiratory demands. We hypothesized that adjustments in R_{law} would be dependent upon vocal SPL target, reflecting deference to a phonatory goal (SPL) in a constrained voice condition (i.e., loud vocal SPL), even as workload increased, and would interfere with respiratory homeostasis (minute ventilation [V_e] and end-tidal carbon dioxide [$P_{ET}CO_2$]) through an obstructive effect. Such findings would be consistent with a theory of action (see Figure 1).

Method

Study Design

This research utilized a prospective, repeated-measures experimental design with random assignment of counter-balanced sequences of conditions to minimize order effects. Reporting of this study was consistent with the Strengthening of Reporting of Observational Studies in Epidemiology statement (von Elm et al., 2008).

Setting

Participant recruitment and data collection sessions across all participants occurred over a period of 2 months, and all sessions for a given participant were completed within a 3-week time frame. Screening and preexperimental training occurred at the University of Pittsburgh Voice Center. The subsequent experimental protocol was completed at the University of Pittsburgh Physical Activity and Weight Management Research Center where environmental conditions (i.e., relative humidity and temperature) met recommended standards for fitness facilities (American College of Sports Medicine, 2012). Participants were instructed to maintain normal fluid and dietary intake prior to all sessions and to wear similar clothing suitable for exercise to all sessions. To allow for metabolic recovery, participants returned no less than 24 hr after a previous session.

Participants

Research activities were approved by the University of Pittsburgh Institutional Review Board (IRB# 12030732).

Figure 1. Conceptual schematic of goal-dependent model of motor control for voice. VO_2 = oxygen consumption; SPL = sound pressure level; R_{law} = laryngeal airway resistance; V_e = minute ventilation; $P_{ET}CO_2$ = end-tidal carbon dioxide.

Participants were ages 18–35 years and recruited from the Pittsburgh metropolitan region using institutional review board–approved flyers distributed in physical and electronic formats. Based on power analyses described shortly, 32 women completed the full experimental protocol. A small group of men ($n = 4$) was also included to gather data on potential gender effects (Harms, 2006; Hunter, Tanner, & Smith, 2011; Sheel & Guenette, 2008); their data were explored descriptively only and are presented in Tables 2 and 3. After passing an online screening, participants were invited for an in-clinic screening if determined to have a perceptually normal vocal quality over the telephone by a speech-language pathologist.

Eligibility criteria included the following: (a) by self-report, recreationally active (90–150 min per week in aerobic exercise or sports activity); negative history of voice problems, voice treatment, or more than 1 year of voice training with the exception of high school choir; negative history of smoking in the past 5 years; not currently pregnant or within the last 6 months (confirmed by negative result on rapid urine test conducted at the screening visit); negative history of medication use during the month prior to participation, with the exception of medications that regulate menstrual cycle; regular monthly menstruation as of the most recent cycle or amenorrhea due to menstrual-regulating medications at the time of data collection; and no contraindications to participate in exercise as outlined by the American College of Sports Medicine (2009); (b) by clinician judgment, sufficient English comprehension to provide consent and follow instructions, ability to match and sustain a pitch (men: C_3 [$130\text{ Hz} \pm 1$ semitone [ST]; female: A_3 [$220\text{ Hz} \pm 1$ ST] for 3 s when given a reference tone, and free of signs (or symptoms, per self-report) of active allergies or upper respiratory infection on days of participation; (c) no indication of voice handicap (score of > 11 on the Voice Handicap Index-10; Arffa, Krishna, Gartner-Schmidt, & Rosen, 2012; Rosen, Lee, Osborne, Zullo, & Murry, 2004); and (d) normal voice as judged by a speech-language pathologist, with an overall severity score ≤ 10 mm on the Consensus Auditory Perceptual Evaluation of Voice (Kempster, Gerratt, Verdolini Abbott, Barkmeier-Kraemer, & Hillman, 2009); normal hearing with pure-tone audiometry conducted by a speech-language pathologist using a Maico Diagnostics Audiometer at 30 dB at 250 and 500 Hz as well as 1, 2, 4, and 8 kHz in the better ear; normal larynx as judged by a fellowship-trained laryngologist during flexible laryngeal endoscopy with stroboscopy after administration of both lidocaine and neosynephrine; normal body mass index ($18.5\text{--}24.9\text{ kg/m}^2$ [National Heart, Lung and Blood Institute, Obesity Education Initiative, Expert Panel, 1998] $\pm 1.0\text{ kg/m}^2$); normal resting blood pressure ($90/60\text{--}120/80\text{ mmHg}$; Chobanian et al., 2003); normal resting heart rate ($50\text{--}80$ beats per minute [bpm]; American College of Sports Medicine, 2009); and normal pulmonary function (forced vital capacity and forced expiratory volume in 1 s [FVC and FEV1, respectively] values at 80% or above predicted for age, height, and gender; Crapo, Morris, & Gardner, 1981) as assessed with spirometry by a speech-language pathologist (Miller et al., 2005) using a

Koko spirometer (Grace Medical). Participants who met the criteria were enrolled into the full experimental protocol.

Study Size

An a priori power analysis indicated 30 participants was required to achieve 80% statistical power using a repeated-measures design with an alpha of .05 and an anticipated moderate effect size for R_{law} and Ve. Counterbalancing of vocal SPL and workload conditions described shortly required the total participant number be a multiple of 4, and therefore, 32 female participants completed the protocol. Four men also participated, and a tabular summary of their data is presented separately from the data of female participants.

Procedure and Equipment

Screening and Exercise Testing

Volunteers satisfying the criteria were enrolled. Participants engaged in preexperimental procedures to determine low and high treadmill workloads for achieving target heart rates of the systematically manipulated experimental treadmill workloads, with the exception of the rest condition. Experimental procedures required exercise on a Biodex Gait Trainer 2 motorized treadmill (Biodex Medical Systems, Inc.) using a ramping protocol as heart rate was continuously monitored with a Polar T31 Transmitter Heart Rate Sensor (Polar Electro, Inc.). The protocol involved a systematic increase in treadmill grade and speed following every minute of testing, until participants achieved an age-predicted heart rate for the high-workload condition as determined by the Karvonen formula (target heart rate [HR] = [max HR, i.e., $220 - \text{age}$] – resting HR * 0.70] + resting HR; Kelly, 2006).

Training of Voice Task

Instrumental setup utilized the Phonatory Aerodynamic System 6600 (PAS 6600), and calibration was carried out according to the manufacturer's instructions (KayPENTAX, 2009). Participants stood on a treadmill and held the PAS 6600 mask unit to their face to cover the mouth and nose while two straps from a neoprene mask secured the PAS 6600 in place. Participants maintained their grip on the PAS 6600 handles throughout testing. Before commencing R_{law} training, participants' spontaneous vocal SPL and fundamental frequency (f_0) were determined using well-vetted procedures (Russell et al., 1998) while reading the *Rainbow Passage* (Fairbanks, 1960) for 5 min having been instructed to "speak normally." The middle 3 min of the passage were analyzed for spontaneous vocal SPL and f_0 .

Then, participants learned procedures for R_{law} data collection. Training required participants to produce a string of seven repeated /pa:/ syllables at a rate of 1.5 syllables per second in both spontaneous and loud voices until produced consistently at an individually determined f_0 (± 1 ST) as established by a certified voice-specialized clinician's auditory-perception and PAS 6600 output. A reference pitch was provided with a pitch pipe, and a Matrix MR500 Metronome (manufactured in South Korea) maintained a recurring sound at 88 bpm. Participants who had difficulty matching

the reference pitch received vocal models of the pitch in the appropriate octave until they successfully achieved the reference pitch.

The target vocal intensity for loud vocal SPL was the average spontaneous vocal SPL derived from reading plus 10 dB (± 2 dB). This loud vocal SPL target ensured an adequate change of ventilatory stress (Russell et al., 1998) and provided a distinctly different loudness compared to spontaneous vocal SPL (Hoffman-Ruddy, Lehman, Crandell, Ingram, & Sapienza, 2001). Most participants demonstrated loud vocal SPL without difficulty; for some, coaching was provided with hand signals. Training ceased after both principal investigator and participant expressed confidence in correct execution of the R_{law} task.

Equipment and Setup

Instrumental setup (see Figure 2) utilized components of the PAS 6600 and the CareFusion Encore Metabolic Cart (CareFusion Corp). The PAS 6600 was used for measurement of intraoral pressure and airflow rate, as well as recording the voice signal. PAS 6600 hardware consists of a pneumotach coupled to a facemask with an integral intraoral pressure tube and an external microphone. The external microphone on the PAS 6600 was repositioned to accommodate the serial attachment of the CareFusion Encore mass flow sensor behind the PAS 6600 pneumotach. The new position of the external microphone behind the CareFusion Encore mass flow sensor placed it further from the mouth, requiring manual calibration before data collection.

Calibration of the PAS 6600 pneumotach was carried out by depressing a 1.0-L syringe for 2–4 s (proper value: $1.0 \text{ L} \pm 1\%–2\%$). The PAS 6600 system automatically calibrates intraoral pressure and was completed before data collection for each experimental condition for each participant.

Figure 2. Diagram of setup for experimental data collection. IOP = intra-oral pressure.

The repositioned external microphone was manually calibrated using published procedures before each testing session since the distance from the mouth increased with the new microphone location (Russell et al., 1998). To calibrate, the PAS 6600 was attached to a Styrofoam display head that housed a speaker within the mouth. A 200-Hz tone played for 30 s while the PAS 6600 microphone recorded. At the same level as the external microphone, a Larson Davis System 824 Sound Level Meter (Larson Davis Inc.) set to slow setting and C weighting collected the 200-Hz tone's SPL in dB to provide a correction factor. This correction factor was added to the SPL value calculated by the PAS 6600 at data analysis. The CareFusion Encore mass flow sensor was calibrated by depressing a 3.0-L syringe for 2–4 s (proper value: $3.0 \text{ L} \pm 1\%–2\%$). Gas concentrations were calibrated prior to each participant session using two-span gases: (a) 26% O_2 , 0% CO_2 , and the balance in nitrogen and (b) 16% O_2 , 0% CO_2 , and the balance in nitrogen.

Experimental Procedure

An overview of experimental procedures is shown in Table 1. First, participants stood on a SensorMedics 2000 Treadmill (SensorMedics Corp.) wearing a heart rate sensor and breathed quietly into the PAS 6600 for 5 min of tidal breathing. Concerns are negligible for rising CO_2 levels with the use of a face mask over the duration of the experimental protocol (Huber, Stathopoulos, Bormann, & Johnson, 1998). Facemask still in place, participants produced a seven-syllable /pa:/ sequence at rest while standing, using the predetermined f_0 and spontaneous vocal SPL and then the predetermined f_0 and loud vocal SPL (+ 10 dB above spontaneous SPL, ± 2 dB). The instruction for the non-goal-oriented spontaneous vocal SPL condition was, "Speak three sets of seven syllables of /pa:/ with your typical voice until instructed to stop at the following pitch and speaking rate." The instruction for the goal-oriented loud vocal SPL condition was, "Speak three sets of seven syllables of /pa:/ continuously, with a loud and clear voice until told to stop at the following pitch and speaking rate."

During loud vocal SPL, participants received a hand gesture from the experimenter to increase or decrease vocal intensity depending on visual inspection of the PAS 6600 output screen displaying two reference lines for the target vocal intensity range. Some participants completed additional sets if inspection determined loud vocal SPL had not been achieved. Clarity was included in the instruction as voice quality interacts with loudness, and it is a valid ecological communication goal for intelligibility purposes. However, clarity was not an inclusion criterion for using data during analysis; the instruction served to motivate participants in a high-effort voice production. Attainment of vocal clarity was not determined to be a problem during collection of data as determined informally through auditory perception.

Following completion of the first set of R_{law} tasks, participants observed a 1-min rest period. Subsequently, participants completed a rest-to-work transition on the treadmill. The appropriate workload was set per participant-specific

Table 1. Template for experimental procedures.

Condition	Activity	Time (min)
First experimental session		
Rest	Breathe quietly with no voicing (rest no-voice baseline)	5
	Produce a consonant–vowel syllable string (spontaneous [spon] voice baseline)	½
	Breathe quietly with no voicing to return to rest baseline	1
	Produce a consonant–vowel syllable string (loud voice)	½
Rest-to-work transition	Walk on treadmill to achieve a low workload	3
50% of age-predicted maximal heart rate (low)	Breathe quietly with no voicing at a low workload (50% no voice)	1
	Produce a consonant–vowel syllable string (spon 50%)	½
	Breathe quietly with no voicing to return to 50% baseline	1
	Produce a consonant–vowel syllable string (loud 50%)	½
Recovery period	Walk on treadmill until resting heart rate is achieved	3
Second experimental session		
Rest	Breathe quietly with no voicing (rest no-voice baseline)	5
	Produce a consonant–vowel syllable string (spon baseline)	½
	Breathe quietly with no voicing to return to rest baseline	1
	Produce a consonant–vowel syllable string (loud voice)	½
Rest-to-work transition	Run on treadmill to achieve a high workload	3
70% of age-predicted maximal heart rate (high)	Breathe quietly with no voicing at a high workload (70% no voice)	1
	Produce a consonant–vowel syllable string (spon 70%)	½
	Breathe quietly with no voicing to return to 70% baseline	1
	Produce a consonant–vowel syllable string (loud 70%)	½
Recovery period	Walk on treadmill until resting heart rate is achieved	3

speed and grade adjustments to achieve workloads of either 50% or 70% of age-predicted maximal heart rate. Participants maintained that workload for 2 min to achieve steady state in oxygen consumption (VO_2 ; McArdle et al., 2010), establishing new steady-state baselines for each workload. The same R_{law} tasks were repeated as previously, using the same vocal SPL conditions, after workload steady state had been achieved. Finally, participants completed a recovery period until heart rate responses resolved to baseline. These procedures were repeated during the second session after participants reviewed R_{law} task procedures. The experimental protocol at the second session required participants to engage in the workload they had not yet encountered (50% or 70%). Within participants, the same order of spontaneous versus loud vocal SPL was used for the first session.

Variables

Independent variables were vocal SPL condition (no voice [quiet breathing], spontaneous, and loud vocal SPL) and workload (standing [rest], walking at 50% of age-predicted maximal heart rate [low], and walking at 70% of age-predicted maximal heart rate [high]). Dependent variables were R_{law} (in $\text{cmH}_2\text{O}/[\text{L}/\text{s}]$), V_e (in L/min), and P_{ETCO_2} (in mmHg). Covariates were f_0 (in Hz) and SPL (in dB).

Data Sources and Measurement Methods

R_{law} , mean P_s , and mean U were derived from the PAS 6600 signal during phonation. The middle five pressure peaks generated during /p/ of the respective signals and their corresponding airflow signal during the vowel portion were manually selected and examined for acceptable morphology, intensity, and frequency based on visual inspection (Helou & Solomon, 2011; Holmberg et al., 1988; Smitheran & Hixon,

1981). For acceptable tokens, f_0 during all voice conditions could vary by ± 1 ST, while vocal SPL during the loud condition could fluctuate by ± 2 dB as determined during data analysis using the PAS 6600 output. A digital tone generator (<http://www.seventhstring.com/tuningfork/tuningfork.html>; Limited, 2009) confirmed f_0 values miscalculated by the PAS 6600 system at data analysis, using the nearest frequency of the tone produced by the online generator that best corresponded to the participant's pitch. Criteria for selection were determined a priori and applied rigorously; samples not meeting the criteria were discarded. Values for P_s , U , f_0 , and SPL were calculated using the PAS 6600 software and retained for statistical analysis.

The CareFusion Encore data acquisition system software used breath-by-breath analysis to capture respiratory data. A research assistant recorded times for when participants started and stopped an R_{law} task to ensure respiratory and phonatory data were time locked. Respiratory data were continuously recorded during all voice conditions of the R_{law} task, all periods of silence for each workload, and the rest condition when participants stood on the treadmill. Baseline values of V_e and P_{ETCO_2} were calculated by averaging data of voicing (spontaneous vocal SPL) while standing (at rest). For voiced segments, V_e data were calculated from the entire period during which the participant phonated (i.e., from beginning to end of the R_{law} task). The last 30 s of each quiet breathing period before phonating were used as respiratory data for the nonphonated conditions at each workload level (i.e., V_e and VO_2). Data for P_{ETCO_2} were extracted from a 15-s segment following completion of an R_{law} task. A heart rate sensor provided pulse data. Covariates were order of workload and vocal SPL conditions, attainment of f_0 and SPL targets, and their respective values.

Bias

A counterbalancing sequence of conditions was randomly assigned to participants at the first experimental session, with one exception described shortly (see Table 5). Online software (Haahr, 1998) generated a randomized list of counterbalanced conditions. The same ordering of spontaneous vocal SPL versus loud vocal SPL was used, within participants, for R_{law} tasks across the experimental protocol.

Statistical Analyses

Confirmation of Workloads

Paired-samples t test was performed to determine whether target and observed heart rates differed at low and high workloads. A one-way repeated-measures analysis of variance (ANOVA-RM) was performed on observed heart rates during quiet breathing as a function of workload. Post hoc comparisons were completed using a Bonferroni adjustment.

Generalized Estimating Equations

Generalized estimating equations (GEEs; Zeger & Liang, 1986; Zeger, Liang, & Albert, 1988) with robust standard error estimates (Huber/White Sandwich estimators) were used to evaluate associations, adjusting for confounders. We specified a Gaussian (i.e., normal) distributional family for unratioed continuous dependent variables (i.e., V_e , VO_2 , and $P_{ET}CO_2$) and a gamma family for R_{law} , which is ratioed, due to its strict positivity and excess skew in the positive direction. For both families, a log link function was selected with an exchangeable correlation structure (Ballinger, 2004). Data for f_0 and SPL were centered. Indicators were created for not meeting vocal SPL and f_0 targets; this allowed for interpretation of model constants as typical responses for the average participant who achieved vocal SPL and f_0 targets. Analyses required fitting two models: one for silent conditions (no adjustments except sequence order) and one for voice conditions (with f_0 and vocal SPL adjustments; StataCorp, 2017).

The results for the GEE models are presented as ratios representing relative percentage changes. Ratios > 1 are interpreted as an increase in the level of an outcome, and ratios < 1 are interpreted as a decrease in the level of an outcome. Ratios sufficiently close to 1 are not significantly associated with either an increase or a decrease. To assess the relevance of all potential predictors of each outcome, 95% confidence intervals were calculated. Statistical significance was set at $p < .05$, and all reported p values are two-sided.

Results

Participant Characteristics

One hundred ninety-nine individuals completed an online prescreening form to determine initial eligibility. Of those, 32 recreationally active and vocally untrained women met criteria for participation and completed all experimental

procedures. One female participant who experienced an unanticipated adverse event (i.e., vasovagal response) during data collection was replaced to preserve the target sample size for statistical analyses. Four men also completed the experimental protocol, but analysis of their data was done only descriptively. Participant characteristics are shown in Table 2.

Target Heart Rate and Workload Exposure

The experiment imposed heart rates that were greater than resting and that were achieved by treadmill exercise. Treadmill workloads included standing (rest) and walking at 50% of age-predicted maximal heart rate (low) and 70% of age-predicted maximal heart rate (high). The following summary describes relative attainment of different treadmill workloads as assessed by heart rate data.

Using a paired-samples t test, no differences were observed between target and observed heart rates at low (133.1 ± 5.0 vs. 134.1 ± 5.8 bpm) and high (158.6 ± 3.7 vs. 159.1 ± 5.5 bpm) workloads, $t(31) = 1.599$, $p = .120$, $d = 0.28$ and $t(31) = 0.758$, $p = .454$, $d = 0.13$, respectively. A one-way ANOVA-RM was performed on observed heart rates as a function of treadmill workload to ensure that heart rates were significantly different among treadmill workload conditions. The assumption of sphericity had been violated, Mauchly's $W = .661$, $\chi^2(2) = 12.426$, $p = .002$, and a Greenhouse–Geisser adjustment was performed. The one-way ANOVA-RM revealed a main effect of workload, $F(1.494, 46.299) = 2866.643$, $p < .001$, $\eta^2 = .989$. Post hoc analyses revealed mean heart rate was significantly different for all comparisons, in predicted directions (rest [69.4 ± 9.6 bpm] vs. low [134.1 ± 5.8 bpm], $p < .001$; rest vs. high [159.1 ± 5.5 bpm], $p < .001$; and low vs. high, $p < .001$).

Table 2. Participant physical and voice characteristics.

Characteristic	Female ($n = 32$)	Male ($n = 4$)
	M (SD)	M (SD)
Age (years)	23.0 (3.4)	24.8 (3.4)
Body weight (kg)	59.8 (7.4)	69.5 (13.0)
Body height (cm)	165.5 (6.5)	178.4 (11.0)
BMI	21.8 (2.2)	21.8 (1.7)
Heart rate (bpm)	69.4 (9.2)	65.3 (8.6)
Systolic blood pressure (mmHg)	99.6 (7.4)	110.8 (5.1)
Diastolic blood pressure (mmHg)	64.9 (5.4)	66.5 (6.4)
FVC ^a (L), % predicted	4.0 (0.5), 98%	5.3 (1.2), 95%
FEV1 ^a (L), % predicted	3.4 (0.3), 97%	4.4 (0.8), 95%
CAPE-V rating (mm)	3.4 (2.3)	2.5 (1.9)
VHI-10 score	1.9 (2.0)	3.5 (3.3)

Note. BMI = body mass index; FVC = forced vital capacity; FEV1 = forced expiratory volume in the first second; CAPE-V = Consensus Auditory–Perceptual Evaluation of Voice; VHI-10 Voice Handicap Index-10.

^a% Predicted based on normative data from Crapo et al. (1981).

Voice Goal Attainment

The experiment constrained f0 across all conditions with voice and constrained vocal SPL in loud vocal SPL conditions with vocal SPL free to vary in the spontaneous vocal SPL conditions. f0 Criteria were not met in 45% of opportunities during the experiment, and f0 violations primarily occurred in the loud vocal SPL conditions as commonly occurs with increases in vocal intensity. The lack of f0 attainment was considered of minimal concern given it lacks a clear influence over R_{law} (Holmberg et al., 1989). In the loud vocal SPL condition, participants were expected to attain a vocal intensity 10 dB or greater than their spontaneous speaking vocal intensity. Some participants—in loud vocal SPL conditions—exhibited vocal intensities that were greater than the expected vocal SPL target, but participants were never below. Further specifics on f0 and vocal SPL are described subsequently (see Table 3).

f0

One participant had missing f0 data due to a technical issue. Means and standard deviations of f0 for women and men are shown in Table 3. Data on f0 were not excluded from statistical analyses when participants did not attain the target f0 given its lack of a clear relationship with R_{law} (Holmberg et al., 1989). A 2×3 ANOVA-RM was performed

on f0 as a function of vocal SPL and workload to determine differences in f0 across conditions. A violation of the assumption of sphericity existed for the workload effect, Mauchly's $W = .745$, $\chi^2(2) = 7.371$, $p = .025$. The 2×3 ANOVA-RM revealed main effects of vocal SPL, $F(1, 26) = 53.233$, $p < .001$, partial $\eta^2 = .672$, and workload, $F(1.680, 43.693) = 15.246$, $p < .001$, partial $\eta^2 = .370$. Post hoc comparisons revealed significantly higher f0 during loud vocal SPL ($M = 253.7$, $SE = 5.226$) than spontaneous vocal SPL ($M = 228.1$, $SE = 5.038$). Participants had significantly higher f0s in low ($M = 242.3$, $SE = 5.179$) and high ($M = 251.3$, $SE = 6.510$) workloads compared to rest ($M = 229.3$, $SE = 4.083$), $p = .001$ and $p < .001$, respectively.

Vocal Intensity (SPL)

Means and standard deviations of vocal SPL conditions for women and men are reported in Table 3. Follow-up analyses were performed on data from participants who met the loud vocal SPL target. A 2×3 ANOVA-RM was performed on SPL as a function of vocal SPL and workload. The ANOVA-RM revealed an interaction effect of vocal SPL and workload, $F(2, 50) = 11.347$, $p < .001$, partial $\eta^2 = .312$. The simple main effect of workload for spontaneous vocal SPL, $F(2, 50) = 44.811$, $p < .001$, partial $\eta^2 = .642$, and loud vocal SPL, $F(2, 52) = 14.026$, $p < .001$, partial

Table 3. Unadjusted group means (standard deviations) of phonatory and respiratory measures for women and men.

Condition by sex	Primary phonatory	Secondary phonatory		Primary respiratory		Covariates	
	R_{law} (cmH ₂ O/[L/s])	P_s (cmH ₂ O)	U (L/s)	Ve (L/min)	P_{ETCO_2} (mm Hg)	f0 (Hz)	SPL (dB)
	M (SD)	M (SD)	M (SD)	M (SD)	M (SD)	M (SD)	M (SD)
Women							
Rest-standing							
Quiet/silent				11.9 (1.7)	32.1 (2.5)		
Spontaneous	62.6 (20.3)	9.5 (2.3)	0.16 (0.05)	9.7 (2.9)	34.0 (2.2)	217.9 (21.0)	86.5 (3.3)
Loud	120.5 (45.8)	16.5 (4.2)	0.15 (0.05)	10.6 (2.7)	33.1 (2.0)	240.7 (23.9)	95.1 (2.7)
Low workload							
Quiet/silent				36.1 (6.8)	43.7 (3.0)		
Spontaneous	69.8 (33.3)	12.0 (3.4)	0.20 (0.07)	15.3 (4.5)	54.5 (4.5)	229.4 (29.2)	91.3 (4.4)
Loud	104.1 (40.5)	17.0 (4.1)	0.18 (0.06)	16.4 (8.7)	53.9 (4.1)	255.2 (30.1)	97.3 (3.6)
High workload							
Quiet/silent				46.6 (8.9)	45.2 (3.7)		
Spontaneous	69.3 (28.6)	12.7 (3.4)	0.21 (0.07)	18.0 (4.0)	59.5 (5.3)	237.2 (35.2)	91.6 (3.9)
Loud	92.7 (42.4)	16.8 (5.6)	0.20 (0.07)	19.3 (13.7)	59.1 (4.5)	265.4 (36.0)	97.3 (2.7)
Men							
Rest-standing							
Quiet/silent				12.7 (1.4)	34.9 (1.5)		
Spontaneous	40.6 (15.6)	7.2 (0.8)	0.19 (0.05)	8.9 (3.1)	36.7 (2.2)	122.9 (14.5)	94.0 (3.0)
Loud	53.8 (25.7)	14.5 (5.9)	0.28 (0.02)	8.5 (0.5)	34.9 (2.4)	162.5 (17.2)	100.8 (1.4)
Low workload							
Quiet/silent				43.0 (11.1)	47.1 (4.3)		
Spontaneous	28.0 (9.3)	8.6 (1.0)	0.33 (0.10)	12.2 (3.2)	58.3 (6.3)	136.3 (26.5)	97.6 (1.8)
Loud	53.4 (25.0)	18.4 (4.8)	0.37 (0.09)	13.4 (4.5)	56.7 (5.0)	172.3 (22.3)	102.5 (2.1)
High workload							
Quiet/silent				57.8 (12.4)	52.2 (6.8)		
Spontaneous	24.6 (0.5)	9.8 (3.2)	0.33 (0.01)	16.8 (3.1)	62.9 (4.1)	129.7 (13.6)	97.4 (3.5)
Loud	45.3 (32.2)	15.8 (6.3)	0.41 (0.14)	22.4 (7.7)	60.6 (4.6)	193.6 (39.3)	106.2 (0.6)

Note. R_{law} = laryngeal airway resistance; P_s = estimated subglottal pressure; U = translaryngeal airflow; Ve = minute ventilation; P_{ETCO_2} = end-tidal carbon dioxide; f0 = fundamental frequency; SPL = sound pressure level.

$\eta^2 = .350$, was significant. Post hoc comparisons revealed significantly higher vocal SPL during spontaneous vocal SPL in low and high workloads compared to rest, $p < .001$ and $p < .001$, respectively. Participants had significantly higher vocal SPLs during loud vocal SPL in low and high workloads compared to rest, $p < .001$ and $p < .001$, respectively. The simple main effect of vocal SPL for rest, low, and high workloads was significant. At each workload, participants had a higher vocal intensity in loud vocal SPL compared to spontaneous vocal SPL: rest, $t(31) = 15.684$, $p < .001$; low, $t(27) = 7.138$, $p < .001$; and high, $t(29) = 9.089$, $p < .001$.

GEEs

GEE analyses were conducted to investigate differences among conditions, some which imposed vocal intensity and heart rate targets and other conditions during which vocal intensity was free to vary. GEE analyses with robust standard error estimates were used, specifying a Gaussian family for unratiod data and a gamma family for ratioed data, log link function, and an exchangeable correlation structure. Confounder adjustments, as dictated by model type, included (a) continuous variables (f0 and SPL values), mean centered (subtracting sample means); (b) indicators for meeting vocal SPL and f0, coded as categorical variables (met f0 [0/1] and met vocal SPL [0/1]); and (c) counterbalancing sequence. This summary highlights results of phonatory and respiratory responder variables to different vocal SPL and workload conditions.

Aerobic Demands of Workload

Results of the GEE model for VO_2 are shown in Table 4. For participants who met vocal SPL and f0 targets, participants in quiet breathing during the rest workload had an average VO_2 of 4.63 L/(kg/min). In quiet breathing, VO_2 increased significantly for all workload comparisons (low vs. rest, high vs. rest, and high vs. low; $ps < .001$).

R_{law}

Unadjusted means and standard deviations for R_{law} and its component parts of P_s and transglottal U are reported in Table 3. Unadjusted individual and group data on R_{law} are plotted in Figure 3a. For women, 14% (37/256) of the total number of cells containing R_{law} data were excluded from analysis for not meeting morphology criteria. Missing data accounted for 9% and 14% in spontaneous vocal SPL and loud vocal SPL at rest, 19% for both vocal SPL conditions at a low workload, and 19% and 13% in spontaneous vocal SPL and loud vocal SPL at a high workload, respectively.

Results of the GEE models for R_{law} are shown in Table 5. For participants who met vocal SPL and f0 targets, adjusting for actual f0 and SPL values, participants at rest with a spontaneous vocal SPL had an average R_{law} of 72.6 cm H_2O /(L/s). Changing spontaneous vocal SPL to loud while maintaining rest workload, R_{law} significantly increased by an estimated 53% relative to the response at spontaneous vocal SPL (95% confidence interval [19.2%, 95.5%], $p = .001$). R_{law} significantly increased when changing spontaneous vocal SPL to loud at a low workload (+ 25.3%, 95% confidence interval [5.5%, 48.7%], $p = .010$). When requiring loud vocal SPL, R_{law} decreased significantly under low and high workloads as compared to rest (low vs. rest, $p < .001$; high vs. rest, $p < .001$).

V_e

Unadjusted means and standard deviations for respiratory variables are reported in Table 3. Unadjusted individual and group data on V_e are plotted in Figure 3b. Results of the GEE models for V_e are shown in Table 6. For participants who met vocal SPL and f0 targets, participants in quiet breathing during the rest workload had an average V_e of 12.2 L/min. In quiet breathing, V_e increased significantly for all workload comparisons (low vs. rest, high vs. rest, and high vs. low; $ps < .001$). For participants who met vocal SPL and f0 targets, adjusting for actual f0 and SPL values, participants in the rest workload with spontaneous vocal SPL had an average V_e of 10.6 L/min. Changing rest workload to low or high while maintaining spontaneous vocal SPL, V_e significantly increased relative to the response at rest ($ps < .001$). Changing rest workload to low or high while maintaining loud vocal SPL, V_e significantly increased relative to the response at rest ($ps < .001$). V_e significantly increased when changing spontaneous vocal SPL to loud at low and high workloads, $p = .001$ and $p < .001$, respectively.

P_{ETCO_2}

Results of the GEE models for P_{ETCO_2} are shown in Table 7. For participants who met vocal SPL and f0 targets, participants in quiet breathing during the rest workload had an average P_{ETCO_2} of 32.17 mmHg. In quiet breathing, P_{ETCO_2} increased significantly for all workload comparisons (low vs. rest, high vs. rest, and high vs. low; $ps < .001$). For participants who met vocal SPL and f0 targets, adjusting for actual f0 and SPL values, participants at rest workload with spontaneous vocal SPL had an average P_{ETCO_2} of 33.53 mmHg. Changing rest workload to low or high while

Table 4. VO_2 contrasts: pairwise comparisons of marginal linear predictions (women only).

Reference condition	Comparison condition	exp(b) Comp/Ref	SE	<i>p</i>	95% Confidence interval
Quiet_Rest	Quiet_Low	5.22	0.12	< .001	[4.99, 5.46]
	Quiet_High	6.67	0.17	< .001	[6.35, 7.01]
Quiet_Low	Quiet_High	1.28	0.02	< .001	[1.24, 1.32]

Note. Bolded *p* values (e.g., **< .001**) represent statistically significant results. VO_2 = oxygen consumption.

Figure 3. (a) Plot of unadjusted individual and group data on laryngeal airway resistance by vocal sound pressure level (SPL) and workload condition. (b) Plot of unadjusted individual and group data on minute ventilation by vocal SPL and workload condition.

maintaining spontaneous vocal SPL, $P_{ET}CO_2$ significantly increased relative to the response at rest ($ps < .001$). Changing rest workload to low or high while maintaining loud vocal SPL, $P_{ET}CO_2$ significantly increased relative to the response at rest ($ps < .001$).

Discussion

Loud voice produces an acoustic signal of sufficient discriminative power that contrasts from interfering signals and facilitates perceptual distinctiveness (Bourne & Garnier,

Table 5. R_{law} contrasts: pairwise comparisons of marginal linear predictions (women only).

Reference condition	Comparison condition	exp(b) Comp/Ref	SE	<i>p</i>	95% Confidence interval
Norm_Rest	Norm_Low	1.01	0.11	.951	[0.82, 1.24]
	Norm_High	0.95	0.10	.656	[0.78, 1.16]
	Loud_Rest	1.53	0.19	.001	[1.19, 1.95]
	Loud_Low	1.26	0.19	.131	[0.93, 1.70]
	Loud_High	1.12	0.17	.457	[0.83, 1.50]
Norm_Low	Norm_High	0.95	0.06	.417	[0.84, 1.07]
	Loud_Rest	1.52	0.10	< .001	[1.33, 1.73]
	Loud_Low	1.25	0.11	.010	[1.05, 1.49]
	Loud_High	1.11	0.10	.242	[0.93, 1.32]
Norm_High	Loud_Rest	1.60	0.14	< .001	[1.34, 1.90]
	Loud_Low	1.32	0.15	.014	[1.06, 1.64]
	Loud_High	1.17	0.13	.148	[0.94, 1.45]
Loud_Rest	Loud_Low	0.83	0.04	< .001	[0.74, 0.92]
	Loud_High	0.73	0.05	< .001	[0.64, 0.84]
Loud_Low	Loud_High	0.89	0.05	.046	[0.79, 1.00]

Note. Bolded *p* values (e.g., **< .001**) represent statistically significant results. R_{law} = laryngeal airway resistance.

2012; DeLeo LeBorgne, Lee, Stemple, & Bush, 2010; Stone, Cleveland, Sundberg, & Prokop, 2003). Aspects of voice such as vocal SPL for some professional voice users are relatively fixed due to occupational demands (i.e., loud speech; Hoffman-Ruddy et al., 2001). As for patients with respiratory conditions, the obstructive effect of the larynx on ventilation when speaking exacerbates breathing symptoms (Lee et al., 1998). Using aerodynamic outcome measures, this study investigated whether phonatory glottal valving in the face of different workload conditions is dependent on SPL targets. The hypothesis was that adjustments in glottal valving as measured by R_{law} and respiratory response as measured by V_e would be dependent upon vocal SPL targets. Specifically, we hypothesized R_{law} would be greater in loud vocal SPL

than in the spontaneous vocal SPL condition that was free to vary, regardless of treadmill workload, due to glottal valving adjustments that favor voicing in loud vocal SPL but that reduce V_e and result in hypercapnia. We further hypothesized that the difference in R_{law} between loud and spontaneous vocal SPLs would widen as treadmill workload increased from rest to low and high due to glottal valving adjustments in spontaneous vocal SPL that favor the body's metabolic respiratory requirements to increase V_e for greater CO_2 expulsion. Results were largely congruent with hypotheses, partially validating a theory of action in vocal motor control. Consistent with a theory of action, phonatory goals such as vocal SPL seemed important when specified, and left unconstrained, glottal valving appeared to favor the body's

Table 6. V_e contrasts: pairwise comparisons of marginal linear predictions (women only).

Reference condition	Comparison condition	exp(b) Comp/Ref	SE	<i>p</i>	95% Confidence interval
Quiet_Rest	Quiet_Low	3.04	0.08	< .001	[2.88, 3.20]
	Quiet_High	3.92	0.11	< .001	[3.71, 4.15]
Quiet_Low	Quiet_High	1.29	0.02	< .001	[1.24, 1.34]
Norm_Rest	Norm_Low	1.86	0.33	< .001	[1.31, 2.64]
	Norm_High	2.37	0.26	< .001	[1.91, 2.94]
	Loud_Rest	1.20	0.22	.311	[0.84, 1.72]
	Loud_Low	3.83	1.03	< .001	[2.25, 6.50]
	Loud_High	5.80	1.70	< .001	[3.27, 10.29]
Norm_Low	Norm_High	1.27	0.17	.072	[0.98, 1.65]
	Loud_Rest	0.65	0.10	.004	[0.48, 0.87]
	Loud_Low	2.06	0.45	.001	[1.34, 3.16]
	Loud_High	3.11	0.62	< .001	[2.10, 4.61]
Norm_High	Loud_Rest	0.51	0.07	< .001	[0.38, 0.68]
	Loud_Low	1.62	0.36	.032	[1.04, 2.51]
	Loud_High	2.45	0.49	< .001	[1.65, 3.63]
Loud_Rest	Loud_Low	3.18	0.68	< .001	[2.09, 4.83]
	Loud_High	4.81	1.14	< .001	[3.03, 7.65]
Loud_Low	Loud_High	1.51	0.32	.048	[1.00, 2.29]

Note. Bolded *p* values (e.g., **< .001**) represent statistically significant results. V_e = minute ventilation.

Table 7. $P_{ET}CO_2$ contrasts: pairwise comparisons of marginal linear predictions (women only).

Reference condition	Comparison condition	exp(b) Comp/Ref	SE	<i>p</i>	95% Confidence interval
Quiet_Rest	Quiet_Low	1.36	0.02	< .001	[1.32, 1.40]
	Quiet_High	1.41	0.02	< .001	[1.36, 1.46]
Quiet_Low	Quiet_High	1.03	0.01	< .001	[1.01, 1.05]
Norm_Rest	Norm_Low	1.60	0.03	< .001	[1.55, 1.66]
	Norm_High	1.74	0.03	< .001	[1.67, 1.80]
	Loud_Rest	0.99	0.02	.606	[0.95, 1.03]
	Loud_Low	1.60	0.04	< .001	[1.53, 1.68]
	Loud_High	1.76	0.05	< .001	[1.66, 1.86]
	Norm_High	1.08	0.02	< .001	[1.05, 1.11]
Norm_Low	Loud_Rest	0.62	0.01	< .001	[0.59, 0.64]
	Loud_Low	1.00	0.01	.902	[0.98, 1.02]
	Loud_High	1.09	0.02	< .001	[1.05, 1.14]
	Norm_High	0.57	0.01	< .001	[0.55, 0.59]
Norm_High	Loud_Rest	0.92	0.01	< .001	[0.90, 0.95]
	Loud_Low	1.01	0.02	.574	[0.97, 1.05]
	Loud_High	1.62	0.02	< .001	[1.58, 1.67]
Loud_Rest	Loud_Low	1.77	0.03	< .001	[1.72, 1.83]
	Loud_High	1.09	0.01	< .001	[1.07, 1.12]

Note. Bolded *p* values (e.g., **< .001**) represent statistically significant results. $P_{ET}CO_2$ = end-tidal carbon dioxide.

metabolic needs as evidenced by lower R_{law} values. Discussion of results is limited to women who met their loud vocal SPL target.

Key Results

R_{law}

Consistent with hypotheses, in women who met their loud vocal SPL target, R_{law} values at rest were significantly higher in loud vocal SPL than during spontaneous vocal SPL. The higher R_{law} in loud vocal SPL than in spontaneous vocal SPL also was evident at a low workload, although the magnitude of difference diminished as participants went from standing to walking on the treadmill at a low workload intensity. R_{law} at a high workload was not significantly different between loud and spontaneous vocal SPLs. Contrary to hypotheses, R_{law} decreased slightly under low and high treadmill workloads when compared to rest, but decreases were greater in loud vocal SPL than in spontaneous vocal SPL.

These findings suggest loud voice can “win” using physiology specific to a vocal SPL target in the face of high-respiratory drive conditions, at least in the short term. That is, metabolic needs increased to accomplish treadmill walking in the low-workload condition, yet individuals still met vocal SPL targets in the loud condition. In fact, regardless of workload, individuals met vocal SPL targets in the loud condition, and some even exceeded their loud vocal SPL target. R_{law} decreased in loud vocal SPL as workload intensity increased, but the loud vocal SPL target was still attained.

The lack of R_{law} differences between loud vocal SPL and spontaneous vocal SPL conditions at a high workload appears to be due to increases in P_{sub} in the spontaneous vocal SPL condition. Though SPL was free to vary in the

spontaneous vocal SPL condition, SPL increased systematically in the spontaneous condition as workload intensity increased. This increase in SPL may be due to larger lung pressures as the respiratory system increases ventilation to meet metabolic demands for a greater intensity of exercise. Despite the lack of differences between the vocal SPL conditions, R_{law} in the loud vocal SPL condition was still greater at a high workload than at rest. Together, these findings provide evidence that vocal motor control is goal dependent and that speakers demonstrate a physiology of vocal activity despite initial conditions that perturb the respiratory system.

Respiratory Variables

As predicted, V_e increased significantly at low and high workloads from a baseline of quiet breathing at rest, indicating hyperpnea. During spontaneous and loud vocal SPL, increases in workload to low and high significantly increased V_e from baseline values. V_e did not significantly differ between loud vocal SPL and spontaneous vocal SPL at rest. Consistent with hypotheses, loud vocal SPL produced significantly greater V_e as compared to spontaneous vocal SPL at low and high workloads.

Increasing the intensity of a physiologic stressor typically increases ventilation, while increases in airway resistance from phonation generally decreases ventilation (Bunn & Mead, 1971; Doust & Patrick, 1981). Changes in ventilation are known to potentially impact gas exchange. Consistent with hypotheses, $P_{ET}CO_2$ increased significantly at low and high workloads from quiet breathing at rest. An increase in workload significantly affected $P_{ET}CO_2$ within each vocal SPL condition. Both spontaneous vocal SPL and loud vocal SPL demonstrated significantly greater $P_{ET}CO_2$ at low and high workloads compared to rest. Inconsistent with hypotheses, $P_{ET}CO_2$ did not differ between spontaneous vocal SPL and loud vocal SPL at each workload despite differences

in R_{law} between those voice types at rest and low workload. This lack of differences is likely due to the short duration of the R_{law} task.

Interpretation of the Data

Presumably, increases in airflow and breathy voice quality occur with relatively open vocal folds (Peterson, Verdolini-Marston, Barkmeier, & Hoffman, 1994). Although airflow increases during speech in high respiratory drive compared with speech under eupnea (Bailey & Hoit, 2002; Bunn & Mead, 1971; Doust & Patrick, 1981; Hoit et al., 2007), this increase is mainly linked to nonvoiced expirations, frequently at ends of phrases. In contrast, voiced expirations demonstrate only slight increases in airflow compared to spontaneous speech under normal drive. If loud voice is associated with increased vocal fold adduction, then consequently, exhalatory airflow in phonation should be impeded and blood gas levels simultaneously negatively impacted from decreased ventilation (Bailey & Hoit, 2002; Bunn & Mead, 1971; Doust & Patrick, 1981; Hoit et al., 2007; Otis & Clark, 1968). The following expands on findings that participants achieved loud vocal SPL targets under different workload conditions in line with a goal-independent model of vocal motor control.

Phonatory Outcomes

The physiology of the respiratory–laryngeal systems consists of multiple degrees of freedom (Hixon, Weismer, & Hoit, 2008; Pennestri, Cavacece, & Vita, 2005; Sellars & Sellars, 1983; Smith & Mead, 1986; von Leden & Moore, 1961). A theory of action states that motor behavior is goal oriented and functionally specific even in changing initial conditions. Goal-oriented loud voice resulted in a greater change in R_{law} from baseline compared to spontaneous voice to meet a loud acoustic goal, at least over a short duration, at rest and at a low workload. This finding is consistent with research on R_{law} in conditions of normal respiratory drive, in which increases in SPL cause R_{law} to increase (Holmberg et al., 1988; Wilson & Leeper, 1992). Breath holding demonstrates similar goal-oriented control, in which the termination point can be prolonged with distraction or motivation, even when partial pressure of CO_2 rises to supposedly dangerous levels (Parkes, 2006). The participants in this study demonstrated a similar ability to override metabolic need in attainment of loud voice, at least for a short duration. Participants somewhat maintained R_{law} even in spontaneous voice, which was supposed to allow deference toward respiratory goals.

Changes in R_{law} from baseline decreased as workload increased, and this finding was especially true in loud voice. When the goal was respiration, the pulmonary–laryngeal coordinative structure served gas exchange to meet somatic goals, as evidenced by expected changes in R_{law} relative to loud vocal SPL at a high compared to low workload. This study is the first to investigate R_{law} changes as a function of physical activity. Gillespie et al. (2015) found in their female participants that R_{law} and its component parts of P_s and U in spontaneous voice (i.e., unconstrained by

instructions or other experimental maneuvers) did not differ between eupnea and hypercapnia induced via inspiration of CO_2 despite a significant increase in Ve from eupnea. That is, R_{law} exhibited robustness against challenges to homeostasis in individuals free to vary dimensions of voice such as f_0 and vocal SPL during data collection. They suggested R_{law} may be a control parameter for voice that individuals maintain at a relatively constant level, despite respiratory gas exchange. In contrast, we found that R_{law} changes depending on initial conditions, but despite these alterations and the specific R_{law} value, participants achieved specified vocal intensity targets.

Differences in findings between that study and the current one may be due to ventilation requirements. Participants in the current study increased Ve in spontaneous voice at a low workload by approximately 24 L/min over baseline quiet breathing at rest and in spontaneous voice at a high workload by 35 L/min over baseline quiet breathing at rest. Participants in Gillespie's study only increased Ve by 12 L/min from eupnea to hyperpnea. Airway dilation is a known consequence of increased respiratory drive during exercise, including the larynx (Farrell, Joyner, & Caiozzo, 2012). The current study suggests that R_{law} is susceptible to changes in ventilation, although not to the point at which ventilatory requirements prevent individuals from varying SPL. Participants continued to maintain a greater increase in R_{law} in loud voice compared to spontaneous voice, in relation to baseline, across rest and low workloads. In light of this difference, phonatory goals seem important when specified, but left unconstrained, phonation favors metabolic need as evidenced by lower R_{law} .

Research on neural mechanisms of breathing support a motor control organization in which two pathways exist for breathing, that is, a cortical pathway that serves volitional acts of breathing and exerts influence on the medullary respiratory center and a bulbar pathway for central pattern generation of breathing for life (Jürgens, 2009; Ludlow, 2005, 2011). The finding on R_{law} provides new evidence that participants make attempts to meet phonatory goals, at least over a short duration, even when metabolic need is high. This evidence is in line with overcoming increasing metabolic pressures of other volitional acts of breathing (Parkes, 2006), suggesting cortical processes exert influence on lower breathing centers.

Respiratory Outcomes

The study also investigated Ve as a measure of adequate and efficient gas exchange to understand how the body achieves somatic goals in the face of voice production. This study found increases in workload induced a systematic increase in Ve from baseline during breathing and in spontaneous and loud voices, providing evidence of a hyperpneic response due to increases in respiratory drive (McArdle et al., 2010). Gillespie et al. (2015) found participants significantly increased Ve in response to inspired CO_2 . This finding likely reflects the response of peripheral chemoreceptors that react to changes in partial pressures of CO_2 to maintain respiratory homeostasis. However, unlike Gillespie et al. (2015),

the hyperpneic response observed during treadmill exercise in this study most likely reflects several ventilator control mechanisms, including locomotor-linked stimuli, namely, central command (feedforward) and muscle receptors (feedback), as well as matching to metabolic needs via partial pressures of O₂ and CO₂ (feedback; Farrell et al., 2012).

The obstructive nature of the larynx during phonation appeared to manifest differently when considering different SPLs at low and high workloads but not at rest. This finding indicates that changes in R_{law} that occur with loud voice, compared to spontaneous voice, impact ventilation. Taken together, evidence suggests that participants strive to meet SPL targets for a short duration despite sacrificing somatic goals to some extent. Moreover, implicit somatic goals only impact acoustic goals, whether spontaneous or loud, when they substantially increase in strength (Baker et al., 2008; Meckel, Rotstein, & Inbar, 2002). That finding reflects a motor control of respiration that favors the ability of the individual to override ventilatory needs when prioritizing SPL targets, even when somatic goals are strong.

Intense exercise results in more CO₂ production than moderate exercise (McArdle et al., 2010). That participants exhibited a significantly larger increase in $P_{ET}CO_2$ from baseline in the high workload compared to low is not surprising. Research shows that exercise results in increases in $P_{ET}CO_2$ and reflects the importance of lung perfusion and alveolar ventilation to eliminate CO₂, restore partial pressures of CO₂, and maintain acid–base balance (McArdle et al., 2010). More CO₂ in exhaled air reflects normal respiratory function. Gillespie et al. (2015) found that mean $P_{ET}CO_2$ levels during voiced segments were significantly greater in hypercapnia than eupnea, and $P_{ET}CO_2$ was significantly greater in voiced segments compared to nonvoiced ones during eupnea and hypercapnia. Unexpectedly, $P_{ET}CO_2$ did not differ between normal and loud voices at all workloads. This finding indicates that both SPLs provide a similar ventilatory limitation and hypoventilatory response, at least in the short term. In contrast, one study found that loud voice in normal drive results in less $P_{ET}CO_2$ than spontaneous speech, presumably due to the increased ventilation observed in loud speech (Russell et al., 1998). The difference between that study and the current one likely lies in the nature of the ventilatory response from speech. At rest, speech results in hyperventilation, but during exercise, speech results in hypoventilation. Another difference between studies may be task duration, where a longer task may be required to result in differences in $P_{ET}CO_2$.

Generalizability

This study provided a window into phonatory and respiratory function during simultaneous phonation and physical activity. Data indicate acoustic goals matter, even in the face of significant respiratory perturbations. This new knowledge will guide development of evaluation and treatment approaches for voice users who experience respiratory perturbations.

Limitations

Several limitations deserve mention. Although heart rates increase linearly with workload changes, using maximal oxygen consumption to establish workloads may have decreased response variability. Differences in aerobic fitness may have led to variability in the response to a physiologic stressor, for which future studies should control by determining maximal oxygen consumption. The short duration of the voice task likely did not allow metabolic variables to attain steady state when voicing, and future research could require a longer voice task. Participants exhibited difficulty matching pitch with loud voice, and some participants overshot their loud voice goal. Future studies should test ability to match pitch and loudness in all voice conditions, including in high respiratory drive. Finally, participants exhibited some pressure peaks that did not meet morphology requirements, possibly from jarring with treadmill walking. Despite this data loss, results were in line with what was predicted, and it would be unlikely that the “nonpredicted” data were preferentially lost. Future studies may consider a cycle ergometer as an alternative to a treadmill.

Conclusions

This study set out to test a theory of action in investigating the function of the respiratory–laryngeal coordinative structure under different respiratory and phonatory goals. Individuals who focus on an acoustic goal such as loud voice demonstrate R_{law} values that differ from spontaneous voice, despite increasing metabolic need. Participants accomplished goal-directed behavior for loud and, to some extent, spontaneous voice production, despite respiratory perturbations. These results demonstrate greater flexibility in physiology to achieve loud voice, and to some extent spontaneous voice, than previously thought. In relation to respiratory goals, the increased flow restriction that phonation places on airflow during loud voice does appear to impact ventilation requirements and metabolic needs, but for a short duration, acoustic goals may supersede respiratory goals. Spontaneous voice allows for less interference with somatic needs than loud voice, and the ventilatory response better serves metabolic needs when respiratory goals take precedence.

Acknowledgments

This research was supported with awards to Ziegler that included a F31 Fellowship Grant (F31 DC012960) from the National Institute on Deafness and Other Communication Disorders, Research Development Funds from the University of Pittsburgh’s School of Health and Rehabilitation Sciences, and the New Century Scholars Doctoral Scholarship from the American Speech-Language-Hearing Foundation.

The first author is grateful to dissertation committee members Susan Shaiman and James Coyle who contributed to the conception and design of this research and to the late statistician, Kevin Kim. The first author greatly appreciates the valuable contributions of F-31 co-mentors Ronald Scherer and Jessica Huber in planning the experiment and providing training in measurement techniques. He thanks Amanda Gillespie, Leah Helou, Adrianna

Shembel, and Aaron Pham and the team at the University of Pittsburgh Voice Center for involvement in recruiting and running participants. The first author also acknowledges Jack Wiedrick, MS, at Oregon Health & Science University for assistance with statistical analyses and, finally, Donna Graville, CCC-SLP, at Oregon Health & Science University for providing time and support to bring this dissertation research to publication. These data were presented at the Annual Convention of the American Speech-Language-Hearing Association, Denver, CO, last November 19–21, 2015.

References

- American College of Sports Medicine.** (2009). *American College of Sports Medicine's guidelines for exercise testing and prescription* (8th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.
- American College of Sports Medicine.** (2012). *American College of Sports Medicine's health/fitness facility standards and guidelines* (4th ed.). Champaign, IL: Human Kinetics.
- Arffa, R. E., Krishna, P., Gartner-Schmidt, J., & Rosen, C. A.** (2012). Normative values for the Voice Handicap Index-10. *Journal of Voice*, 26(4), 462–465.
- Bailey, E. F., & Hoit, J. D.** (2002). Speaking and breathing in high respiratory drive. *Journal of Speech, Language, and Hearing Research*, 45(1), 89–99.
- Baken, R. J., & Orlikoff, R. F.** (2000). *Clinical measurement of speech and voice* (2nd ed.). San Diego, CA: Singular.
- Baker, S. E., Hipp, J., & Alessio, H.** (2008). Ventilation and speech characteristics during submaximal aerobic exercise. *Journal of Speech, Language, and Hearing Research*, 51(5), 1203–1214.
- Ballinger, G. A.** (2004). Using generalized estimating equations for longitudinal data analysis. *Organizational Research Methods*, 7(2), 127–150.
- Blitzer, A., Brin, M. F., & Ramig, L. O.** (2009). *Neurologic disorders of the larynx* (2nd ed.). New York, NY: Thieme.
- Bourne, T., & Garnier, M.** (2012). Physiological and acoustic characteristics of the female music theater voice. *The Journal of the Acoustical Society of America*, 131(2), 1586–1594.
- Brancatisano, A., Dodd, D. S., & Engel, L. A.** (1991). Posterior cricoarytenoid activity and glottic size during hyperpnea in humans. *Journal of Applied Physiology*, 71(3), 977–982.
- Brancatisano, T., Collett, P. W., & Engel, L. A.** (1983). Respiratory movements of the vocal cords. *Journal of Applied Physiology*, 54(5), 1269–1276.
- Bunn, J. C., & Mead, J.** (1971). Control of ventilation during speech. *Journal of Applied Physiology*, 31(6), 870–872.
- Butler, J. E.** (2007). Drive to the human respiratory muscles. *Respiratory Physiology & Neurobiology*, 159(2), 115–126.
- Chobanian, A. V., Bakris, G. L., Black, H. R., Cushman, W. C., Green, L. A., Izzo, J. L., Jr., . . . National High Blood Pressure Education Program Coordinating Committee.** (2003). The seventh report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure: The JNC 7 report. *Journal of the American Medical Association*, 289(19), 2560–2572.
- Crapo, R. O., Morris, A. H., & Gardner, R. M.** (1981). Reference spirometric values using techniques and equipment that meet ATS recommendations. *The American Review of Respiratory Disease*, 123(6), 659–664.
- DeLeo LeBorgne, W., Lee, L., Stemple, J. C., & Bush, H.** (2010). Perceptual findings on the Broadway belt voice. *Journal of Voice*, 24(6), 678–689.
- Doust, J. H., & Patrick, J. M.** (1981). The limitation of exercise ventilation during speech. *Respiratory Physiology & Neurobiology*, 46(2), 137–147.
- England, S. J., & Bartlett, D., Jr.** (1982). Changes in respiratory movements of the human vocal cords during hyperpnea. *Journal of Applied Physiology*, 52(3), 780–785.
- Fairbanks, G.** (1960). *Voice and articulation drillbook* (2nd ed.). New York, NY: Harper & Row Publishers.
- Farrell, P. A., Joyner, M. J., & Caiozzo, V. J.** (2012). *American College of Sports Medicine's advanced exercise physiology* (2nd ed.). Philadelphia, PA: Lippincott Williams & Wilkins.
- Finnegan, E. M., Luschei, E. S., & Hoffman, H. T.** (2000). Modulations in respiratory and laryngeal activity associated with changes in vocal intensity during speech. *Journal of Speech, Language, and Hearing Research*, 43(4), 934–950.
- Fowler, C. A., Rubin, P., Remez, R. E., & Turvey, M. T.** (1980). Implications for speech production of a general theory of action. In B. Butterworth (Ed.), *Language production* (Vol. 1, pp. 373–420). New York, NY: Academic Press.
- Gillespie, A. I., Slivka, W., Atwood, C. W., Jr., & Verdolini Abbott, K.** (2015). The effects of hyper- and hypocapnia on phonatory laryngeal airway resistance in women. *Journal of Speech, Language, and Hearing Research*, 58, 638–652.
- Grillo, E. U., & Verdolini, K.** (2008). Evidence for distinguishing pressed, normal, resonant, and breathy voice qualities by laryngeal resistance and vocal efficiency in vocally trained subjects. *Journal of Voice*, 22(5), 546–552.
- Haahr, M.** (1998). *List randomizer*. Retrieved from <https://www.random.org/lists/>
- Harms, C. A.** (2006). Does gender affect pulmonary function and exercise capacity. *Respiratory Physiology & Neurobiology*, 151(2–3), 124–131.
- Helou, L. B., & Solomon, N. P.** (2011). *Data done right: Avoiding common mistakes in aerodynamics*, Voice Foundation Symposium, Philadelphia, PA, June 3 2011. New York, NY: Elsevier.
- Hixon, T. J., Goldman, M. D., & Mead, J.** (1973). Kinematics of the chest wall during speech production: Volume displacements of the rib cage, abdomen, and lung. *Journal of Speech and Hearing Research*, 16(1), 78–115.
- Hixon, T. J., Mead, J., & Goldman, M. D.** (1976). Dynamics of the chest wall during speech production: Function of the thorax, rib cage, diaphragm, and abdomen. *Journal of Speech and Hearing Research*, 19(2), 297–356.
- Hixon, T. J., Weismer, G., & Hoit, J. D.** (2008). *Preclinical speech science: Anatomy, physiology, acoustics, perception*. San Diego, CA: Plural.
- Hoffman-Ruddy, B., Lehman, J., Crandell, C., Ingram, D., & Sapienza, C.** (2001). Laryngostroboscopic, acoustic, and environmental characteristics of high-risk vocal performers. *Journal of Voice*, 15(4), 543–552.
- Hoit, J. D., Lansing, R. W., & Perona, K. E.** (2007). Speaking-related dyspnea in healthy adults. *Journal of Speech, Language, and Hearing Research*, 50(2), 361–374.
- Holmberg, E.** (1980). Laryngeal airway resistance as a function of phonation type. *The Journal of the Acoustical Society of America*, 68(S1), S101.
- Holmberg, E. B., Hillman, R. E., & Perkell, J. S.** (1988). Glottal airflow and transglottal air pressure measurements for male and female speakers in soft, normal, and loud voice. *The Journal of the Acoustical Society of America*, 84(2), 511–529.
- Holmberg, E. B., Hillman, R. E., & Perkell, J. S.** (1989). Glottal airflow and transglottal air pressure measurements for male and female speakers in low, normal, and high pitch. *Journal of Voice*, 3(4), 294–305.
- Holmberg, E. B., Hillman, R. E., Perkell, J. S., & Gress, C.** (1994). Relationships between intra-speaker variation in aerodynamic measures of voice production and variation in SPL

- across repeated recordings. *Journal of Speech and Hearing Research*, 37(3), 484–495.
- Huber, J. E., Stathopoulos, E. T., Bormann, L. A., & Johnson, K. (1998). Effects of a circumferentially vented mask on breathing patterns of women as measured by respiratory kinematic techniques. *Journal of Speech, Language, and Hearing Research*, 41, 472–478.
- Hunter, E. J., Tanner, K., & Smith, M. E. (2011). Gender differences affecting vocal health of women in vocally demanding careers. *Logopedics, Phoniatrics, Vocology*, 36(3), 128–136.
- Jürgens, U. (2009). The neural control of vocalization in mammals: A review. *Journal of Voice*, 23(1), 1–10.
- KayPENTAX (2009). *Phonatory Aerodynamic System Model 6600 instruction manual*. Lincoln Park, NJ: KayPentax Medical Co.
- Kelly, L. E. (Ed.) (2006). *Adapted physical education national standards* (2nd ed.). Champaign, IL: Human Kinetics.
- Kempster, G. B., Gerratt, B. R., Verdolini Abbott, K., Barkmeier-Kraemer, J., & Hillman, R. E. (2009). Consensus auditory-perceptual evaluation of voice: Development of a standardized clinical protocol. *American Journal of Speech-Language Pathology*, 18(2), 124–132.
- Lee, L., Friesen, M., Lambert, I. R., & Loudon, R. G. (1998). Evaluation of dyspnea during physical activity and speech activities in patients with pulmonary diseases. *Chest*, 113(3), 625–632.
- Leeper, H. A., Jr., & Graves, D. K. (1984). Consistency of laryngeal airway resistance in adult women. *Journal of Communication Disorders*, 17(3), 153–163.
- Ludlow, C. L. (2005). Central nervous system control of the laryngeal muscles in humans. *Respiratory Physiology & Neurobiology*, 147(2–3), 205–222.
- Ludlow, C. L. (2011). Central nervous system control of interactions between vocalization and respiration in mammals. *Head & Neck*, 33(Suppl. 1), S21–S25.
- McArdle, W. D., Katch, F. I., & Katch, V. L. (2010). *Exercise physiology: Nutrition, energy, and human performance* (7th ed.). Baltimore, MD: Lippincott Williams & Wilkins.
- Meckel, Y., Rotstein, A., & Inbar, O. (2002). The effects of speech production on physiologic responses during submaximal exercise. *Medicine and Science in Sports and Exercise*, 34(8), 1337–1343.
- Miller, M. R., Hankinson, J., Brusasco, V., Burgos, F., Casaburi, R., Coates, A., ... ATS/ERS Task Force. (2005). Standardisation of spirometry. *The European Respiratory Journal*, 26(2), 319–338.
- National Heart, Lung and Blood Institute, Obesity Education Initiative, Expert Panel. (1998). *Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: The evidence report*. Rockville, MD: U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Heart, Lung and Blood Institute.
- Otis, A. B., & Clark, R. G. (1968). Ventilatory implications of phonation and phonatory implications of ventilation. *Annals of the New York Academy of Sciences*, 155, 122–128.
- Parkes, M. J. (2006). Breath-holding and its breakpoint. *Experimental Physiology*, 91(1), 1–15.
- Pennestri, E., Cavacece, M., & Vita, L. (2005). *On the computation of degrees-of-freedom: A didactic perspective*, American Society of Mechanical Engineers' 2005 International Design Engineering Technical Conferences and Computers and Information in Engineering Conference, San Diego, CA, September 24–28 2005. New York, NY: ASME Technical Publishing Office.
- Peterson, K. L., Verdolini-Marston, K., Barkmeier, J. M., & Hoffman, H. T. (1994). Comparison of aerodynamic and electroglottographic parameters in evaluating clinically relevant voicing patterns. *Annals of Otology, Rhinology & Laryngology*, 103(5, Pt. 1), 335–346.
- Reed, E. S. (1982). An outline of a theory of action systems. *Journal of Motor Behavior*, 14(2), 98–134.
- Rosen, C. A., Lee, A. S., Osborne, J., Zullo, T., & Murry, T. (2004). Development and validation of the Voice Handicap Index-10. *The Laryngoscope*, 114(9), 1549–1556.
- Rotstein, A., Meckel, Y., & Inbar, O. (2004). Perceived speech difficulty during exercise and its relation to exercise intensity and physiological responses. *European Journal of Applied Physiology*, 92(4–5), 431–436.
- Russell, B. A., Cerny, F. J., & Stathopoulos, E. T. (1998). Effects of varied vocal intensity on ventilation and energy expenditure in women and men. *Journal of Speech, Language, and Hearing Research*, 41(2), 239–248.
- Sandage, M. J., Connor, N. P., & Pascoe, D. D. (2013). Voice function differences following resting breathing vs. submaximal exercise. *Journal of Voice*, 27(5), 572–578.
- Sellers, I., & Sellers, S. (1983). Cricothyroid joint structure and function. *Journal of Laryngology and Otology*, 97(11), 1027–1034.
- Seventh String Limited. (2009). The Seventh String Tuning Fork [Software]. Retrieved from <http://www.seventhstring.com/tuningfork/tuningfork.html>
- Sheel, A. W., & Guenette, J. A. (2008). Mechanics of breathing during exercise in men and women: Sex versus body size differences. *Exercise and Sport Sciences Reviews*, 36(3), 128–134.
- Sivasankar, M., & Erickson, E. (2009). Short-duration accelerated breathing challenges affect phonation. *The Laryngoscope*, 119(8), 1658–1663.
- Smith, J. C., & Mead, J. (1986). Three degree of freedom description of movement of the human chest wall. *Journal of Applied Physiology*, 60(3), 928–934.
- Smitheran, J. R., & Hixon, T. J. (1981). A clinical method for estimating laryngeal airway resistance during vowel production. *Journal of Speech and Hearing Disorders*, 46(2), 138–146.
- StataCorp. (2017). Stata statistical software: Release 15 [Software]. College Station, TX: StataCorp LLC.
- Stathopoulos, E. T., & Sapienza, C. (1993). Respiratory and laryngeal function of women and men during vocal intensity variation. *Journal of Speech and Hearing Research*, 36(1), 64–75.
- Stone, R. E., Jr., Cleveland, T. F., Sundberg, P. J., & Prokop, J. (2003). Aerodynamic and acoustical measures of speech, operatic, and Broadway vocal styles in a professional female singer. *Journal of Voice*, 17(3), 283–297.
- Titze, I. R. (1988). The physics of small-amplitude oscillation of the vocal folds. *The Journal of the Acoustical Society of America*, 83(4), 1536–1552.
- Titze, I. R. (2000). *Principles of voice production*. Englewood Cliffs, NJ: Prentice-Hall.
- Titze, I. R. (2002). Regulating glottal airflow in phonation: Application of the maximum power transfer theorem to a low dimensional phonation model. *The Journal of the Acoustical Society of America*, 111(1, Pt. 1), 367–376.
- Verdolini, K., Druker, D. G., Palmer, P. M., & Samawi, H. (1998). Laryngeal adduction in resonant voice. *Journal of Voice*, 12(3), 315–327.
- von Elm, E., Altman, D. G., Egger, M., Pocock, S. J., Gøtzsche, P. C., Vandenbroucke, J. P., & STROBE Initiative. (2008). The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: Guidelines for reporting observational studies. *Journal of Clinical Epidemiology*, 61(4), 344–349.

-
- von Leden, H., & Moore, P.** (1961). The mechanics of the cricoarytenoid joint. *Archives of Otolaryngology*, 73(5), 541–550.
- Wilson, J. V., & Leeper, H. A.** (1992). Changes in laryngeal airway resistance in young adult men and women as a function of vocal sound pressure level and syllable context. *Journal of Voice*, 6(3), 235–245.
- Zeger, S. L., & Liang, K. Y.** (1986). Longitudinal data analysis for discrete and continuous outcomes. *Biometrics*, 42(1), 121–130.
- Zeger, S. L., Liang, K. Y., & Albert, P. S.** (1988). Models for longitudinal data: A generalized estimating equation approach. *Biometrics*, 44(4), 1049–1060.