Published in final edited form as:
J Commun Disord. 2014 ; 48: 1–17. doi:10.1016/j.jcomdis.2013.12.001.

Increased Vocal Intensity due to the Lombard Effect in Speakers with Parkinson's Disease: Simultaneous Laryngeal and Respiratory Strategies

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Abstract

Purpose—The objective of the present study was to investigate whether speakers with hypophonia, secondary to Parkinson's disease (PD), would increase their vocal intensity when speaking in a noisy environment (Lombard Effect). The other objective was to examine the underlying laryngeal and respiratory strategies used to increase vocal intensity.

Methods—Thirty-three participants with PD were included for study. Each participant was fitted with the SpeechVive™ device that played multi-talker babble noise into one ear during speech. Using acoustic, aerodynamic and respiratory kinematic techniques, the simultaneous laryngeal and respiratory mechanisms used to regulate vocal intensity were examined.

Results—Significant group results showed that most speakers with PD (26/33) were successful at increasing their vocal intensity when speaking in the condition of multi-talker babble noise. They were able to support their increased vocal intensity and subglottal pressure with combined strategies from both the laryngeal and respiratory mechanisms. Individual speaker analysis indicated that the particular laryngeal and respiratory interactions differed among speakers.

Conclusions—The SpeechVive™ device elicited higher vocal intensities from patients with PD. Speakers used different combinations of laryngeal and respiratory physiologic mechanisms to increase vocal intensity, thus suggesting that disease process does not uniformly affect the speech subsystems.

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Keywords

vocal intensity; Parkinson's disease; Lombard effect; respiratory; laryngeal

1.0 INTRODUCTION

Speech production is continuously modulated during everyday activities to meet the needs of the communicative environment, such as personal conversations, speaking to a small group, and conversing in noisy environments. When confronted with these diverse tasks, speakers must be able to adjust and control their vocal intensity.

Typical speakers change their breathing patterns in order to more efficiently produce the subglottal air pressures (P_S) needed to increase vocal intensity (Finnegan, Luschei, & Hoffman, 2000; Huber, 2007; Huber, Chandrasekaran, & Wolstencroft, 2005; Winkworth & Davis, 1997). In healthy individuals , combined passive and active forces are used to finely control P_S to regulate vocal intensity (Stathopoulos & Sapienza, 1997). Specifically, typical speakers expand the thorax (inhale to higher lung and rib cage volumes) to take advantage of greater elastic recoil forces, thereby limiting the amount of thoracic muscle contraction needed to increase P_S (Hixon, Goldman, & Mead, 1973; Huber, 2007). Further, typical speakers may increase their abdominal muscle contraction, as seen by smaller abdominal volumes during loud speech, to help maintain thoracic resistance and alveolar pressures (Hixon et al., 1973; Hoit, Plassman, Lansing, & Hixon, 1988), but this has not been reported consistently (Huber, 2007; Stathopoulos & Sapienza, 1997). Hereafter, the use of higher lung and rib cage volume ranges and potentially smaller abdominal volumes during increased vocal intensity is considered to be "improved respiratory support."

The pathophysiology of Parkinson's disease (PD) substantially affects the function of motor systems in the body (Wichmann & DeLong, 1996). In addition to the limb motor system, the disease process affects the respiratory motor system. It has been demonstrated that both inspiratory and expiratory muscle impairments in patients with PD, more specifically decreased strength or poor coordination of respiratory muscles (de Bruin, de Bruin, Lees, & Pride, 1993; Haas, Trew, & Castle, 2004), may contribute to decreased expiratory flows (Bogaard, Hovestadt, Meerwaldt, Meche, & Stigt, 1989). The speech and voice deficits commonly reported in PD can also be attributed to deficits in neuromuscular function (Darley, Aronson, & Brown, 1969; Duffy, 2005). For example, muscular rigidity, a hallmark symptom of PD, has been suggested to contribute to the observed reduction in pulmonary function (Solomon & Hixon, 1993). These authors suggest that a breakdown in the synergistic force of the muscles of the rib cage and abdomen could contribute to inefficient breathing patterns during the speech production of individuals with PD. For example, individuals with PD have shown evidence of oppositional movement of rib cage and abdomen during expiration (Solomon & Hixon, 1993), and more variable lung volumes than healthy speakers (Huber, Stathopoulos, Ramig, & Lancaster, 2003). In addition, speakers with PD often have more difficulty controlling their vocal intensity (Sadagopan & Huber, 2007), and also planning in advance to support longer utterances (Bunton, 2005), particularly during extemporaneous speech (Huber & Darling, 2011). In summary, due to

muscle rigidity, reduced muscle strength, and difficulties coordinating respiratory movements when speaking, individuals with PD are likely to be challenged when responding to everyday communicative demands, in particular increasing vocal intensity.

While the respiratory system has been identified as an important contributor to increased vocal intensity, it does not act in isolation. The laryngeal mechanism also plays an important role in adjustments of intensity. Laryngeal strategies to increase vocal intensity are achieved through changes to glottal shape which in turn affects the resistance to glottal airflow (Isshiki, 1964). Changes to glottal shape can be observed by utilizing glottal airflow measures (Holmberg, Hillman, & Perkell, 1988; Stathopoulos & Sapienza, 1997). Using a noninvasive circumferentially-vented pneumotachograph mask, the oral airflow (V_o) waveform during speech can be inverse filtered to obtain a derived glottal airflow waveform (Rothenberg, 1973). During typical speech production by a broad age range of males and females, glottal aerodynamic events were shown to change with increased vocal intensity (Stathopoulos & Sapienza, 1997). Specifically, open quotient (OQ) decreased reflecting that the vocal folds were in a closed state for a greater part of the entire cycle. Peak-to-peak glottal airflow increased, reflecting that the amplitude of vocal fold vibration increased. Minimum glottal airflow decreased during the “closed” part of the cycle, indicating that there was increased vocal fold adduction. Last, maximum flow declination rate (MFDR), which reflects speed of vocal fold closure, increased at higher vocal intensity levels (Stathopoulos & Sapienza, 1997). These changes in laryngeal function help to achieve greater levels of P_s thereby improving laryngeal “support” at higher vocal intensity levels. In summary, adjustments in vocal intensity are regulated by simultaneous changes to both the respiratory and laryngeal systems (Stathopoulos & Sapienza, 1997).

Laryngeal muscle function during speech and non-speech activities is also affected by the pathophysiology of PD. For example, a recent EMG study showed that during rest and vocalization, 73% of participants with PD presented with increased laryngeal muscle activity, thus possibly reflecting hypertonicity and muscle rigidity of the laryngeal musculature (Zarzur, Duprat, Shinzato, & Eckley, 2007). Increased levels of laryngeal muscle activity have also been associated with vocal fold bowing (Gallena, Smith, Zeffiro, & Ludlow, 2001).

Differences in glottal shape offer insight into problems with motor function of the laryngeal muscles. Videostroboscopic studies have confirmed the presence of vocal fold bowing, increased glottal opening over the entire period of vibration, abnormal phase closure, and asymmetric vibratory patterns during phonation by individuals with PD (Dromey, Ramig, & Johnson, 1995; Hanson, Gerratt, & Ward, 1984; Perez, Ramig, Smith, & Dromey, 1996; Smith, Ramig, Dromey, Perez, & Samandari, 1995). These changes to the laryngeal mechanism are likely to impact the ability of individuals with PD to increase the vocal fold resistance and adduction forces necessary for producing high intensity speech.

Despite problems with muscle function, some patients with PD increased vocal intensity after voice treatment (LSVT®), showing improvements in the speech mechanism. In a single subject study of one male with PD, acoustic and aerodynamic data indicated increased vocal intensity and improved laryngeal function following one month of treatment (LSVT®)

(Dromey et al., 1995). Similarly, a laryngeal imaging study demonstrated improved vocal fold adduction post-LSVT® in a group of patients with PD (Smith et al., 1995). More recently, Baumgartner, Sapir, and Ramig, (2001) have shown that perceptual characteristics of breathiness and hoarseness were ameliorated after the LSVT® program, also implying improved laryngeal muscle function. Similar improvements have also been reported in studies of respiratory function in populations with neurological illness. For example, individuals with PD have been shown to use more effective respiratory support patterns when higher intensity speech is elicited with background noise (Huber & Darling, 2011; Sadagopan & Huber, 2007). To date, however, individuals with PD have not been examined for simultaneous adjustments of the respiratory and laryngeal mechanisms to increase vocal intensity.

Research on the use of external cueing such as producing speech in noise to increase vocal intensity, the Lombard effect, has shown promising results (Pick, Siegel, Fox, Garber, & Kearney, 1989). The Lombard effect has been used successfully to elicit nonvolitional higher vocal intensity levels from typical young and older adult speakers (Ho, Bradshaw, Iansek, & Alfredson, 1999; Huber, 2007, 2008; Huber et al., 2005; Winkworth & Davis, 1997). Winkworth and Davis (1997) propose that the increased vocal intensity levels are more likely to resemble changes to vocal intensity under naturally occurring conditions. The Lombard effect may be a useful technique for eliciting higher vocal intensity in individuals with PD who have problems with hypophonia (Adams & Lang, 1992). Sadagopan and Huber (2007) looked at the effect of loudness cues on the respiratory strategies used by individuals with PD. Results of the study showed that under a condition of background noise, individuals with PD demonstrated a greater increase in sound pressure level (SPL) (an acoustic measure of vocal intensity) and more efficient respiratory patterns than when cued in more traditional ways to increase loudness (talking twice as loud as comfortable or targeting a specific SPL on a SPL meter). Sadagopan and Huber's (2007) data confirm the usefulness of natural or external cueing in the treatment of hypophonia.

While there are now numerous investigations identifying the speech and voice characteristics of individuals with PD, there are scarce data examining the underlying mechanisms contributing to voice and speech impairments. We do not have a clear understanding of the strategies speakers with PD are capable of using to increase vocal intensity. The goal of the current study is to examine the interrelated function of the laryngeal and respiratory systems while individuals with PD naturally increase their vocal intensity in a condition of background noise.

The general hypothesis of the present investigation is that when participants with PD increase vocal intensity, underlying laryngeal and respiratory strategies will change.

1.1. Acoustic Changes

It is hypothesized that speakers will increase vocal intensity, measured acoustically by SPL, when speaking in a condition of multi-talker babble noise. SPL will be used interchangeably with vocal intensity (Titze, 1994).

1.2. Laryngeal Aerodynamic Changes

Improved laryngeal function will be reflected through several glottal aerodynamic measures indicating better control of subglottal pressure (P_s), increased amplitude of vocal fold vibration, and improved vocal fold adduction.

1.3. Respiratory Kinematic and Air Volume Changes

Concomitant support from the respiratory system during increased vocal intensity will result in increased lung and rib cage volume ranges. In addition, smaller abdominal volume ranges will indicate greater abdominal muscle support during the higher vocal intensity speech task.

2.0 METHODS

2.1. Participants

Data collection procedures were approved by committees on the Use of Human Research Participants at both Purdue University and the University at Buffalo. Thirty-three adults with PD, 6 women and 27 men, participated in the study. The mean age (with standard deviation) of the women was 75.50 (7.82) years, and the mean age of the men was 69.07 (10.05) years. Criteria for inclusion were: 1) diagnosis of idiopathic PD by a neurologist; 2) diagnosis of hypophonia; 3) speaker of Standard American English; 4) no history of other neurological diseases other than PD; 5) no history of respiratory problems; 6) no history of head, neck, or chest surgery except for implantation of a deep-brain stimulator (DBS); 7) no history of smoking in the past 5 years; and 8) no bilateral hearing aid use in order to accommodate device fitting to one ear; unilateral hearing aid accepted.

Table 1 includes a description of participant characteristics including sex, age, time since diagnosis, Hoehn and Yahr stage, severity of hypophonia, better-ear pure tone hearing thresholds, medications related to PD, and history of behavioral speech and voice therapy. Three patients had previously received a DBS to the subthalamic nucleus; their participant numbers are starred in Table 1. None of the participants were receiving behavioral treatment for speech or voice at the time of the study. All patients were tested during the “on” state of their medication cycle.

A certified speech-language pathologist who was unaffiliated with the study ascertained the diagnosis of hypophonia. Hypophonia was defined as soft speech noted during conversational speech and/or patient/caregiver complaint of problems communicating.

An audiologist unaffiliated with the study completed audiological evaluations to document hearing thresholds. The ear with better hearing sensitivity was determined by air conduction pure tone testing. The pure tone averages for the better ear are reported in Table 1. Noise was presented to that better ear through a small hearing-aid type speaker called a “SpeechVive™” (Section 2.2.1. Device for Eliciting the Lombard Effect).

2.2. Equipment

2.2.1 Device for Eliciting the Lombard Effect—A specially engineered device (SpeechVive™) was used to present multi-talker babble (Auditec of St. Louis), which

sounds like unintelligible background talking. The SpeechVive™ detected the voice waveform from an accelerometer placed near the sternal notch of the participant's throat. When the amplitude of the waveform from the accelerometer increased to a preset level, the SpeechVive™ played multi-talker babble. The multi-talker babble was presented through a small hearing-aid-type speaker and was delivered to the participant's ear through an open-ear fitting (Phonak Fit n'Go) to ensure there was no occlusion effect.

2.2.2. Acoustic Recordings—The acoustic waveform was transduced via a head-mounted omnidirectional condenser microphone (Shure Beta 53 Model # WBH53B; Countryman E6 Model # E610P5L2). For each participant, the same microphone was used for all recordings. The microphone waveform was digitally recorded at a frequency of 44.1 kHz using a digital audio recorder (Marantz PMD-670). Using GoldWave (Version 5.25, 2008), the waveform was resampled at a rate of 22 kHz with a low-pass filter of 9 kHz applied for anti-aliasing. The microphone was calibrated on days of testing to a known sound pressure level (either 94 or 110 dB SPL) using a piston phone (Quest, CA22) set to 1000 Hz. The head-mounted microphone waveform was digitized in synchronization with the aerodynamic and kinematic waveforms using LabChart (AD Instruments, version 7.1.2).

2.2.3. Laryngeal Aerodynamic Recordings—Intraoral Air Pressure (P_o). Intraoral air pressure was used to estimate subglottal pressure (P_s). Intraoral air pressure was sensed with a pitot tube (a small hollow polyethylene tube) and transduced with a Glottal Enterprises PTL-1 transducer. The waveform was amplified with the Glottal Enterprises amplifier (MS100-A2). Air pressure data were acquired using LabChart (AD Instruments, Version 7.1.2). Weekly air pressure calibrations were derived from a Glottal Enterprises MCU-4 Pneumotach Calibration Unit.

Oral Airflow (V_o). The oral airflow waveform was captured using a circumferentially-vented pneumotachograph mask (Glottal Enterprises, CV Mask) and was used to make an estimate of glottal airflow (V_g). The mask size used (medium or large) was dependent upon each participant's face size. Oral airflow was transduced using the Glottal Enterprises PTW-1 transducer and was amplified using the Glottal Enterprises amplifier (MS100-A2). Data were acquired using LabChart. The medium and large masks were calibrated using the Glottal Enterprises model MCU-4 Pneumotach calibration unit once per week. To account for electronic drift of the equipment, a referent 0 liters per second (L/s) period was digitized just before the mask was placed on the participant's face for the speech tasks.

2.2.4. Respiratory Kinematic and Air Volume Recordings—Rib cage (RC) and abdominal (AB) movements were transduced with the Inductotrace System (Series # 10.9000, Ambulatory Monitoring) which uses inductive plethysmography. An elastic band housing the inductance coil (respiband) was placed around the RC, inferior to the axilla, to transduce movements of the RC. A second respiband was placed around the abdomen at the level of the umbilicus, ensuring that it was below the last rib, to transduce movements of the AB. Respiratory kinematic waveforms were recorded and digitized at 2,000 Hz using LabChart. A Future Med Discovery-2 Spirometer was used to measure vital capacity (VC) and forced vital capacity. A VacuMed Universal Ventilation Meter (UVM) was used to collect air volume data during the respiratory calibration tasks.

2.3. Procedures

2.3.1. Eliciting the Lombard Effect—All speech task data were collected in one session (acoustic, laryngeal aerodynamic, and respiratory) first without the SpeechVive™ (speech-in-quiet condition), and then with the SpeechVive™ in place (speech-in-noise condition). The multi-talker babble has been shown to naturally elicit louder speech due to the Lombard effect (Garnier, Henrich, & Dubois, 2010).

First, the experimenter determined each participant's vocal intensity by instructing them to speak at comfortable loudness and pitch for 2 minutes (speech-in-quiet). Sound pressure levels were noted from an SPL meter placed at a mouth to microphone distance of 30.5 cm. Then, the SpeechVive™ open-fitting ear mold was placed into the participant's better ear as determined by hearing threshold results. Noise was presented to that better ear through a small hearing-aid type speaker. The detection level was adjusted by the experimenter until the SpeechVive™ activated and deactivated at the onset and offset of speech. The amplitude of the multi-talker babble was then increased during conversational speech until each participant spoke approximately 3 dB above his/her own comfortable SPL (speech-in-noise).

2.3.2. Acoustic Recordings—During a monologue speech task, the head-mounted microphone was placed 6 cm from the center of the lips at about a 45° angle from the midline of the lips. Care was taken so that the microphone did not contact the participant's cheek.

2.3.3. Laryngeal Aerodynamic Recordings—In order to make estimates of subglottal pressure and glottal airflow from intraoral air pressure and oral airflow, participants were instructed to say the sentence "Buy pop or pop a papa." Sentence productions were modeled for each participant so that they were relatively monotone compared to natural speech.

Intraoral air pressure was obtained during the sentence, which was loaded with the voiceless stop /p/ in order to make estimates of subglottal air pressure. Three to five sentence repetitions were collected until two to three acceptable productions were recorded. Acceptable productions were defined as those in which participants: 1. Produced the voiceless stop /p/. 2. Maintained velopharyngeal closure and tight lip closure around a pitot tube place in the participant's oral cavity as monitored by simultaneous 0 oral airflow during the stop closure duration. 3. Produced the sentence at a slow rate (to achieve broad topped intraoral air pressure pulses) and in a monotone voice (to attain each intraoral air pressure pulse in a stressed syllable context).

2.3.4. Calibration: Respiratory Kinematic, and Air Volume—To obtain both maximal lung volume (LV) and rib cage (RC) capacity for each participant, vital capacity maneuvers were used and performed at least three times with the respibands in place. All lung volumes were expressed as a percentage of vital capacity (%VC) based on the vital capacity maneuver. Maximal abdominal (AB) capacity maneuvers were attempted (Hoit & Hixon, 1987), but the majority of participants could not complete the maximum abdomen-out maneuver. The maximum abdomen-out maneuver requires coordination of diaphragm contraction and abdominal muscle relaxation; this coordination appears to be difficult for participants with PD. Thus, maximum AB capacity could not be determined. Therefore, to

maintain a parallel measurement schema, both RC and AB data are reported in volts (V) displacement.

In order to estimate lung volume displacement from combined movements of the RC and AB during speech (Konno & Mead, 1967), the least squares method of calibration was used (Chadha et al., 1982; Sackner et al., 1989). These calibration procedures have been described previously (Huber et al., 2005; Huber & Darling, 2011). Rib cage, abdomen, and spirometer waveforms were collected during two 45-s periods of rest breathing, during which time participants were instructed to “relax”, and during two 45-s periods of "speech-like" breathing, for which participants were instructed to silentlyread a sentence on each exhalation.

In order to calibrate the sum of the RC and AB for LV, a correction factor was computed from the spirometer, RC, and AB waveforms during the rest breathing and “speech-like” breathing. The Moore-Penrose pseudoinverse function was used in Matlab (Mathworks, version 2010a) to solve for k_1 in the following formula for each set of RC, AB, and spirometer (SP) data points during the breathing tasks.

$$\mathrm{SP}=k_1(\mathrm{RC})+1(\mathrm{AB}) \quad [1]$$

This estimation of LV was verified by visually checking the LV waveform [k_1(RC) + 1(AB)] against the actual SP waveform for a "speech-like" breathing trial. Thereafter, the estimated LV waveform was computed for each point during the speech tasks using the correction factor (k_1) in the following formula.

$$\mathrm{LV}=k_1(\mathrm{RC})+1(\mathrm{AB}) \quad [2]$$

Prior to measurement, respiratory kinematic waveforms were low-pass filtered at 40 Hz.

2.4. Measurements (Figure 1)

2.4.1. Acoustic Measures

1. Sound Pressure Level (SPL). A naturalistic connected speech sample was obtained for acoustic measurement of SPL. Pauses longer than 150 msec. were identified from the acoustic waveform during the monologue, and these pauses served as utterance boundaries. Average sound pressure level was measured from the acoustic signal for each utterance using TF32 (Milenkovic, 2003). Calibration values from the microphone calibration were factored into the measurement of SPL.

2.4.2. Laryngeal Aerodynamic Measures

1. Estimated subglottal air pressure (P_s). Estimated P_s was measured from the sentence production “Buy pop or pop a papa.” The voiced plosive /b/was eliminated and the six voiceless bilabial stops were measured. Intraoral air pressure (P_o) was measured at the pressure peak of two adjacent /p/ productions

(P_{o1} and P_{o2}). Pharyngeal air pressure (P_{ph}) was defined as the intraoral pressure at mid-vowel between the two adjacent /p/ productions. Estimated P_s was calculated using the formula following Smitheran and Hixon (1981); and by applying the technique to an appropriate speech task (Demolin et al., 1997; Kitajima & Fujita, 1990; Löfqvist, Carlborg, & Kitzing, 1982):

$$\text{Estimated}P_s=((P_{o1}+P_{o2})-P_{ph})/2 \quad [3]$$

In order to make glottal airflow measures, the glottal airflow waveform was derived from the V_o waveform (Holmberg et al., 1988; Rothenberg, 1973; Stathopoulos & Sapienza, 1997) during the above sentence task. During the sentence, the average V_o waveform was first used to monitor 0 flow during the stop closure to ensure complete velopharyngeal and lip closure. Second, a Time-Frequency analysis program (TF32; Milenkovic, 2003) was used to inverse filter the original V_o waveform of each vowel in the sentence. The cursors were placed around a mid-portion of three /a/ vowels in the sentence “Buy pop or pop a papa.” The mid-portion of each vowel was selected through visual inspection using several criteria while working within TF32: 1) a properly inverse filtered waveform as seen by a smoothed, periodic appearing waveform, 2) a stable frequency and intensity trace, and 3) the presence of harmonic structure in the narrowband spectrographic display. From the V_g waveform, several measures were used which reflected glottal shape:

2. Peak-to-peak glottal airflow (PP V_g). Peak-to-peak glottal airflow was defined as the difference between the peak airflow for a cycle and the average airflow while the glottis was maximally closed.
3. Open quotient (OQ). Open quotient was defined as the fraction of the glottal cycle time that the glottis was open. Open quotient was calculated as open glottis time/total glottal cycle time.
4. Maximum flow declination rate (MFDR). Maximum flow declination rate was defined as the maximum difference between flow samples that occurred as the glottal pulse down-sloped during the closing part of the cycle.

2.4.3. Respiratory Kinematic and Air Volume Measures—A naturalistic connected speech sample was obtained for respiratory kinematic measures. In order to obtain enough samples for respiratory analysis, participants were asked to provide a two-minute monologue on a neutral topic of their choice.

Using the microphone acoustic waveform as a guide, utterance initiations and terminations were verified by observing voicing onset and offset for each breath group. Utterance excursions for LV, RC, and AB were calculated by subtracting each respective termination from initiation. Further, all kinematic measures were referenced to end-expiratory level (EEL) (Stathopoulos & Sapienza, 1997). To determine where EEL occurred for each participant, three consistent rest breaths were required before the start of each speech task. Three end expiratory points were located and average EEL was calculated for LV, RC volume, and AB volume. Utterance length was calculated by counting the number of syllables produced during each breath.

The following respiratory kinematic and air volume measures were made during the monologue speech task and were expressed relative to end-expiratory level: Lung Volume: 1. initiation (LVI), 2. termination (LVT), 3. excursion (LVE), Rib Cage Volume: 4. initiation (RCI), 5. termination (RCT), 6.excursion (RCE), Abdominal Volume: 7. initiation (ABI), 8. termination (ABT), and 9. excursion (ABE). Lung volume measures were also expressed as a percent of vital capacity.

2.5. Statistical Analysis

To establish inter-measurer reliability, the data from six men and two women were randomly chosen for remeasurement (total of eight participants). An analysis of variance (ANOVA) was used to examine differences across the two sets of measurements. All tests were statistically non-significant (Table 2).

If a participant did not have at least one good trial for measurement in both the speech-in-quiet and speech-in-noise conditions, they were excluded from data analysis for the affected measurements. When mask leaks or irregular waveforms precluded valid measurement of oral airflow, aerodynamic data from these trials were excluded from analysis. Oral airflow data could not be measured for six participants (M05, M11, M12, M19, M20, and M21). When participants did not achieve adequate lip closure or were unable to perform the task for the estimation of P_s, these trials were excluded from analysis. Subglottal pressure data could not be estimated for five participants (M05, M11, M19, M20, and M21).

A repeated measures analysis of variance (ANOVA) was used to examine whether the dependent variables significantly changed when the participants spoke with the SpeechVive™ off (speech-in-quiet) vs. the SpeechVive™ on (speech-in-noise) condition. Participant was included as a random effect in the model due to the expected inter-subject differences in response to the speech-in-noise condition and the differences across subjects in disease and hypophonia severity. The alpha level was set at $p<.05$.

3.0 RESULTS

Means and standard errors for all dependent variables in the speech-in-quiet and the speech-in-noise conditions are presented in Table 3.

3.1. Acoustic Measures (Monologue Task)

1. Sound Pressure Level (SPL). There was a significant increase in SPL when the participants spoke in noise as compared to speech-in-quiet [$F(1) = 253.74$, $p<.0001$]. There was a significant interaction between person and condition for SPL [$F(32) = 11.81$, $p<.0001$]. Twenty-six out of 33 individuals increased SPL when speaking in noise (Figure 2).

3.2. Laryngeal Aerodynamic Measures (Sentence Production Task)

1. Estimated subglottal air pressure (P_s) significantly increased when the participants spoke in noise as compared to speech-in-quiet [$F(1) = 77.09$, $p<.0001$]. There was a

significant interaction between person and condition [F(27) = 18.82, p<.0001]. Nineteen out of 28 individuals increased P_s when speaking in noise (Figure 3).

2. Peak-to-peak glottal airflow (PP V_g) significantly increased when participants spoke in noise as compared to speech-in-quiet [F(1) = 8.29, p=0.004]. There was a significant interaction between person and condition for (PP V_g) [F(26) = 5.28, p<0.0001]. Fifteen of 27 individuals increased (PP V_g) when speaking in noise (Figure 3).

3. Open quotient (OQ) significantly decreased when the participants spoke in noise as compared to speech-in-quiet [F(1) = 16.28, p<0.0001]. There was a significant interaction between person and condition for OQ [F(26) = 4.75, p<0.0001]. Twenty-one out of 27 individuals decreased OQ when speaking in noise (Figure 4).

4. Maximum flow declination rate (MFDR) significantly increased when the participants spoke in noise as compared to speech-in-quiet [F(1) = 28.49, p<0.0001]. There was a significant interaction between person and condition for MFDR [F(26) = 5.99, p<.0001]. Seventeen out of 27 individuals increased MFDR when speaking in noise (Figure 4).

3.3. Respiratory Kinematic and Air Volume Measures (Monologue)

3.3.1. Lung Volume Measures

1. There was a significant increase in lung volume initiation (LVI) when the participants spoke in noise as compared to speech-in-quiet [F(1) = 90.72, p<.0001]. There was a significant interaction between person and condition for LVI [F(32) = 26.28, p<.0001]. Nineteen out of 33 individuals increased LVI when speaking in noise (Figure 5).

2. There was a significant increase in lung volume termination (LVT) when the participants spoke in noise as compared to speech-in-quiet [F(1) = 74.53, p<.0001]. There was a significant interaction between person and condition for LVT [F(32) = 25.71, p<.0001]. Twenty out of 33 individuals increased LVT when speaking in noise (Figure 5). These are the same 19 participants who increased LVI in noise, with the addition of one speaker (M24).

3. There was no significant change in lung volume excursion (LVE) [F(1) = .16, p=.69] when the participants spoke in noise as compared to speech-in-quiet. There was a significant interaction between person and condition for LVE [F(32) = 2.33, p<.0001]. Seventeen out of 33 individuals increased LVE when speaking in noise.

3.3.2. Rib Cage Volume Measures

4. There was a significant increase in rib cage volume initiation (RCI) when the participants spoke in noise as compared to speech-in-quiet [F(1) = 90.03, p<.0001]. There was a significant interaction between person and condition for RCI [F(32) = 23.81, p<.0001]. Sixteen out of 33 individuals increased RCI when speaking in noise.

5. There was a significant increase in rib cage volume termination (RCT) when the participants spoke in noise as compared to speech-in-quiet [F(1) = 97.02, p<.0001]. There was a significant interaction between person and condition for RCT [F(32) = 21.27, p<.0001]. Sixteen out of 33 individuals increased RCT when speaking in noise. These are the same participants who increased RCI in noise.

6. There was no significant change in rib cage volume excursion (RCE) [F(1) = 1.69, p=.19] when the participants spoke in noise as compared to speech-in-quiet. There was a significant interaction between person and condition for RCE [F(32) = 2.12, p=.0003]. Sixteen out of 33 individuals increased RCE when speaking in noise.

3.3.3. Abdominal Volume Measures

7. There was a significant increase in abdominal volume initiation (ABI) when the participants spoke in noise as compared to speech-in-quiet [F(1) = 21.00, p<.0001]. There was a significant interaction between person and condition for ABI [F(32) = 22.62, p<.0001]. Nineteen out of 33 individuals increased ABI when speaking in noise.

8. There was a significant increase in abdominal volume termination (ABT) when the participants spoke in noise as compared to speech-in-quiet [F(1) = 8.23, p=.004]. There was a significant interaction between person and condition for ABT [F(32) = 26.16, p<.0001]. Nineteen out of 33 individuals increased ABT when speaking in noise.

9. There was no significant change in abdominal volume excursion (ABE) when the participants spoke in noise as compared to speech-in-quiet [F(1) = 2.17, p=.14]. There was a significant interaction between person and condition for ABE [F(32) = 2.05, p=.0005]. Seventeen out of 33 individuals increased ABE when speaking in noise.

3.3.4. Utterance Length

10. There was no significant change in utterance length [F(1) = 2.12, p=.15] when the participants spoke in noise as compared to speech-in-quiet. There was a significant interaction between person and condition for utterance length [F(32) = 3.04, p<.0001]. Seventeen out of 33 participants decreased utterance length when speaking in noise.

4.0 DISCUSSION

The purpose of this study was to examine whether the SpeechVive™ would automatically elicit higher SPLs from participants with PD, and to examine the changes in laryngeal and respiratory function used to support higher SPLs. Additionally, individual speakers were examined to determine whether varied combinations of laryngeal and respiratory strategies were used to achieve higher intensity speech.

4.1. Acoustic Measures

Results showed that the SpeechVive™ successfully elicited significantly higher SPLs from the group of participants in this study (Figure 2).

4.2. Laryngeal Aerodynamic Adjustments to Increase Vocal Intensity

Laryngeal aerodynamic measures indicate improved laryngeal resistance when participants with PD increased their vocal intensity. The present group data showed significant improvements in P_s, PP V_g, OQ and MFDR when participants spoke in noise as compared to speech-in-quiet. The laryngeal aerodynamic results are consistent with previous work when individuals with PD increased their vocal intensity (Dromey et al., 1995; Ramig & Dromey, 1996; Smith et al., 1995). For example, Ramig and Dromey's (1996) investigation of 17 speakers with PD showed an LSVT® treatment effect for vocal intensity with concomitant increases in P_s, MFDR, and adduction quotient, but no significant decrease in OQ. In a qualitative videostroboscopy investigation, Smith et al. (1995) found that when individuals with PD increased their vocal intensity, it was judged that there was some evidence of improved glottal adduction and better glottal efficiency. In all, previous data confirm the supposition that increasing P_s, improving adduction, and sharpening the shape of the V_g waveform, will help individuals with PD to regulate increased vocal intensity.

Isshiki's (1964) classic article of one male speaker described the relationship between glottal resistance, glottal airflow, and vocal intensity. He summarized the three factors that determined vocal intensity: glottal resistance (P_s/airflow), glottal airflow, and glottal efficiency. Titze (1994) substantiates the three mechanisms and discusses the important relationships among lung pressure, glottal airflow, and vocal intensity. As vocal intensity increases, vocal efficiency consequently increases by "sharpening" the glottal airflow waveform. Isshiki's (1964) and Titze's (1994) discussions on OQ indicated that it was also an important efficiency variable when increasing vocal intensity. Decreased OQ and a narrowing of the glottal airflow waveform (faster opening and closing times) reflect the improved acoustic to aerodynamic efficiency. Vocal fold resistance is regulated by closing the vocal folds faster, and keeping them adducted for a greater length of time (i.e., more forceful contraction of laryngeal muscles). The data from our patients with PD substantiates the conclusion that higher intensity speech, elicited through the Lombard effect, results in more aerodynamic efficiency.

4.3. Respiratory Kinematic and Air Volume Adjustments for Increased Vocal Intensity

Speaking in noise also resulted in changes to respiratory function in the speakers with PD. Group data showed that, for higher vocal intensity, participants spoke at higher lung, RC, and AB volumes ranges without any significant changes in the concomitant excursions. Like typical speakers, individuals with PD shifted to higher lung and RC volume ranges to take advantage of the higher recoil pressures to generate higher P_s for higher SPLs. Conversely, it is notable that the larger abdominal volumes indicate that participants with PD display poor abdominal muscle control during speech.

Closer examination of the individual participant data using an x-y plot of movement of the RC vs. AB movement provides more detailed information on respiratory function for speech

(Figure 6). M20 is a good example of a speaker with PD who uses the respiratory system in a manner similar to normal speakers. When speaking at a higher vocal intensity, his LV and RC volume ranges were higher, and he also used AB muscles to support his speech respiratory function as observed by the abdominal position to the left of the abdominal end expiratory level (AB EEL is the vertical line at 0 in Figure 6). This leftward position indicates abdominal muscle contraction for both speech-in-quiet and speech-in-noise conditions. The notable fact about M20 is that we were not able to obtain glottal aerodynamic measures. The reason for this was that there was an unusual amount of noise in his voice indicating an inability to control vocal intensity through laryngeal resistance. His only alternative is to use the respiratory system to increase P_s. On the other hand, F06 shows a striking respiratory deviation from normal speakers. Her lung and rib cage volumes increase to take advantage of higher recoil pressures when speaking in noise, but the position of the abdomen is to the right of AB EEL (Figure 6). This larger abdominal position, to the right of AB EEL, for both conditions could only be accomplished through a passive response of the AB to contraction of the diaphragm. When the diaphragm contracts and flattens during inhalation prior to the speech utterance, it pushes downward and outward on the AB contents, thereby displacing the anterior muscular AB wall outward at utterance initiation/beginning of breath group exhalation (large triangles). With no active AB muscle contraction to counteract the displacement, the anterior wall will be pushed outward beyond the relaxed position. In short, this female speaker with PD was not able to use her AB muscles to support speech breathing.

The present data agree quite well with previous speech breathing data of speakers with PD. Solomon and Hixon (1993) reported that individuals with PD demonstrated significantly higher ABI and ABT values compared to healthy age-matched controls. They also hypothesized that weakness of the AB musculature may explain the greater than normal outward displacement of the AB during the inspiratory and expiratory cycles of speech breathing. Huber et al. (2003) extended the speech breathing results to individuals with PD when instructed to increase their vocal intensity. In the present study, individual speakers used varied respiratory strategies to increase their vocal intensity. For example, one participant with PD used higher lung and RC volumes to take advantage of higher recoil forces with little contribution from the AB, as demonstrated by speaker F06. Another participant, M20 used a combination of increased recoil forces by inhaling to larger lung and RC volumes, but also used more AB expiratory muscle support to produce higher SPLs.

More recently, Sadagopan and Huber (2007) reported respiratory function results for individuals with PD who naturally increased vocal intensity while speaking in noise. Participants with PD significantly increased their LVE, RCE, and ABE when they spoke at higher SPLs. Generally, the significant excursions were accomplished by slightly higher lung, RC, and AB initiations and terminations to take advantage of their natural recoil forces when speaking at higher SPLs. This mechanical adjustment of the chest wall indicates the use of a more efficient respiratory strategy while speaking at higher SPLs, similar to typical younger adults described in previous research (Hoit & Hixon, 1987; Huber, 2007; Stathopoulos & Sapienza, 1997).

Last, there were no changes in utterance length when the individuals spoke at higher vocal intensity, suggesting that the altered respiratory and laryngeal mechanisms allowed the speakers to maintain utterance length even while generating higher SPLs.

4.4. Individual Speaker Variation: Interactive Strategies to Increase Vocal Intensity

An important consideration in this investigation is participant variability. Table 4 allows examination of three additional participants' diverse laryngeal and respiratory strategies used to increase vocal intensity.

4.4.1. M01—M01 substantially increased SPL by 8 dB and P_s by 3 cm H_2O when speaking in noise (Figures 2 and 3 and Table 4 for summary). This speaker used a strong respiratory strategy when he increased SPL, with some laryngeal support. M01's MFDR increased reflecting a faster closing of the vocal folds (Figure 4). His open quotient and peak to peak flow changed slightly. From a respiratory perspective, M01 used both higher lung and rib cage volumes, helping him to generate higher P_s through his natural recoil pressures (Figures 5 and 6). Last, he also used his AB muscles to help support speech production; both of his expiratory "limbs" are to the left of AB EEL indicating AB muscle contraction during his speech utterances (Figure 6). Both speech-in-quiet and speech-in-noise were produced at approximately the same AB position. It was judged that M01 used strong respiratory support to increase his vocal intensity, but did not rely as much on active laryngeal valving. This pattern was seen in 7 of 33 participants.

4.4.2. F05—F05 increased SPL by 2.5 dB and P_s by 1.9 cm H20 (Figures 2 and 3) and used a combination of respiratory and laryngeal strategies to increase her vocal intensity. With the increased vocal intensity, her OQ decreased indicating an increase in glottal adduction/resistance (Figure 4). F05 also increased MFDR, suggesting a fine-tuning of the shape of the glottal airflow waveform to regulate increased vocal intensity (Figure 4). The respiratory kinematic measures show strong support through the use of higher lung and rib cage volumes (Figures 5 and 6). However, unlike M01, her larger abdominal volumes indicate that she had some difficulty maintaining AB support for speech at higher SPLs. Her AB moved outward (to the right) during speech-in-noise (Figure 6). This may be due to AB muscle weakness and difficulty in the face of more forceful diaphragmatic contractions for higher LVI in higher intensity speech. Overall, this speaker showed a combination of respiratory and laryngeal support for increasing vocal intensity. This interactional pattern was the most prevalent and occurred in 20 of our 33 participants.

4.4.3. M03—M03 increased SPL by 2.4 dB and P_s by .4 cm H_2O (Figures 2 and 3). This speaker used laryngeal support with very little respiratory support. M03 decreased his open quotient and increased his MFDR, which would reflect an increase in glottal resistance and improved vocal fold adduction. On the other hand, M03 showed weaker respiratory support by decreasing RC volume ranges and using abdominal volumes to the right of AB EEL (Figure 6). The respiratory data indicated that he did not take advantage of his natural recoil forces and that he could not control abdominal muscle contraction (Figures 5 and 6). The data suggest that the diaphragm drove the AB wall outward during inspiration. It is likely that this patient had both weak AB muscles and a stiffer RC. M03 seemed to be increasing

SPL primarily through increased laryngeal valving. This pattern occurred in 6 of our 33 participants.

4.5. Potential Clinical Implications

The importance of the success of the SpeechVive™ lies in the fact that most of our behavioral voice treatment approaches depend on conscious response from patients with PD. Currently, the most widely used tool for the treatment of hypophonia is the Lee Silverman Voice Treatment (LSVT®) program. LSVT® focuses on increasing vocal intensity through the completion of repetitive and intensive speech drills with a focus on increasing the patient's awareness of his/her vocal loudness levels (Fox, Morrison, Ramig, & Sapir, 2002). Therefore, similar to other voice therapy programs, LSVT® requires a conscious response from patients. However, for some patients with PD, generalization outside of the therapy room and in untrained speaking situations remains difficult, possibly due to the inherent sensory and cognitive problems associated with PD (Countryman & Ramig, 1993; Fox et al., 2002). The use of a reflexive response, such as the Lombard effect, to elicit increased vocal intensity may circumvent potential performance issues related to the associated sensory and cognitive deficits.

Examination of each participant's productions shows highly individualized physiological response for increasing vocal intensity. Some individuals seemed to maintain good capability of using a combination of both respiratory and laryngeal strategies for maintaining P_s and vocal intensity, much like typical speakers. On the other hand, there were individuals who seemed to display very poor use of the respiratory or laryngeal mechanism to increase P_s and vocal intensity. It should be observed from the participant table that the present participants with PD spanned a broad range of disease and hypophonia severity levels, and some were patients who had undergone DBS surgery. It is likely that patients at different disease stages and with DBS respond differently to treatment. The inclusion of patients across the spectrum of PD provides the opportunity for examination of a diverse sample of individuals with PD. Since we have used a within-subject design, each patient is compared to him or herself. Thus, while some of the results reflect the heterogeneity of our sample, the present data add to the empirical literature in its ability to deal with the general heterogeneous PD population.

Last, clinical investigations are under way to determine whether daily use of SpeechVive™ to elicit the Lombard Effect can provide an effective treatment for hypophonia related to Parkinson disease. The present positive results of eliciting higher vocal intensity in a one-session application gave strong evidence to plan a full treatment study. An 8-week training program (5 days per week for a minimum of 30 minutes per day) is currently being completed, along with a listener study to examine improvements in intelligibility.

4.6. Methodological Considerations

One of the strengths of the current investigation was that multiple speaking tasks were used to evaluate the speech of individuals with Parkinson's disease. One, a sentence repetition task was necessary in order to obtain estimated P_s and glottal aerodynamic data. And two, a monologue task, which was more representational of natural speech, was used to determine

increases to conversational SPL and the concomitant respiratory mechanism adjustments. Since we know that increases in P_s result in increased SPL during speech, participant performance was not entirely parallel across the two speech tasks. The caveat lies in the fact that when participants spoke in noise, there were some (6/33) who increased SPL during the monologue, but apparently decreased SPL during the sentence task (as implied by decreased P_s). For example, F01 increased her SPL during the monologue by about 4 dB (Figure 2), but she decreased her P_s by about 1.5 cm H2O during the sentence task (Figure 3). It's very likely that when she increased her SPL during the monologue, she also increased her P_s, but estimated P_s cannot be determined in a connected speech task. Yet, even with these methodological limitations in place, the P_s and SPL data follow one another for the majority of participants showing a robust effect across speech tasks.

4.7. Summary

The mean data from the present participants with PD show that speaking in noise while wearing the SpeechVive™ generally elicits both higher SPLs and P_s. Further, most participants were able to improve their underlying laryngeal and respiratory physiologic support. There was a substantial amount of individual variation in how participants increased their vocal intensity. Some participants increased vocal fold adduction and sharpened the glottal airflow waveform to support increased vocal intensity. At the respiratory level, some participants used higher lung and rib cage volumes to take advantage of recoil pressures to generate higher P_s and vocal intensities. Last, while some participants with PD were able to use the abdominal musculature to support increased vocal intensity, many of the participants showed significantly larger abdominal volumes at higher vocal intensities, demonstrating that many speakers with PD have significantly less abdominal muscle support than typical speakers.

Acknowledgements

This research was supported by Grant Number 5R01DC9409 funded by the National Institutes of Health, the National Institute on Deafness and Other Communication Disorders and a pilot grant from the Indiana Clinical and Translational Sciences Institute (CTSI, grant number TR000006) funded by the National Institutes of Health, National Center for Advancing Translational Sciences, Clinical and Translational Sciences Award. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institute on Deafness and Other Communication Disorders, the National Institutes of Health, or Indiana CTSI. We would also like to thank James Jones and Kirk Foster from the Biomedical Engineering Department at Purdue University for the technical expertise in the design and development of the SpeechVive™.

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Learning Outcomes

Readers will be able to:

1. identify speech characteristics of people with Parkinson's disease (PD)
2. identify typical respiratory strategies for increasing sound pressure level (SPL)
3. identify typical laryngeal strategies for increasing SPL
4. define the Lombard effect

CEU Questions

1. Increasing your vocal intensity when speaking in a noisy environment is called:
   - **a.** Bernoulli Effect
   - **b.** Lombard effect
   - **c.** Boyle's Law
   - **d.** Poisville's Effect
2. A typical respiratory mechanism for increasing vocal intensity is to breathe at:
   - **a.** Lower lung volumes
   - **b.** Mid lung volume ranges
   - **c.** Higher lung volumes
   - **d.** Same lung volumes as comfortable vocal intensity
3. Laryngeal factors which can help you increase your vocal intensity include:
   - **a.** Increased open quotient
   - **b.** Decreased maximum flow declination rate
   - **c.** Decreased open quotient
   - **d.** Increased maximum flow declination rate
   - **e.** C and D
4. A typical speech characteristic of individuals with Parkinson's disease includes:
   - **a.** Lower vocal intensities
   - **b.** Decreased vocal tremor
   - **c.** Hyponasality
   - **d.** Unusually high fundamental frequency
5. To increase vocal intensity, people with Parkinson's disease tend to use:
   - **a.** The same combination of laryngeal and respiratory strategies
   - **b.** Only the respiratory mechanism
   - **c.** Only the laryngeal mechanism
   - **d.** Diverse combinations of respiratory and laryngeal strategies

Answer key: 1 (b), 2 (c), 3 (e), 4 (a), 5 (d)

Highlights

- Individuals with Parkinson's disease often speak with lower vocal intensity
- Using the Lombard effect to increase vocal intensity is effective
- Speakers use diverse combinations of respiratory and laryngeal strategies to increase their vocal intensity

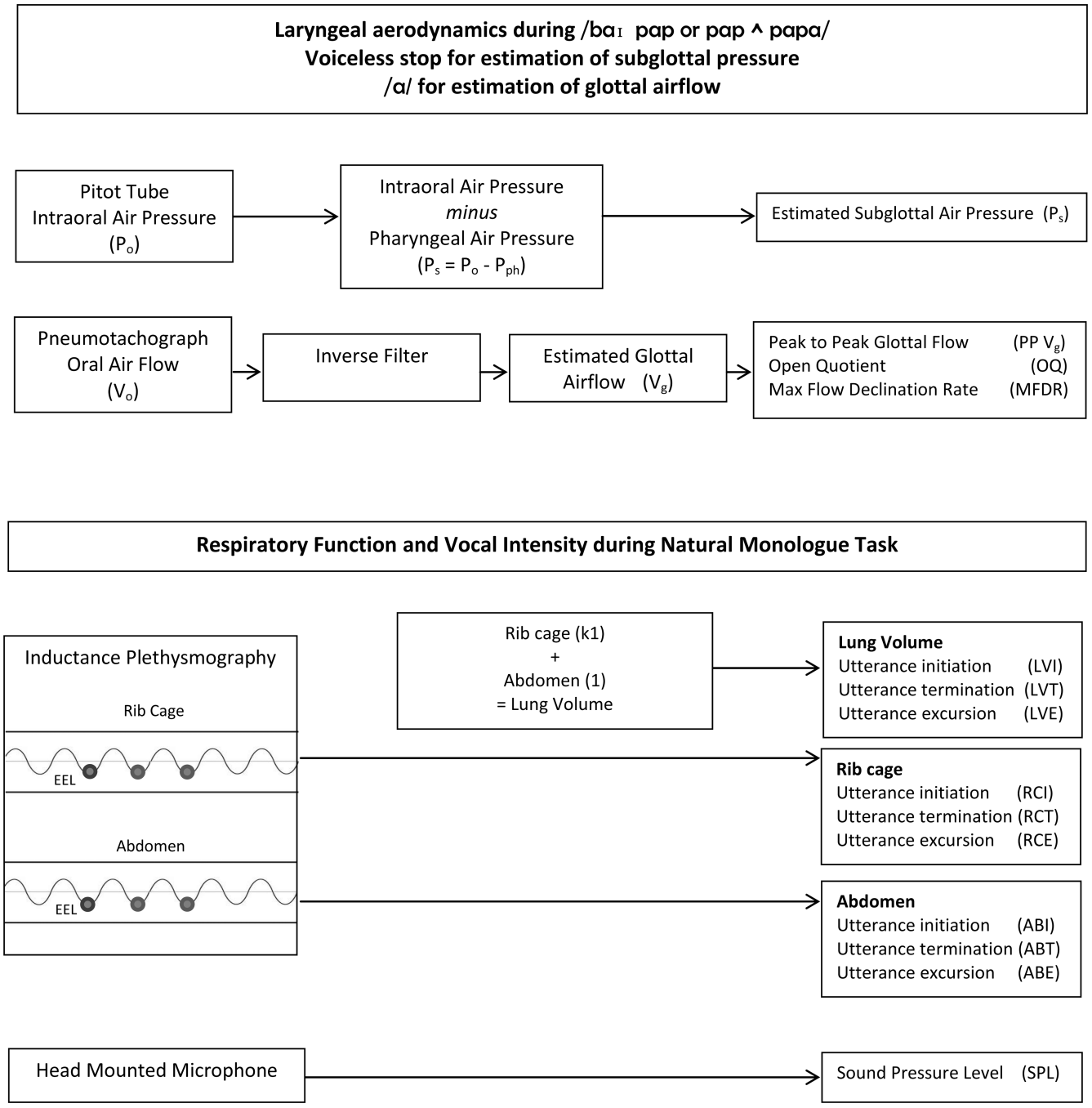


Figure 1.

Schematic of equipment and measurements for laryngeal aerodynamic, respiratory kinematic and acoustic recordings.

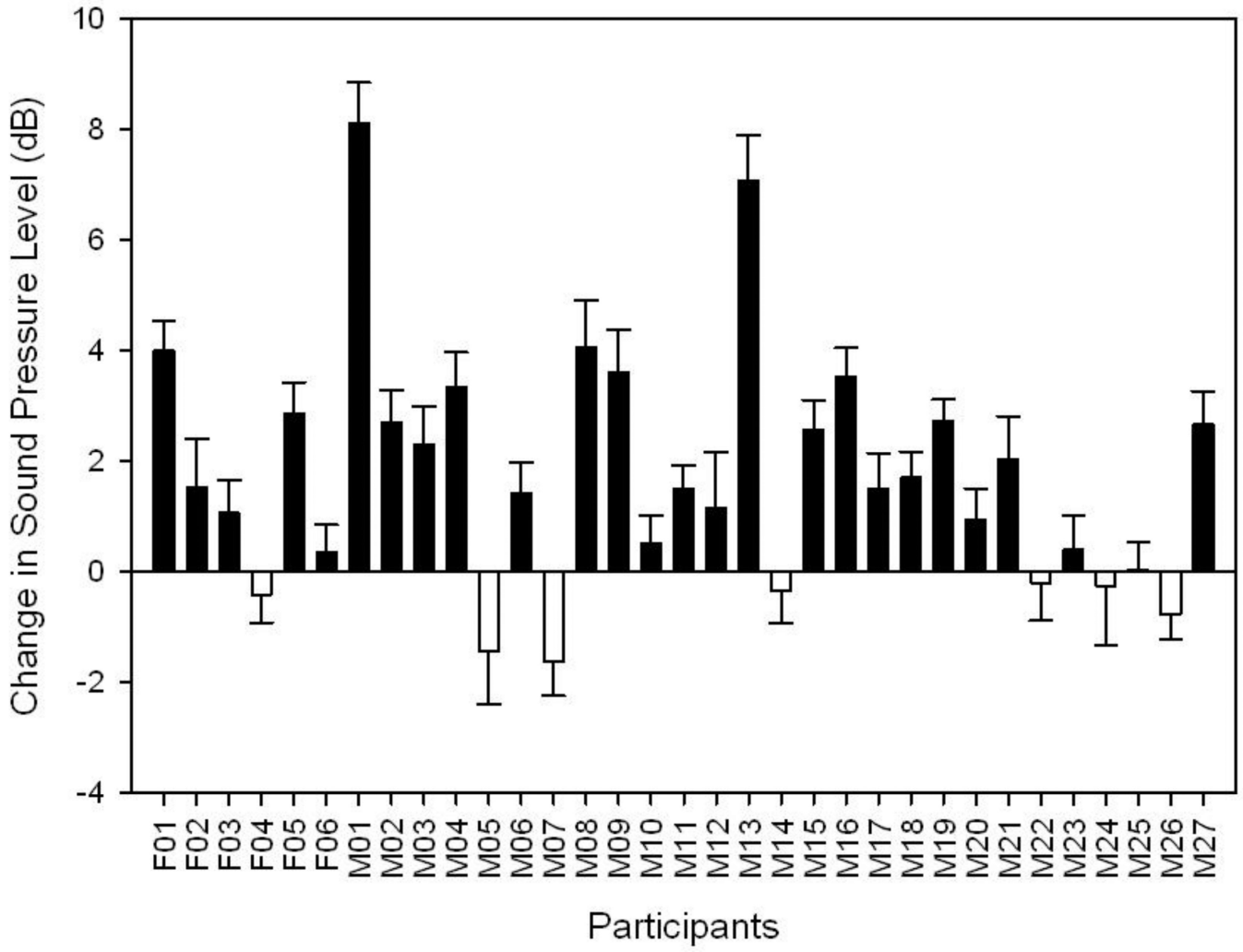


Figure 2.

Change in Sound Pressure Level (speech-in-noise minus speech-in-quiet) for each participant in the monologue. Bars denote the mean change, lines denote the standard error. Black bars follow the significant condition effect. White bars do not follow the condition effect.

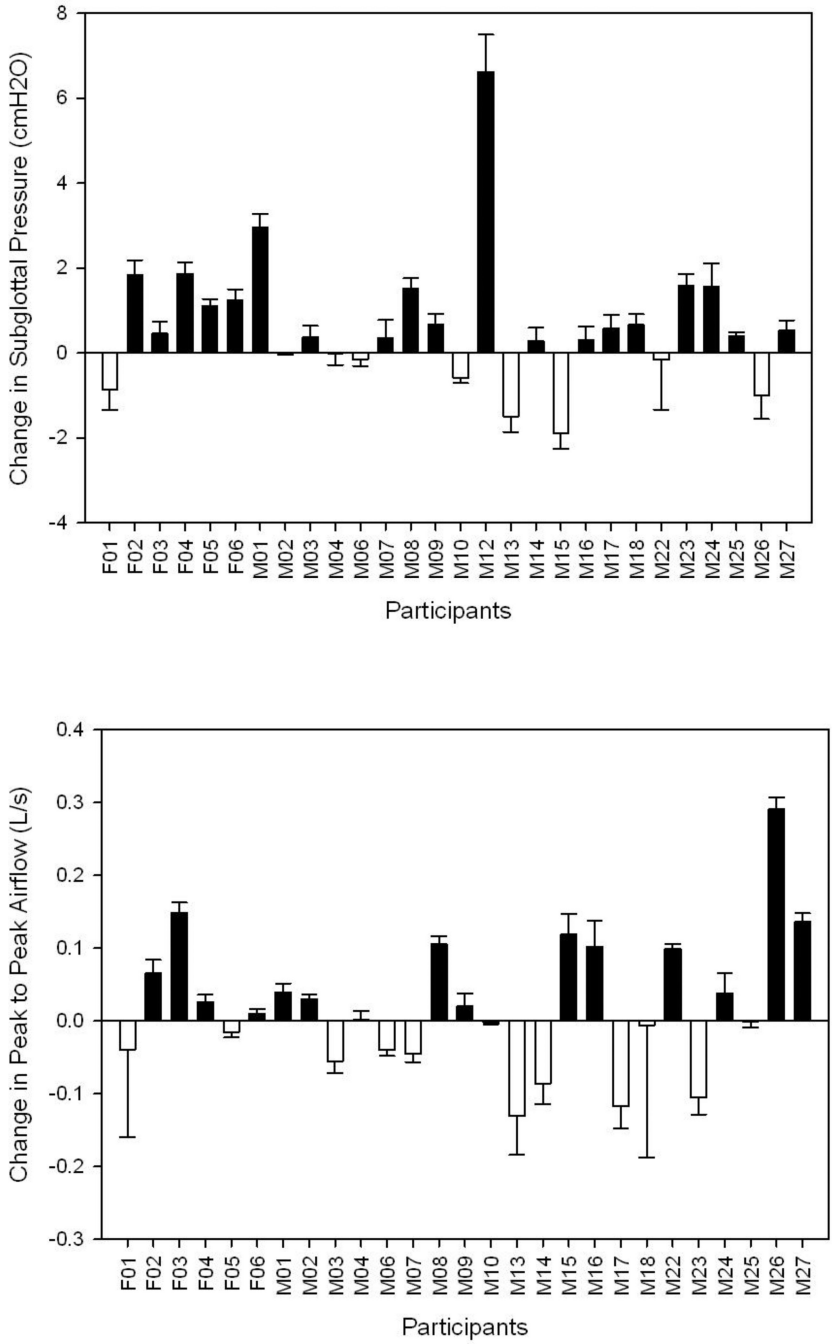


Figure 3.

Change in Subglottal Air Pressure and Peak to Peak Glottal Airflow (speech-in-noise minus speech-in-quiet) for each participant in the sentence task. Bars denote the mean change, lines denote the standard error. Black bars follow the significant condition effect. White bars do not follow the condition effect.

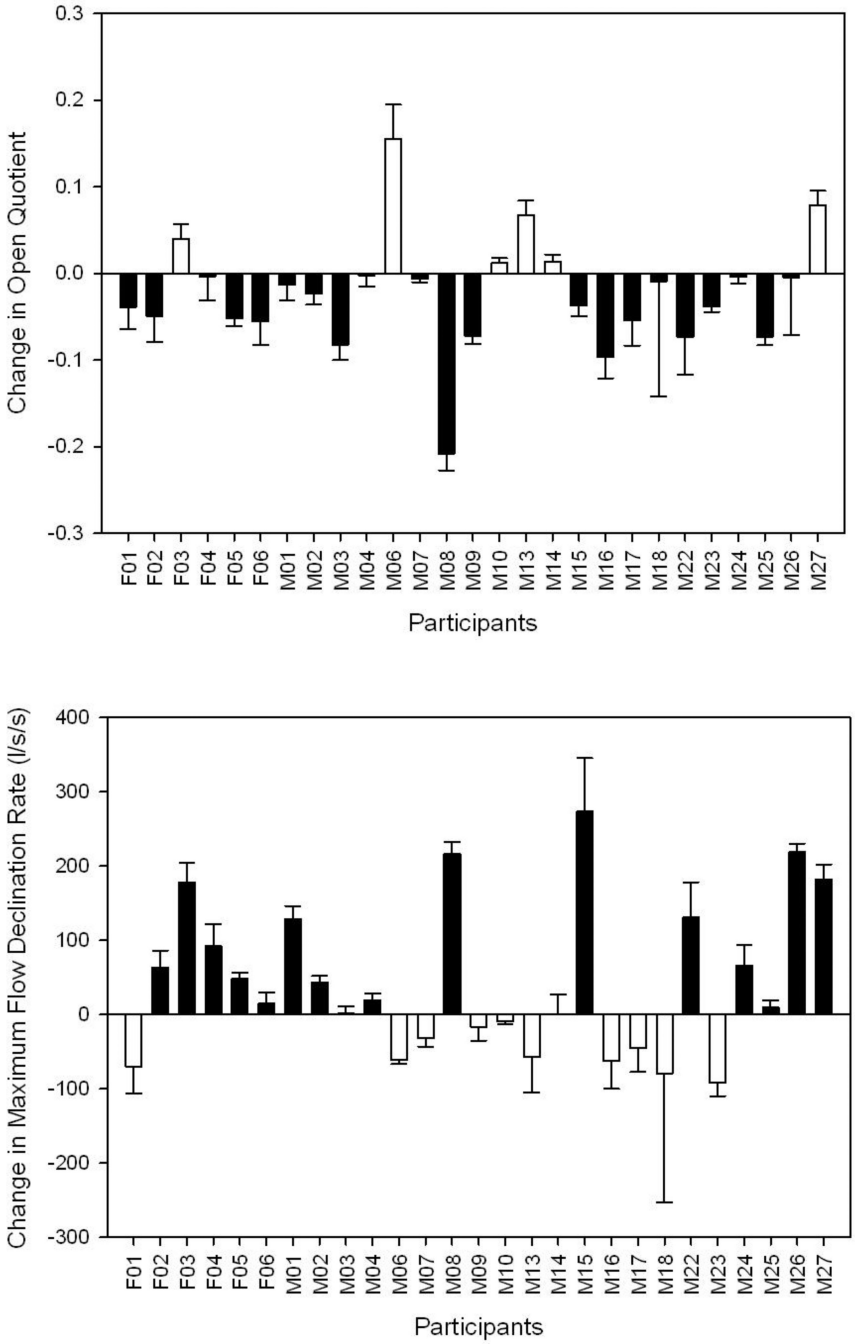


Figure 4.

Change in Open Quotient and Maximum Flow Declination Rate (speech-in-noise minus speech-in-quiet) for each participant in the sentence task. Bars denote the mean change, lines denote the standard error. Black bars follow the significant condition effect. White bars do not follow the condition effect.

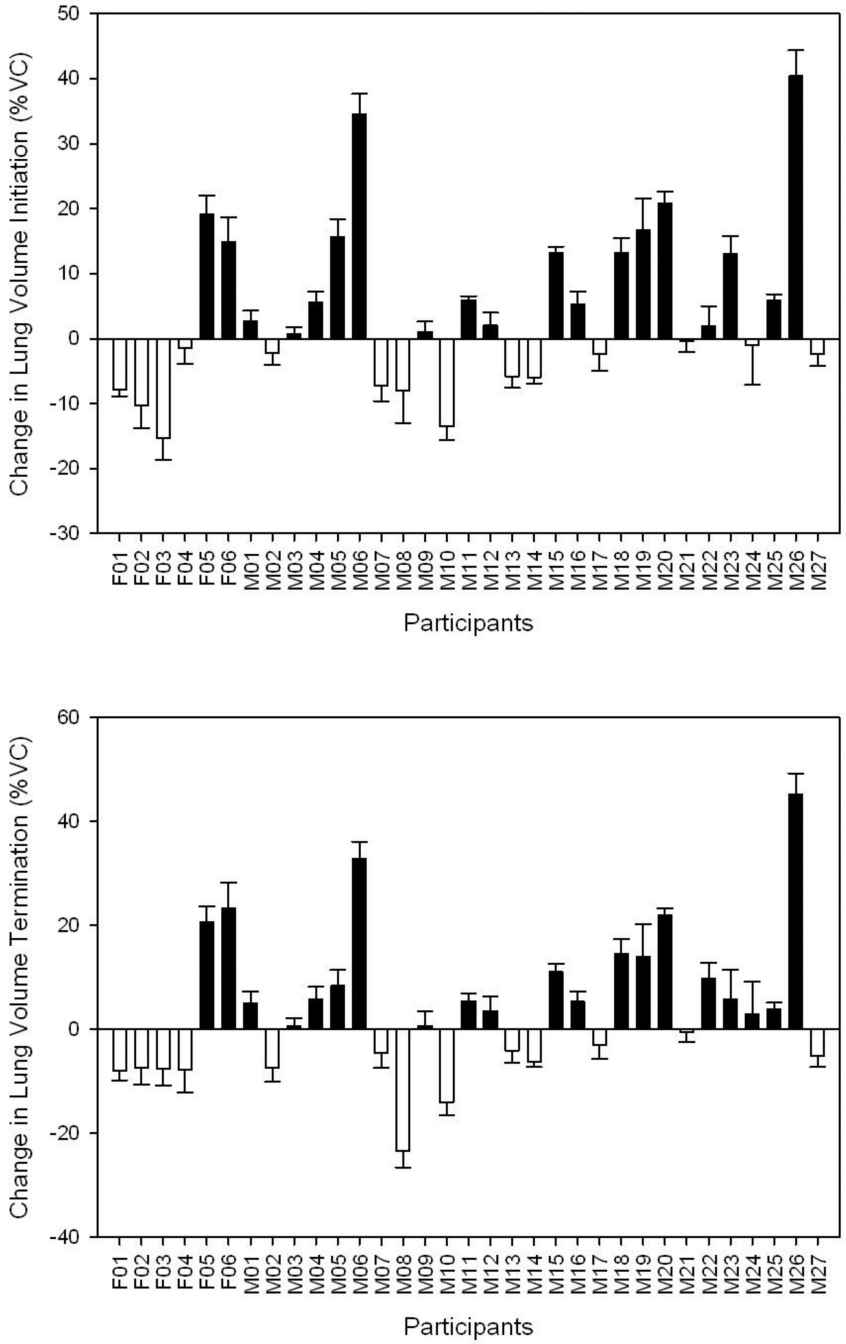
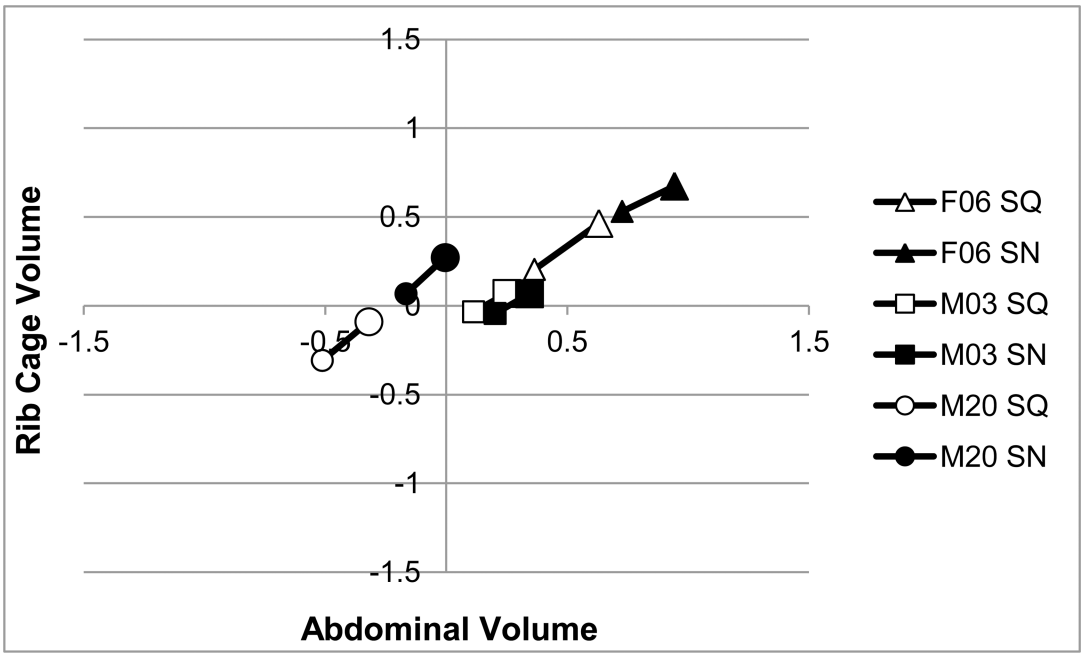


Figure 5.

Change in Lung Volume Initiation and Termination (speech-in-noise minus speech-in-quiet) for each participant in the monologue. Bars denote the mean change, lines denote the standard error. Black bars follow the significant condition effect. White bars do not follow the condition effect.



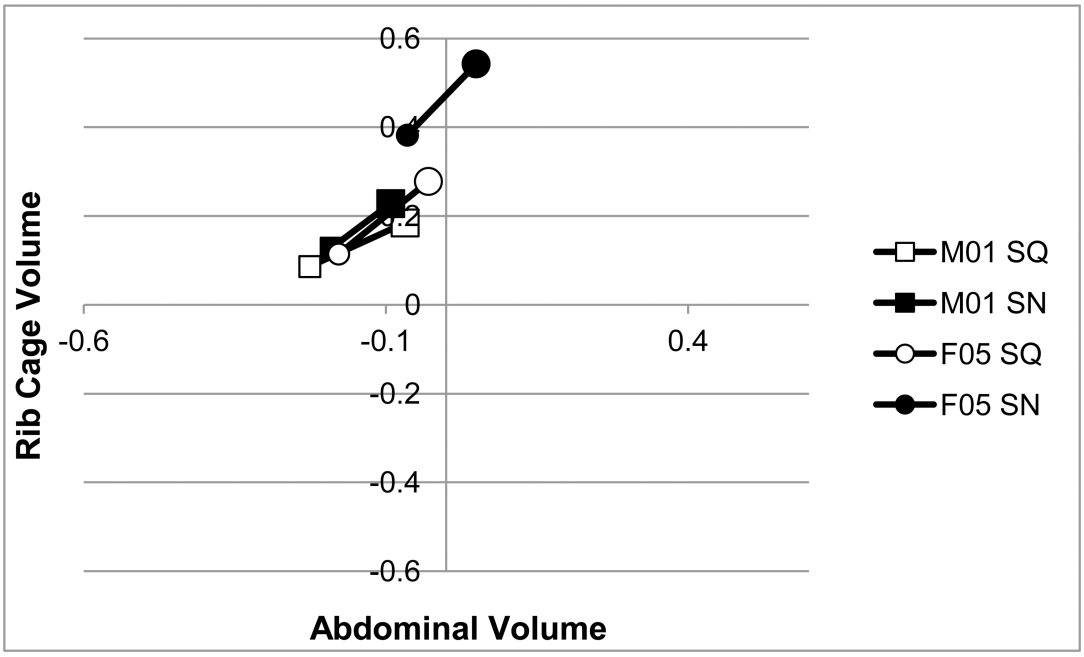


Figure 6.

Rib Cage Volume (y-axis) by Abdominal Volume (x-axis) average motion-motion plots for five participants in the monologue. White symbols reflect speech-in-quiet (SQ). Dark symbols reflect speech-in-noise (SN). Larger symbols indicate the mean initiation of utterance. Smaller symbols indicate the mean termination of utterance. Vertical 0 represents rib cage EEL. Horizontal 0 represents abdominal EEL.

J Commun Disord. Author manuscript; available in PMC 2015 March 01.

Table 1

Participant characteristics including sex (F=female, M=male), age in years, time since diagnosis, Hoehn & Yahr stage, hypophonia severity rating, pure tone averages (PTA) across 500Hz, 1000 Hz and 2000 Hz, PD-related medications, and previous behavioral speech and voice therapy.

Participant #	Age at Date of Testing (years)	Time Since Diagnosis (years)	Hoehn & Yahr Stage	Hypophonia Ratings	Pure Tone Average (PTA) in dB	PD Related Drug(s)	Previous Behavioral Therapy
F01	78	10	4	Moderate	23	Sinemet, Mirapex, Zelapar	None
F02	74	13	2	Moderate	33	None	None
F03*	76	5	3	Normal	35	Requip, Stalevo	None
F04	81	4	2	Mild	37	Carbidopa-Levodopa	None
F05	61	7	2	Mild	8	Mirapex, Stalevo	None
F06	83	29	3	Mild	35	Mirapex, Sinemet	Speech Therapy and LSVT®
M01	68	3	2	Mild	22	Azilect Tablet, Carbidopa-Levodopa, Mirapex	LSVT®
M02	79	6	3	Mild	23	None	LSVT®
M03	47	21	4	Mild	8	Sinemet	LSVT®
M04	68	11	2	Mild	17	Sinemet, Mirapex ER	None
M05	76	5	3	Moderate	25	Carbidopa-Levodopa, Mirapex	None
M06	66	6	2	Mild	12	Amantadine, Azilect	Speech Therapy
M07	81	5	2	Mild	33	Azilect, Carbidopa-Levodopa	None
M08	68	1	3	Moderate	10	Azilect, Carbidopa-Levodopa	None
M09	74	3	5	Moderate-Severe	48	Carbidopa-Levodopa	Swallowing Therapy
M10	69	5	1	Mild	12	Amantadine, Mirapex	LSVT®
M11	60	1	2 3	Moderate	8	Mirapex	None
M12	67	10		Very Severe	25	None	Speech Therapy
M13	71	6	2	Mild	53	Amantadine, Mirapex	None
M14*	62	13	2	Mild	18	Amantadine, Comtan, Sinemet, Sinemet CR	None
M15	72	12	4	Moderate	28	Sinemet, Tasmar	Speech Therapy and LSVT®

Participant #	Age at Date of Testing (years)	Time Since Diagnosis (years)	Hoehn & Yahr Stage	Hypophonia Ratings	Pure Tone Average (PTA) in dB	PD Related Drug(s)	Previous Behavioral Therapy
M16	86	2	3	Mild	28	Carbidopa-Levodopa, Lodosyn	None
M17	69	1	3	Moderate	17	Amantadine, Ropinerole	None
M18	74	6	4.5	Severe	12	Sinemet	Speech Therapy
M19	73	5	2	Moderate	28	Sinemet	Speech Therapy and LSVT®
M20	37	11	2	Severe	12	Amantadine, Carbidopa- Levodopa	Speech Therapy and\ LSVT®
M21	71	14	2	Moderate	27	Requip XL, Sinemet, Stalevo	None
M22*	73	6	3	Moderate	37	Mirapex, Selegiline	Speech Therapy and LSVT®
M23	73	14	2	Mild	12	Mirapex, Selegiline, Sinemet	Speech Therapy and LSVT®
M24	67	3	1	Moderate	32	Requip XL	None
M25	59	6	2	Mild	22	Artane, Azilect, Requip XL, Symmetrel	None
M26	74	2	1.5	Mild	20	Requip XL	None
M27	81	6	1	Mild	23	Artane, Mirapex	None

*Indicates participant had deep brain stimulation (DBS) of the subthalamic nucleus.

^PTA reported for better ear.

Table 2

Mean Difference and Alpha Levels for Reliability of Measurements

Measure	Mean Difference		Alpha Level
Sound Pressure Level	.2600	dB	.110
Subglottal Pressure	.0100	cmH_2O	.952
Peak-to-Peak Glottal Airflow	.0004	L/s	.996
Open Quotient	.0040		.947
Maximum Flow Declination Rate	2.9200	L/s/s	.979
Lung Volume Initiation	.0400	%VC	.966
Lung Volume Termination	1.1500	%VC	.399
Lung Volume Excursion	1.1100	%VC	.224
Rib Cage Initiation	.0100	V-EEL	.374
Rib Cage Termination	.0190	V-EEL	.105
Rib Cage Excursion	.0090	V-EEL	.257
Abdominal Initiation	.0030	V-EEL	.898
Abdominal Termination	.0090	V-EEL	.740
Abdominal Excursion	.1300	V-EEL	.277
Utterance Length	.8400	syllables	.647

dB = decibels, L/s = liters per second, L/s/s = liters per second per second, %VC = percent of vital capacity, V-EEL = volts relative to end-expiratory level.

Table 3

Group Means and Standard Errors for all Dependent Measures by Condition

Dependent Measure	Condition	Mean	Standard Error
Acoustic Measurements			
Sound Pressure Level (dB)	Speech-in-quiet	79.180	.120
	Speech-in-noise	81.150	.120
Aerodynamic Measurements			
Subglottal Pressure (cmH_2O)	Speech-in-quiet	6.430	.120
	Speech-in-noise	7.070	.140
Peak-to-Peak Glottal Airflow (L/s)	Speech-in-quiet	.344	.020
	Speech-in-noise	.360	.010
Open Quotient	Speech-in-quiet	.710	.010
	Speech-in-noise	.680	.010
Maximum Flow Declination Rate (L/s/s)	Speech-in-quiet	358.530	16.070
	Speech-in-noise	397.500	17.270
Respiratory Measurements			
Lung Volume Initiation relative to EEL (%VC)	Speech-in-quiet	19.700	.620
	Speech-in-noise	24.700	.680
Lung Volume Termination relative to EEL (%VC)	Speech-in-quiet	1.400	.610
	Speech-in-noise	6.400	.680
Lung Volume Excursion (%VC)	Speech-in-quiet	18.300	.430
	Speech-in-noise	18.300	.400
Rib Cage Initiation relative to EEL (volts)	Speech-in-quiet	.279	.007
	Speech-in-noise	.346	.007
Rib Cage Termination relative to EEL (volts)	Speech-in-quiet	.046	.007
	Speech-in-noise	.118	.007
Rib Cage Excursion (volts)	Speech-in-quiet	.232	.006
	Speech-in-noise	.228	.007
Abdominal Volume Initiation relative to EEL (volts)	Speech-in-quiet	.053	.011
	Speech-in-noise	.087	.012
Abdominal Volume Termination relative to EEL (volts)	Speech-in-quiet	−.170	.012
	Speech-in-noise	−.141	.013
Abdominal Volume Excursion (volts)	Speech-in-quiet	.222	.007
	Speech-in-noise	.228	.007
Utterance Length (syllables/breath)	Speech-in-quiet	10.400	.220
	Speech-in-noise	10.500	.230

Table 4

Direction of Change for Each Dependent Variable for Selected Participants for Increases in Vocal Intensity

	Acoustic	Glottal Aerodynamic Measurements					Respiratory Measurements								
Speaker	SPL	P_s	PP V_g	OQ	MFDR	LVI	LVT	LVE	RCI	RCT	RCE	ABI	ABT	ABE	UL
F05	I	I		I	I	I	I		I	I					I
F06	I	I	I	I	I	I	I		I	I					
M01	I	I	I	I	I	I	I		I	I		I			
M03	I	I		I	I	I	I								I
M20	I	n/a	n/a	n/a	n/a	I	I		I	I					

I = indication of improved support. n/a are used where there was no data available for that participant. F= female, M = male. SPL = sound pressure level in monologue, P_S = estimated subglottal air pressure, PP V_g = peak-to-peak glottal airflow, OQ = open quotient, MFDR = maximum flow declination rate, LVI = lung volume initiation, LVT = lung volume termination, LVE = lung volume excursion, RCI = rib cage volume initiation, RCT = rib cage volume termination, RCE = rib cage volume excursion, ABI = abdominal volume initiation, ABT = abdominal volume termination, ABE = abdominal volume excursion, UL = utterance length