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Respiratory Responses to Two Voice Interventions for Parkinson's Disease

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ABSTRACT

Purpose: The purpose of this study was to examine the respiratory strategies used by persons with Parkinson's disease (PD) to support louder speech in response to two voice interventions. Contrasting interventions were selected to investigate the role of internal and external cue strategies on treatment outcomes. LSVT LOUD, which uses an internal cueing framework, and the SpeechVive prosthesis, which employs an external noise cue to elicit louder speech, were studied.

Method: Thirty-four persons with hypophonia secondary to idiopathic PD were assigned to one of three groups: LSVT LOUD (n = 12), SpeechVive (n = 12), or a nontreatment clinical control (n = 10). The LSVT LOUD and SpeechVive participants received 8 weeks of voice intervention. Acoustic and respiratory kinematic data were simultaneously collected at pre-, mid- and posttreatment during a monologue speech sample. Intervention outcomes included sound pressure level (SPL), utterance length, lung volume initiation, lung volume termination, and lung volume excursion.

Results: As compared to controls, the LSVT LOUD and SpeechVive participants significantly increased SPL at mid- and posttreatment, thus confirming a positive intervention effect. Treatment-related changes in speech breathing were further identified, including significantly longer utterance lengths (syllables per breath group) at mid- and posttreatment, as compared to pretreatment. The respiratory strategies used to support louder speech varied by group. The LSVT LOUD participants terminated lung volume at significantly lower levels at mid- and posttreatment, as compared to pretreatment. This finding suggests the use of greater expiratory muscle effort by the LSVT LOUD participants to support louder speech. Participants in the SpeechVive group did not significantly alter their respiratory strategies across the intervention period. Single-subject effect sizes highlight the variability in respiratory strategies used across speakers to support louder speech.

Conclusions: This study provides emerging evidence to suggest that the LSVT LOUD and SpeechVive therapies elicit different respiratory adjustments in persons with PD. The study highlights the need to consider respiratory function when addressing voice targets in persons with PD.

Correspondence to Kelly Richardson: krichardson@umass.edu. ***Disclosure:*** *Jessica E. Huber is the inventor of the SpeechVive device and has shares in SpeechVive, Inc., the company that manufactures and sells the device. She also serves on the Rock Steady Boxing Medical Advisory Board. Sandy Snyder also has shares in SpeechVive, Inc., but has no nonfinancial conflicts of interest to disclose. The other authors have declared that no other competing financial or nonfinancial interests existed at the time of publication.*

Motor impairment is a hallmark feature of Parkinson's disease (PD). The motor-based symptoms are characterized by reduced amplitude and speed of movement and difficulty initiating and coordinating movement (Morris et al., 1994; Sheridan et al., 1987; Stelmach et al., 1989; Warabi et al., 1986). These motor-based impairments affect the respiratory, laryngeal, and supralaryngeal systems and often result in a marked reduction in vocal intensity in the majority of speakers with PD. Reduced

vocal intensity, a condition known as hypophonia, is estimated to affect approximately 75% of patients in some stage of the disease process (Hartelius & Svensson, 1994) and is reported to lead to persistent communication breakdowns (Duffy, 2005; Kent et al., 1999), feelings of depression (Allain et al., 2000; Remy et al., 2005), and loss of autonomy and social isolation (Calne, 2003). As a result, remediating hypophonia is an important focus of voice intervention for persons with PD.

Respiratory Function and PD

Several factors contribute to hypophonia including reduced rib cage (RC) compliance (Solomon & Hixon, 1993; Tzelepis et al., 1988; Weiner et al., 2002), decreased respiratory muscle strength and coordination (De Bruin et al., 1993; Hovestadt et al., 1989), decreased vocal fold approximation (Hanson et al., 1984; Perez et al., 1996), thoraco-abdominal asynchrony (Florêncio et al., 2019), and misaligned temporal coupling of the respiratory–laryngeal mechanisms (Solomon & Hixon, 1993). These physiological changes may adversely affect the efficiency of rest breathing (Tzelepis et al., 1988) and speech breathing (Bunton, 2005; Huber et al., 2003) in persons with PD. In general, two patterns of speech breathing have been previously identified in this clinical population. Individuals with PD have been reported to initiate and terminate speech at higher-than-normal lung volumes (Huber & Darling, 2011). Initiating speech at a higher lung volume allows the speaker to capitalize on passive recoil forces during speech production. In contrast, other studies have shown that speakers with PD initiate and terminate speech at lower-than-normal lung volumes (Bunton, 2005; Darling-White et al., 2022; Huber & Darling-White, 2017). This respiratory pattern requires active expiratory muscle forces to maintain subglottal pressure for speech produced at lower-than-normal lung volumes. These two divergent patterns of speech breathing, identified for persons with PD, may be attributed to differences in disease state. Longitudinal studies of changes in speech breathing in persons with PD suggest that the pattern of increased lung volume initiation (LVI) and lung volume termination (LVT) is more common earlier in the disease state, and the pattern of decreased LVI and LVT is more common as the disease progresses (Darling-White et al., 2022; Huber & Darling-White, 2017). Since both respiratory patterns rely on respiratory muscle strength, a known deficit in persons with PD, speakers may have difficulty generating and sustaining the subglottal pressure required for speech, a task that is more difficult when producing louder speech due to the demand for higher subglottal pressure generation. Although our current voice intervention approaches target increased vocal intensity in persons with PD, the respiratory adjustments used to support louder speech are not well understood. Since therapeutic techniques are meant to be used in everyday communication exchanges, it is critical to understand how the respiratory system is impacted by treatment to avoid maladaptive respiratory patterns, which could make speech more effortful or fatiguing for people with PD.

Voice Interventions for Hypophonia

Two treatments, LSVT LOUD therapy and the SpeechVive prosthesis, are used to remediate hypophonia in persons with PD. Although LSVT LOUD and SpeechVive share the same therapeutic target of increased vocal intensity, they differ substantially in the cue strategy used in therapy. LSVT LOUD targets louder speech using internal, self-initiated cues. Over the course of therapy, persons with PD learn to independently monitor their speech loudness and to make online adjustments to vocal intensity when they perceive that their voice is soft. SpeechVive, in contrast, is a prosthetic device that elicits louder speech using an external noise cue, which leverages the Lombard effect (Lombard, 1911). The Lombard effect is a reflexive response by speakers to enhance audibility in noise-aversive speaking conditions. Neurologically intact (Shrivastav et al., 2014; Whittico et al., 2020) and neurologically impaired (Adams et al., 2020; Richardson & Sussman, 2019; Shrivastav et al., 2014; Stathopoulos et al., 2014) individuals have been shown to significantly increase their vocal intensity when speaking in the presence of background noise. Although prior studies have shown that LSVT LOUD and the SpeechVive prosthesis both yield significant increases in vocal intensity during monologue speech (Ramig, Sapir, Countryman, et al., 2001; Ramig, Sapir, Fox, & Countryman, 2001; Richardson et al., 2022; Stathopoulos et al., 2014), the respiratory adjustments used by speakers in response to treatment are not well understood. This is an important area of study, as there is evidence to suggest that internal and external forms of cueing, such as those utilized in LSVT LOUD and SpeechVive training, may differentially affect motor responses.

Differential Impact of Cue Strategy

The indication that cue strategy may differentially affect motor function was first reported in studies of gait performance. The use of an external auditory cue was shown to significantly increase walking velocity (Ford et al., 2010), cadence (Ford et al., 2010), and stride length (Ford et al., 2010; Rochester et al., 2007) in persons with PD, whereas the use of an internal cue such as "think about taking larger steps" significantly reduced step frequency in persons with PD (Baker et al., 2007). These alterations in gait performance may be explained, in part, by the pathophysiology of PD.

The internal–external control hypothesis proposes that the basal ganglia and supplementary motor area are predominantly involved in internally cued and memory-guided movement (Crawford et al., 1989; Flowers, 1976; Goldberg, 1985; Jueptner & Weiller, 1998; Mushiake & Strick, 1995; Van Donkelaar et al., 1999, 2000), whereas externally cued movement preferentially involves the cerebellum, parietal lobe, and lateral premotor cortex (Goldberg, 1985; Jueptner & Weiller, 1998; Van Donkelaar et al., 1999, 2000). This hypothesis is supported by neuroimaging studies that have shown that internally cued and externally cued movements are associated with different cortical activation patterns (Debaere et al., 2003; Gerloff et al., 1998; Halsband et al., 1994). In persons with PD, decreased activation has been reported in the regions within the basal ganglia–thalamo–motor loop during internally cued tasks and enhanced or preserved within the cerebello–cerebral loop during externally cued tasks (Lewis et al., 2007). Interestingly, studies have further shown that internally generated movements, such as writing and drawing, are often improved in persons with PD with the use of external cues (Briand et al., 1999; Crawford et al., 1989; Flowers, 1976; Martin et al., 1994; Morris et al., 1996; Oliveira et al., 1997).

There is further evidence to suggest that cue strategy may differentially influence respiratory mechanics in persons with PD. Sadagopan and Huber (2007) reported that the respiratory patterns of 14 individuals with PD approached the patterns of neurotypical age- and sex-matched controls when an external cue, multitalker background noise, was used to elicit increased vocal intensity, as compared to the respiratory patterns observed in response to an internal cue (e.g., speak at twice your comfortable loudness). Similar findings were reported by Stathopoulos et al. (2014), who found that the use of an external noise cue, delivered through the SpeechVive prosthesis, evoked respiratory and laryngeal efficiencies during speech production in the majority of speakers with PD. In the Stathopoulos et al. study, 19 of 33 participants increased LVI and LVT the first time they used the SpeechVive prosthesis. In contrast, Huber et al. (2003) found that, in a small sample of people with PD, there were no consistent changes in respiratory patterns after treatment with LSVT LOUD. Three of the participants increased LVIs and LVTs, and three participants did not change LVIs and LVTs. Although these studies pave the way for understanding the respiratory mechanics associated with external and internal forms of cueing, the laboratory testing conditions used in these studies do not mirror the approaches used in voice therapy, nor do they reflect the time course of therapy.

Purpose of the Study

Given the paucity of comparative intervention data, this study sought to examine a theoretically and clinically motivated research question: Do the internal and external cue strategies used in voice therapy elicit different respiratory patterns in individuals with PD? To address this research question, acoustic and respiratory outcomes were compared for two divergent approaches to voice intervention for persons with PD: the LSVT LOUD program, which is predicated on an internal cueing strategy to increase vocal intensity, and the SpeechVive prosthesis, which uses an external noise cue to reflexively increase vocal intensity. Based on prior research, we hypothesized that the respiratory strategies employed for vocal intensity regulation would differ by treatment. Specifically, it was hypothesized that the SpeechVive participants would increase LVI and LVT at higher speech intensities, with no expected change in LVI or LVT for the LSVT LOUD participants.

Materials and Method

The institutional review board at the University of Massachusetts Amherst approved the study procedures, and Purdue University deferred to University of Massachusetts, consistent with the National Institutes of Health's policy for multisite research. Written informed consent was obtained for all study participants. Study participants were paid for their participation and were not charged for LSVT LOUD therapy or the SpeechVive device.

Participant Description

Forty-one individuals with PD were screened for study eligibility. Seven individuals were excluded from the study because they failed to meet the eligibility criteria or they declined to participate. In total, 34 individuals with hypophonia, secondary to idiopathic PD, were enrolled in this multisite study (Purdue University and University of Massachusetts Amherst).

Participants were stratified to one of three groups based on hypophonia severity level: LSVT LOUD (n = 12; M_{age} = 68.42 years, SD = 4.89), SpeechVive (n = 12; M_{age} = 69.58 years, SD = 7.53), or a clinical control group (n = 10; M_{age} = 66.60 years, SD = 10.71). A balanced distribution of hypophonia severity level was targeted across groups. Ratings of hypophonia severity were assigned during the study screening procedures and confirmed by the first author (K.R.), who has 13 years of clinical experience with motor speech and voice disorders. The clinical controls were tested at the same time points as the experimental participants, but voice intervention was withheld. Due to the SARS-CoV-2 pandemic, post-treatment data were not collected for five participants (two LSVT LOUD participants, one SpeechVive participant, and two clinical controls). One LSVT LOUD participant dropped from the treatment or testing protocol after

midtreatment. The pre- and midtreatment data collected for these participants were included for analysis.

Criteria for inclusion were (a) a diagnosis of idiopathic PD and (b) the presence of hypophonia as determined by an American Speech-Language-Hearing Association (ASHA)–certified speech-language pathologist with voice experience. Participants were excluded if they had (a) a recent history of cold or allergy symptoms; (b) a comorbid neurological diagnosis; (c) symptoms of depression as reflected by the Geriatric Depression Scale (Yesavage et al., 1983) and were not under pharmacological management for depressive symptoms; (d) a history of head, neck, or chest surgery except for deep brain stimulation implantation; (e) a history of respiratory problems, such as asthma or chronic obstructive pulmonary disease; (f) a history of smoking in the past 5 years; and (g) the presence of a laryngeal pathology, not related to PD, that would contraindicate voice therapy. Laryngeal pathology was assessed at baseline using a videolaryngoscopic examination. Decreased vocal fold adduction was not part of the exclusionary criteria. Auditory threshold testing was used to assess baseline hearing status. All participants demonstrated hearing thresholds at 40 dB or lower in at least one ear for octave frequencies between 250 and 4000 Hz (Feenaughty et al., 2013).

Table 1 provides a descriptive overview of the participants. The participants presented with mild-to-severe motor involvement as determined by the Hoehn and Yahr staging classification. The Montréal Cognitive Assessment (MoCA; Hoops et al., 2009) was administered at baseline to identify the presence or absence of cognitive impairment. Given that cognitive deficits are commonly reported in individuals with PD, inclusion of this clinical subgroup enhances the ecological validity of the study. The MoCA scores, reported in Table 1, represent the inclusion of 11 participants with mild cognitive impairment (MoCA scores 18–25) and 23 participants with normal cognition (MoCA scores $\geq$ 26). The 11 participants with mild cognitive impairment were uniformly distributed between the

Table 1. Participant description.

ID	Group	Age (years)	H/Y	MoCA	Hypophonia severity	PD-related medications
F03	LSVT LOUD	65	3	30	Mild	Carbidopa–levodopa, Azilect, ropinirole
F04	LSVT LOUD	71	2	27	Mild	Carbidopa–levodopa
F05	LSVT LOUD	75	2	26	Mild–moderate	Carbidopa–levodopa, amantatine
M02	LSVT LOUD	62	3	30	Moderate	Azilect
M03	LSVT LOUD	74	3	26	Mild	Carbidopa–levodopa
M04[a]	LSVT LOUD	70	3	24	Moderate	Sinemet
M08	LSVT LOUD	68	2	21	Moderate	Carbidopa–levodopa
M09	LSVT LOUD	68	4	21	Mild–moderate	Carbidopa–levodopa
M11	LSVT LOUD	65	2	29	Moderate	Carbidopa–levodopa
M12	LSVT LOUD	75	3	23	Mild–moderate	Carbidopa–levodopa
M13	LSVT LOUD	60	1	25	Mild	Carbidopa–levodopa
M14	LSVT LOUD	68	2	27	Moderate	Carbidopa–levodopa
F01	SpeechVive	82	2	23	Moderate	Carbidopa–levodopa
F02	SpeechVive	77	4	27	Mild–moderate	Carbidopa–levodopa
F46	SpeechVive	72	2	29	Mild	Carbidopa–levodopa, amantatine
M01	SpeechVive	75	3	27	Mild–moderate	Sinemet
M05	SpeechVive	70	2	21	Moderate	Carbidopa–levodopa
M06	SpeechVive	52	3	30	Moderate	Carbidopa–levodopa
M07	SpeechVive	68	1	27	Mild	Carbidopa–levodopa
M10	SpeechVive	62	3	23	Mild–moderate	Carbidopa–levodopa, amantatine
M15	SpeechVive	67	2	26	Moderate	Carbidopa–levodopa, selegiline
M43	SpeechVive	70	3	23	Moderate	Carbidopa–levodopa
M45[a]	SpeechVive	69	3	26	Mild	—
M48	SpeechVive	71	5	24	Moderate	Carbidopa–levodopa, pramipexole, amantadine
F31	Control	79	3	26	Mild	Carbidopa–levodopa, pramipexole
F33	Control	54	2	27	Mild	Carbidopa–levodopa, amantadine, selegiline
F34	Control	70	2	27	Moderate	Carbidopa–levodopa, selegiline
F37	Control	80	2	27	Moderate	Carbidopa–levodopa
F40[a]	Control	58	1	29	Mild	Carbidopa–levodopa
M32[a]	Control	54	3	27	Moderate	Carbidopa–levodopa, amantadine
M35	Control	68	3	27	Moderate	—
M38	Control	69	1	27	Mild	Carbidopa–levodopa, Azilect
M39	Control	55	4	26	Moderate	Sinemet, pramipexole
M47	Control	79	5	22	Mild	Carbidopa–levodopa

Note. The first character in the ID column denotes participant sex (M = male; F = female). Hypophonia severity ratings were assigned or confirmed by the first author (K.R.) during connected speech. Em dashes indicate no PD-related medications reported. H/Y = Hoehn and Yahr stage; MoCA = Montréal Cognitive Assessment; PD = Parkinson's disease.

[a]Participant received deep brain stimulation to the subthalamic nucleus.

LSVT LOUD and SpeechVive interventions. All participants recruited for study could fully participate in their assigned voice intervention and follow laboratory instructions. A small number of participants (n = 8) reported a prior history of behavioral speech therapy to address speech and/or swallowing concerns. Behavioral interventions included nonstandardized voice and swallowing exercises and were completed at least 12 months prior to enrollment in the current study. Behavioral interventions focused on salivary management, dysphagia-related issues, cognitive function, and supporting communication. None of the participants followed a validated and standardized approach to voice therapy (e.g., LSVT LOUD), and none had used the SpeechVive device prior to the study. Five of these eight participants (F31, F37, M35, M38, and M47) were clinical controls, and three of the participants (M43, M45, and M48) were assigned to a novel intervention (SpeechVive prosthesis). Four participants received deep brain stimulation to the subthalamic nucleus (DBS-STN); their participant numbers are indicated in Table 1. The participants with DBS-STN were fairly balanced across groups (n = 1 SpeechVive, n = 1 LSVT LOUD, n = 2 clinical controls). The majority of participants (n = 32) were under pharmacological management for their PD-related symptoms, and these participants were tested during the "on" state of their medication cycle. One control participant (M35) and one SpeechVive participant (M45) were not under pharmacology management for their PD symptoms at the time of the study.

Intervention Description

The LSVT LOUD and SpeechVive interventions followed the standardized protocol and treatment dose previously described for each approach (Ramig, Sapir, Countryman, et al., 2001; Ramig, Sapir, Fox, & Countryman, 2001; Stathopoulos et al., 2014).

LSVT LOUD

Twelve LSVT LOUD participants received voice treatment at an outpatient clinic in Western Massachusetts and participated in laboratory testing at the University of Massachusetts Amherst. The ASHA-certified and LSVT LOUD–trained clinician was not involved in any other aspect of the study. The participants received the standard LSVT LOUD protocol: 16 voice intervention sessions over 4 weeks (1 hr per session × 4 days per week × 4 weeks). Furthermore, the participants completed daily homework and carryover activities consistent with the treatment protocol. At the end of the 4-week intervention period, the LSVT LOUD participants were instructed to engage in daily, at-home vocal practice for an additional 4 weeks. The LSVT LOUD Homework Helper application was installed on each participant's mobile device in order to facilitate at-home practice of the therapy techniques. A homework log was maintained by the LSVT LOUD participants and reviewed biweekly by research personnel. The LSVT LOUD participants complied with the clinic and home-based intervention protocols.

SpeechVive Prosthesis

Trained research personnel implemented the SpeechVive intervention. Twelve participants were instructed to wear the SpeechVive prosthesis daily during communication and during 30 min of oral reading for 8 consecutive weeks. The SpeechVive prosthesis presented multitalker noise (Auditec of St. Louis) to one ear when the participant was speaking. Multitalker noise has been shown to naturally elicit louder speech due to the Lombard effect (Garnier et al., 2010). The multitalker noise was presented monaurally through a small speaker with an open-ear fitting to prevent an occlusion effect. The SpeechVive prosthesis was fit to the ear with the best hearing thresholds as determined by baseline audiometric thresholds. The detection level was adjusted by trained research personnel until the noise presented by the SpeechVive activated and deactivated at the onset and offset of speech. In accordance with the SpeechVive protocol, the amplitude of the multitalker noise was adjusted until each participant spoke 3 dB above their comfortable vocal intensity during monologue speech. Six of the 12 participants received intervention with the SpeechVive prosthesis at the University of Massachusetts Amherst, and six participants received the SpeechVive intervention at Purdue University. To monitor compliance with the SpeechVive protocol, usage data were recorded by the SpeechVive prosthesis and reviewed biweekly by research personnel. The SpeechVive participants complied with wearing the device in accordance with the therapy protocol. The SpeechVive participants did not receive any form of behavioral voice therapy.

Clinical Controls

Ten control participants were tested at Purdue University at the same time points as the LSVT LOUD and SpeechVive participants, but voice intervention was withheld. Intervention was offered through an unrelated study to these participants, after their completion of this study.

Equipment

Parallel procedures were used to record acoustic and respiratory kinematic data across laboratory sites. The acoustic signal was transduced using a head-mounted microphone (Sennheiser Model HSP 2; Shure Beta 53). The same microphone was used for each participant across sessions and was positioned at a 45° angle at a 6-cm mouth-to-microphone distance. Gain was provided to the acoustic signal through a preamplifier (Denon DN-700R; Marantz

PM 670). The microphone was calibrated for sound pressure level (SPL) on the day of testing at a known frequency of 1 kHz and a known decibel level of 94 dB SPL via a piston phone (Sper Scientific Acoustical Calibrator Model 850016; Quest QC-20 calibrator). The calibration tone was digitized at the appropriate gain level to allow gain to be included in the microphone calibration. The same methods of recording and calibration were used for each participant across testing sessions.

Respiratory kinematic data were transduced in a seated position using respiratory inductance plethysmography (Respitrace system, Ambulatory Monitoring). To transduce movements of the RC, an inductance band was positioned around the RC, inferior to the axilla. To transduce movements of the abdomen (AB), an inductance band was positioned at the level of the umbilicus, below the lowest ribs. Respiratory kinematic waveforms were digitized at 1 kHz, and the spirometry signals were digitized at 10 kHz in LabChart (ADInstruments, Version 7.1.2). The kinematic waveforms were digitized in synchronization with a head-mounted and room microphone using LabChart (ADInstruments, Version 7.1.2).

Procedure

Speech Tasks

Naturalistic connected speech samples were obtained for all acoustic and respiratory measures. Participants were asked to provide a 30-s monologue on one of the neutral topics presented on a laptop screen. The monologue was produced under the following counterbalanced conditions.

Comfortable intensity (COMF). All participants were asked to produce a monologue "in their typical conversational voice." The COMF directions avoided use of a vocal loudness descriptor (e.g., comfortable). The COMF condition for the LSVT LOUD participants reflects their application of internally generated cues to speak at a higher vocal intensity. The louder speech, targeted in LSVT LOUD therapy, becomes the participants' newly habituated COMF. To prevent a confounding device effect for the SpeechVive participants, the COMF condition was elicited with the SpeechVive prosthesis in place, but no noise emitted from the device.

High intensity (HIGH). The higher vocal intensity condition was elicited differently for the LSVT LOUD and SpeechVive groups and in accordance with their therapeutic protocols. For the SpeechVive participants, multitalker babble noise was presented monaurally through their SpeechVive at the patient's personalized settings during the monologue task. No cue as to loudness (neither typical conversational or louder voice) was provided to the SpeechVive group for the HIGH condition. For the LSVT LOUD group, participants were cued by the examiner to use a louder speaking volume by asking the participants to "speak in a louder voice."

Presentation of the COMF and HIGH conditions was counterbalanced across sessions and participants. Per pilot testing, a rest period was inserted between conditions in order to prevent a carryover effect of the higher vocal amplitude condition. No feedback on loudness was provided for either condition. A fixed interlocutor distance was maintained during the monologue recording.

Calibration of Kinematic Signals

Time-aligned RC, AB, and spirometer waveforms were collected during two 30-s periods of rest breathing, followed by two 30-s periods of "speechlike" breathing where the participants were instructed to silently read "buy Bobby a puppy now if he wants one" on each exhalation. Participants also performed at least three trials of a vital capacity maneuver. In the vital capacity maneuver, participants were instructed to "breathe in as much air as you can and then breathe out as much air as you can." Participants completed the maneuver after a stable breathing pattern was observed for rest breathing, and the examiner encouraged them and cued the switch from inhalation to exhalation. For all calibration maneuvers, participants wore nose clips and were instructed to make a tight lip seal with the spirometer mouthpiece.

To calibrate the sum signal as an estimate of lung volume, the RC and AB movement signals were compared with the digital spirometer signal during the rest breathing and speechlike breathing tasks. If the spirometer signal linearly trended upward or downward, the upward/downward trend was removed using the detrend function in MATLAB before calibrating for lung volume. Custom MATLAB programs were used to determine the best correction factors (k1 and k2) for the RC and AB. A least squares analysis (Moore–Penrose pseudoinverse function) was used to determine k1 and k2 in the following formula, ensuring RC and AB volume could be summed to spirometer with the least error.

$$\text{Spirometer Volume} = \text{RC} \times \text{k1} + \text{AB} \times \text{k2} \quad (1)$$

This method of calibration yielded the smallest percent error for estimation of lung volume in older adults and adults with PD (McKenna & Huber, 2019). Mean error between the estimated lung volumes and actual lung volumes acquired with the spirometer during rest breathing and speechlike breathing was calculated for each participant to determine which correction factor estimate to use for each session (see the Appendix for calibration type, calibration factors, and mean error in liters). For most participants, correction factors were generated for both the RC and the AB using formula Equation 1. However, when one of the correction factors was negative, two other

methods of calibration were considered: (a) using the absolute values from Equation 1 or (b) applying a correction factor only for the RC and setting the AB correction factor to 1. In all cases, the correction factors that provided the least error estimate of the spirometer signal from the sum of the RC and AB (with correction factors applied) were used in the lung volume measurements. During the speech tasks, the correction factors (k1 and k2) determined from the calibration procedure were used to estimate lung volume using the following formula:

$$\text{Estimated Lung Volume} = \text{RC} \times \text{k1} + \text{AB} \times \text{k2}. \quad (2)$$

Measurements

All monologues were orthographically transcribed by a research assistant and checked by a second research assistant. Differences in the transcriptions were resolved by consensus, sometimes with a third, more senior research assistant.

SPL

SPL data were measured across utterances, bounded by pauses greater than 150 ms, using Praat (Boersma & Weenink, 2022). Utterances and pauses were visually identified and annotated in Praat using a time-aligned wideband spectrogram and acoustic waveform. A customized Praat script was then used to extract SPL data for the annotated utterances. Calibration correction factors were then applied to the SPL data.

The following measures were made for each breath group. A breath group was defined as all of the speech produced on a single breath. Breath groups were visually identified by examining the lung volume and RC signals, along with the time-locked acoustic signal.

Utterance Length

The number of syllables produced in each breath group was counted. Utterance length was considered a functional outcome measure, resulting from changes to respiratory patterns with the idea that if treatment improved respiratory patterns, participants may be able to produce longer utterances. However, we recognize that changes at the level of the larynx as a result of therapy may also drive increased utterance lengths.

Lung Volume

The estimated lung volume measurements are expressed as a percentage of vital capacity. LVI was defined as the lung volume at which participants began speaking and was measured at the onset of the time-locked acoustic signal for each breath group. LVT was defined as the lung volume at which participants stopped speaking and was measured at the offset of the time-locked acoustic signal for each breath group. The speech initiation and termination points were verified by listening to the audio signal. LVI and LVT were measured relative to end-expiratory level (EEL) for each participant. To determine where EEL occurred, three consistent rest breaths were measured before the start of each monologue task. EEL was defined as the trough of each rest breath (bottom of tidal volume). An average of the three troughs was used as the EEL for measurement purposes. Positive LVI and LVT numbers reflect values above EEL, and negative numbers reflect values below EEL. Lung volume excursion (LVE) was defined as the lung volume expended during the breath group and was measured by subtracting LVT from LVI. For all study measures, the data measurers were blind to the participants' group assignment (LSVT LOUD, SpeechVive, and clinical control) and treatment session (pre, mid, and post).

Statistical Analysis

The mean SPL data for 34 participants were submitted to a 3 × 3 linear mixed-model analysis of variance (ANOVA; SAS 9.4). The between-subjects factor was group (three levels: LSVT LOUD, SpeechVive, and clinical control) with a within-subject factor of session (three levels: pre, mid, and post). For the LSVT LOUD speakers, COMF SPL data in all sessions were submitted to the model since the purpose of LSVT LOUD is to train a habitually louder voice. The SpeechVive is a prosthetic device, and it is not intended to elicit a training effect. As a result, the HIGH SPL conditions obtained at mid- and posttreatment were compared to the baseline COMF condition. For the clinical controls, COMF SPL data were submitted to the model, as these participants did not receive intervention. The clinical controls were included in the SPL analysis in order to confirm a positive intervention effect.

The utterance length and lung volume measures obtained for the 24 experimental participants were submitted to a 2 × 2 × 3 linear mixed-model ANOVA (SAS 9.4). The between-subjects factor was group (two levels: LSVT LOUD and SpeechVive), and the within-subject factors were condition (two levels: COMF and HIGH) and session (three levels: pre, mid, and post). The clinical controls were not included in these analyses, as the central aim of the study was to assess the respiratory adjustments used by speakers with PD in response to voice intervention.

For all statistical analyses, participant was included as a repeated effect in the model to account for expected intersubject differences in response to treatment. Tukey's honestly significant difference (HSD) post hoc tests were used to explore all significant main effects and interactions. Tukey's HSD adjusted p value was used to control for multiple comparisons. A significance level of .05 was

used for all statistical tests. Cohen's *d* effect-size statistics are reported for statistically significant comparisons.

Single-subject effect sizes were calculated to estimate the degree of change in each dependent variable for all session comparisons. Computation of single-subject effect sizes is recommended for single-subject treatment data to account for variability in intervention outcomes and to circumvent the inherent flaws in the use of visual analysis, which can lead to an inflated Type 1 error (Beeson & Robey, 2006; Busk & Serlin, 1992; Matyas & Greenwood, 1990). For the LSVT LOUD participants, computation of single-subject effect sizes reflects a session comparison of the COMF amplitude condition (pre COMF vs. mid COMF and post COMF). For the SpeechVive participants, the HIGH amplitude condition was used for the mid- and posttreatment comparisons, and the COMF amplitude condition was used for the baseline condition (pre COMF vs. mid HIGH and post HIGH). The single-subject effect size (*d* statistic) was calculated for each dependent measure by subtracting the session means and dividing this value by the standard deviation of the pre- or midtreatment session, depending on the comparison. Significant mid- and posttreatment changes were operationally defined as effect sizes greater than ±1.00 (Maas et al., 2012; Maas & Farinella, 2012). Positive effect sizes reflect an increase in the dependent measure at mid- or posttreatment. A negative effect size reflects a decrease in the dependent measure at mid- or posttreatment.

Reliability Measures

To assess intermeasurer reliability of SPL, utterance length, and the lung volume metrics, 10% of the data were selected for measurement by an independent examiner. A mean intraclass correlation coefficient (ICC) of .969 (ICC range: .949–.989) was reported across dependent variables, indicating excellent agreement between the original and independent examiners.

Results

Inferential statistics for all main and interaction effects are summarized in Table 2. Means and standard deviations are reported for each dependent measure in Table 3. Group effect sizes (*d*) for all significant pairwise comparisons are reported in text. Single-subject effect sizes related to treatment effects are reported in Table 4.

Table 2. Degrees of freedom, *F* statistics, and *p* values for all main effects and interactions.

Measure	Effect	num *df*	den *df*	*F*	*p*	sig
Sound pressure level	Group	2	31	4.34	.022	*
	Session	2	56	62.80	< .0001	*
	Group × Session	4	56	31.15	< .0001	*
Utterance length	Group	1	22	0.30	.587	
	Session	2	38	5.06	.011	*
	Condition	1	22	0.42	.524	
	Group × Session	2	38	0.91	.411	
	Group × Condition	1	22	0.21	.654	
	Session × Condition	2	38	0.39	.681	
	Group × Session × Condition	2	38	2.81	.073	
Lung volume initiation	Group	1	22	0.15	.700	
	Session	2	38	0.78	.466	
	Condition	1	22	1.04	.318	
	Group × Session	2	38	1.86	.170	
	Group × Condition	1	22	6.67	.017	*
	Session × Condition	2	38	4.71	.015	*
	Group × Session × Condition	2	38	2.04	.144	
Lung volume termination	Group	1	22	0.87	.361	
	Session	2	38	15.46	< .0001	*
	Condition	1	22	1.50	.233	
	Group × Session	2	38	1.43	.251	
	Group × Condition	1	22	2.49	.129	
	Session × Condition	2	38	7.47	.002	*
	Group × Session x Condition	2	38	5.21	.010	*
Lung volume excursion	Group	1	22	2.90	.103	
	Session	2	38	9.98	< .001	*
	Condition	1	22	0.06	.814	
	Group × Session	2	38	4.00	.027	*
	Group × Condition	1	22	1.04	.320	
	Session × Condition	2	38	0.42	.662	
	Group × Session × Condition	2	38	0.95	.397	

Note. An asterisk indicates values significant at α = .05. num *df* = numerator degrees of freedom; den *df* = denominator degrees of freedom.

Table 3. Means (and standard deviations) for each dependent measure reported by group (SpeechVive and LSVT LOUD), condition (comfortable intensity [COMF] and high intensity [HIGH]), and session (pre, mid, and post).

		Session and condition					
		Pre session		Mid session		Post session	
Respiratory measure	Group	COMF	HIGH	COMF	HIGH	COMF	HIGH
Sound pressure level (dB)	SpeechVive	77.85 (4.65)	79.30 (4.29)	78.80 (4.13)	80.67 (3.82)	77.69 (4.77)	81.83 (3.76)
	LSVT LOUD	75.89 (4.60)	78.89 (5.32)	77.88 (6.25)	78.57 (4.09)	78.79 (4.52)	81.23 (4.69)
	Control	81.07 (4.06)	—	79.55 (5.98)	—	80.09 (4.25)	—
Utterance length	SpeechVive	10.97 (7.43)	9.73 (6.38)	12.62 (7.89)	12.32 (7.05)	12.47 (7.80)	12.83 (8.51)
	LSVT LOUD	11.71 (7.41)	12.71 (9.47)	13.68 (8.37)	12.38 (7.70)	12.15 (8.48)	11.43 (7.29)
Lung volume initiation	SpeechVive	21.75 (13.31)	18.91 (11.86)	20.42 (16.96)	18.54 (21.13)	21.21 (11.41)	20.73 (8.90)
	LSVT LOUD	22.89 (20.14)	20.15 (16.73)	17.15 (22.03)	18.97 (22.60)	17.87 (15.81)	24.77 (28.73)
Lung volume termination	SpeechVive	6.41 (11.78)	5.72 (11.42)	2.48 (13.29)	0.66 (14.90)	5.41 (9.32)	5.37 (7.90)
	LSVT LOUD	5.84 (16.95)	0.63 (15.25)	–3.21 (18.98)	–0.98 (16.82)	–6.37 (16.75)	0.63 (19.94)
Lung volume excursion	SpeechVive	15.34 (10.21)	13.18 (8.15)	17.94 (13.43)	17.88 (15.51)	15.81 (12.24)	15.36 (9.68)
	LSVT LOUD	17.05 (12.79)	19.52 (16.09)	20.37 (15.05)	19.96 (15.79)	24.24 (20.72)	24.15 (17.60)

Note. Utterance length is reported in syllables per breath group. Lung volume initiation, termination, and excursion are reported as percentage of vital capacity, and initiation and termination are reported relative to end-expiratory level. Em dashes indicate condition data not collected for control participants. Pre = pretreatment; Mid = midtreatment (Week 4); Post = posttreatment (Week 8).

Figure 1 presents mean LVI, LVT, and LVE by group. Figure 2 presents eight individual subject box plots for LVI, LVT, and LVE to illustrate the variability in treatment response within and across experimental groups.

For SPL, there was a significant group ($p = .021$) and session ($p < .001$) effect. A significant difference in SPL was identified between the control and LSVT LOUD speakers, $t(31) = 2.78$, $p = .024$. Despite matching the groups for hypophonia severity level based on an auditory perceptual evaluation, the baseline COMF SPL was higher for the control participants ($M = 81.07$ dB, $SD = 4.88$), as compared to the LSVT LOUD participants ($M = 75.89$ dB, $SD = 5.12$). For the main effect of session, significantly higher SPL values were identified at midtreatment, $t(56) = 8.42$, $p < .001$, $d = 0.611$ ($M = 80.4$, $SD = 3.5$), and posttreatment, $t(56) = 10.42$, $p < .001$, $d = 0.929$ ($M = 81.5$, $SD = 3.4$), as compared to pretreatment ($M = 78.2$, $SD = 3.70$). A significant interaction effect was identified for group and session ($p < .001$). The SpeechVive participants demonstrated a significant increase in SPL pre- to midtreatment, $t(56) = 8.91$, $p < .001$, $d = 0.227$ ($M = +2.66$ dB), and pre- to posttreatment, $t(56) = 6.36$, $p < .001$, $d = 0.448$ ($M = +3.32$ dB). No significant change in SPL was identified mid- to posttreatment, $t(56) = 1.77$, $p = .700$ ($M = +0.65$ dB). For the LSVT LOUD participants, a significant change in SPL was identified for all session comparisons: pre- to midtreatment, $t(56) = 8.22$, $p < .001$, $d = 0.221$ ($M = +2$ dB); mid- to posttreatment, $t(56) = 5.13$, $p = .0001$, $d = 0.156$ ($M = +1$ dB); and pre- to posttreatment, $t(56) = 12.51$, $p < .001$, $d = 0.377$ ($M = +3$ dB). There was no significant change in SPL for the control participants for all session comparisons: pre- to midtreatment, $t(56) = 1.99$, $p = .557$ ($M = -0.41$ dB); mid- to posttreatment, $t(56) = 0.94$, $p = .989$ ($M = +0.12$ dB); or pre- to posttreatment, $t(56) = 0.97$, $p = .987$ ($M = -0.29$ dB). The single-subject effect sizes reported in Table 4 indicate that all experimental participants exhibited a significant and positive effect size for SPL for one or more session comparisons, but two participants in the LSVT LOUD group (M11 and M12) showed a significant decline in SPL for the mid- to postsession comparison.

For utterance length, there was no significant group ($p = .587$) or condition ($p = .524$) effect, but a significant session effect was identified ($p = .011$). A greater number of syllables per breath group were identified at midtreatment, $t(38) = 2.90$, $p = .017$, $d = 0.191$ ($M = 12.70$, $SD = 7.74$), and posttreatment, $t(38) = 2.49$, $p = .044$, $d = 0.133$ ($M = 12.27$, $SD = 8.02$), as compared to pretreatment ($M = 11.22$, $SD = 7.73$), but the effect sizes were small. No significant difference was identified mid- to posttreatment, $t(38) = 0.16$, $p = .986$. No interactions were statistically significant ($p > .05$). As shown in Table 4, six speakers (three LSVT LOUD and three SpeechVive) followed the group trend with a significant and positive effect size reported for utterance length for one or more session comparisons.

For LVI, there was no significant group ($p = .699$), session ($p = .467$), or condition ($p = .318$) effect. A significant interaction effect was identified for group and condition ($p = .017$) as well as session and condition ($p = .015$). However, none of the pairwise comparisons were significant after the conservative adjustment of alpha. No other interaction effects were found to be statistically significant ($p > .05$). The single-subject effect sizes reported in Table 4 indicate that 17 speakers exhibited a significant effect size for LVI for one or more session comparisons:

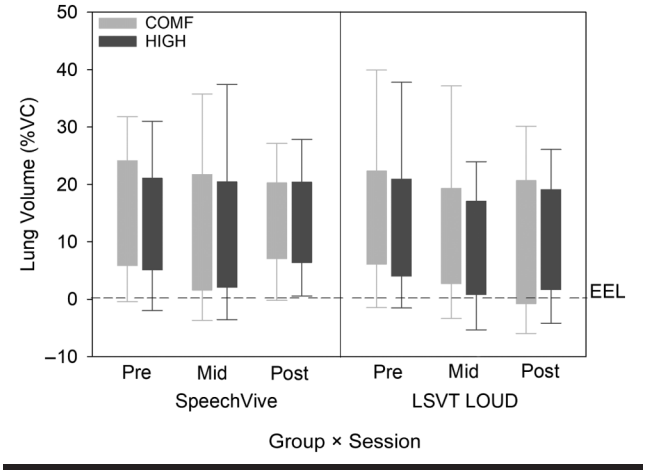
Table 4. Single-subject effect sizes are reported for multiple session comparisons.

		SPL			Utterance length			Lung volume initiation			Lung volume termination			Lung volume excursion		
ID	Group	Pre–Mid	Mid–Post	Pre–Post	Pre–Mid	Mid–Post	Pre–Post	Pre–Mid	Mid–Post	Pre–Post	Pre–Mid	Mid–Post	Pre–Post	Pre–Mid	Mid–Post	Pre–Post
F03	LSVT LOUD	2.15*	0.20	0.62	0.00	0.15	–0.67	–2.06*	1.50*	–0.34	–1.53*	–0.04	–0.04	0.17	–0.27	–0.28
F04	LSVT LOUD	0.23	1.52*	1.59*	0.35	0.15	0.52	0.59	–0.84	–1.02*	0.35	–1.43*	–0.78	–0.08	0.36	0.32
F05	LSVT LOUD	3.19*	—	—	1.61*	—	—	0.07	—	—	–0.57	—	—	0.82	—	—
M02	LSVT LOUD	2.03*	—	—	–0.03	—	—	–0.25	—	—	0.73	—	—	–1.02*	—	—
M03	LSVT LOUD	2.11*	1.08*	1.04*	0.10	–0.13	–0.02	–1.43*	1.05*	7.49*	–3.31*	0.94	–0.82	2.64*	–0.14	0.05
M04[a]	LSVT LOUD	–0.33	1.88*	2.31*	0.42	0.70	0.90	–5.19*	0.85	–2.95*	–7.19*	1.28*	–4.94*	0.93	0.19	1.29*
M08	LSVT LOUD	0.97	1.40*	2.13*	0.74	–0.29	0.34	–0.99	0.59	–0.50	–3.53*	1.51*	–1.77*	1.61*	–0.29	0.74
M09	LSVT LOUD	–0.01	1.02*	0.92	1.18*	–0.11	1.81*	0.94	–1.31*	–0.36	0.62	–1.29*	–0.51	0.26	0.11	0.11
M11	LSVT LOUD	2.70*	–2.20*	0.09	–0.59	0.70	–0.07	0.23	0.60	0.72	0.89	0.05	0.89	–0.46	0.35	–0.17
M12	LSVT LOUD	1.40*	–1.04*	0.51	1.42*	–0.91	0.28	–0.74	–1.00*	–1.85*	–1.47*	–1.07*	–2.45*	0.71	0.09	0.79
M13	LSVT LOUD	2.34*	—	—	0.57	—	—	1.04*	—	—	0.55	—	—	0.26	—	—
M14	LSVT LOUD	1.67*	—	—	0.00	—	—	0.94	—	—	1.22*	—	—	0.00	—	—
F01	SpeechVive	2.94*	0.34	2.94*	0.73	–0.30	0.35	2.94*	–0.75	–0.09	–0.24	0.30	0.30	2.02*	–0.83	–0.31
F02	SpeechVive	1.23*	–0.09	1.60*	0.93	–0.26	0.57	–2.49*	0.53	–2.18*	–1.37*	0.58	–0.96	–0.76	–0.22	–0.88
F46	SpeechVive	0.98	2.15*	3.33*	1.32*	–0.09	–0.04	–0.81	2.80*	1.29*	–0.30	2.36*	0.91	–0.38	1.27*	0.29
M01	SpeechVive	3.76*	1.22*	6.12*	–0.60	1.17*	0.75	2.84*	–0.79	–0.50	0.20	–0.35	–0.27	1.24*	–0.55	0.02
M05	SpeechVive	1.66*	0.34	1.88*	2.38*	0.33	1.39*	–1.48*	–1.03*	–1.75*	–1.03*	–2.88*	–3.35*	0.60	0.93	1.67*
M06	SpeechVive	7.64*	–0.23	1.43*	—	–0.66	—	—	0.09	—	—	0.19	—	—	–0.22	—
M07	SpeechVive	1.94*	1.09*	3.14*	0.40	0.28	0.73	2.56*	–2.15*	–1.65*	–0.04	–1.07*	–1.64*	2.45*	–0.81	0.49
M10	SpeechVive	3.46*	–0.27	3.07*	–1.06*	0.40	–0.90	–1.82*	0.27	–1.65*	–0.65	–0.53	–0.97	–0.49	0.67	–0.09
M15	SpeechVive	3.82*	–1.07*	1.76*	0.81	–0.18	0.63	–6.68*	1.86*	0.85	–4.51*	1.42*	–0.78	–0.14	0.97	1.13*
M43	SpeechVive	3.84*	–1.93*	2.20*	–0.44	–0.22	–0.60	0.57	0.56	1.18*	2.28*	0.80	1.44*	0.00	–0.52	–0.52
M45[a]	SpeechVive	0.16	3.18*	1.21*	0.72	0.05	0.78	1.14*	–0.73	1.18*	1.07*	2.57*	–0.58	0.82	–0.13	0.64
M48	SpeechVive	1.90*	—	—	0.12	—	—	–3.16*	—	—	–1.96*	—	—	–0.16	—	—

Note. The LSVT LOUD speakers reflect a comparison of the comfortable-intensity condition for all session comparisons. The SpeechVive speakers reflect a comparison of the comfortable-intensity condition obtained at pretreatment, as compared to the high-intensity condition (wearing activated SpeechVive prosthesis) at mid- and posttreatment. An asterisk indicates a significant effect size. Utterance length is reported in syllables per breath group. Lung volume initiation, termination, and excursion are reported as percentage of vital capacity, and initiation and termination are reported relative to end-expiratory level. Em dashes indicate session data are unavailable due to SARS-CoV-2 pandemic and/or data could not be reliably measured. The first character in the ID column denotes participant sex (M = male; F = female). SPL = sound pressure level; Pre = pretreatment; Mid = midtreatment; Post = posttreatment.

[a]Participant received deep brain stimulation to the subthalamic nucleus.

Figure 1. Lung volume initiation, termination, and excursion are shown by group (SpeechVive and LSVT LOUD), condition (comfortable intensity [COMF] and high intensity [HIGH]), and session (pretreatment [Pre], midtreatment [Mid], and posttreatment [Post]). Top of bars represent initiation. Bottom of bars represent termination. Vertical distance between initiation and termination represent excursion. Error bars indicate standard deviation for initiation and termination. All data are measured in percent vital capacity (%VC), and initiation and termination are reported relative to end-expiratory level (EEL).



Eight speakers (four LSVT LOUD and four SpeechVive) decreased LVI, five speakers (one LSVT LOUD and four SpeechVive) increased LVI, and four speakers (two LSVT LOUD and two SpeechVive) showed variable movement in LVI for the mid- and posttreatment sessions.

For LVT, there was no significant group ($p = .361$) or condition ($p = .233$) effect. There was a significant session effect ($p < .001$), with significantly lower LVT values reported posttreatment, $t(38) = -3.89$, $p = .001$, $d = -0.19$ ($M = 1.99$, $SD = 14.51$), and midtreatment, $t(38) = -5.31$, $p \leq .001$, $d = -0.30$ ($M = -0.22$, $SD = 16.16$), as compared to pretreatment ($M = 4.81$, $SD = 14.51$). A significant interaction effect was further identified for session and condition ($p = .0018$), with significantly lower LVT values reported for COMF SPL at posttreatment, $t(38) = -5.42$, $p < .0001$, $d = -0.39$ ($M = 0.60$, $SD = 14.10$), and midtreatment, $t(38) = -5.14$, $p < .0001$, $d = -0.41$ ($M = -0.30$, $SD = 16.53$), as compared to COMF SPL at pretreatment ($M = 6.13$, $SD = 14.51$). A significant three-way interaction effect was identified for group, session, and condition ($p = .010$). The LSVT LOUD participants exhibited a significantly lower LVT posttreatment, $t(38) = -5.81$, $p < .0001$, $d = -0.72$ ($M = -6.37$, $SD = 16.75$), and midtreatment, $t(38) = -5.12$, $p = .0005$, $d = -0.50$ ($M = -3.22$, $SD = 18.97$), as compared to pretreatment ($M = 5.84$, $SD = 16.95$), when speaking at their new habitual comfortable SPL. Furthermore, in the posttreatment session, the LSVT LOUD participants significantly increased their LVT when they were cued by the examiner to speak at a higher SPL ($M = 0.63$, $SD = 19.94$), as compared to speaking at comfortable SPL, $t(38) = -3.80$, $p = .0219$, $d = 0.38$ ($M = -6.37$, $SD = 16.75$). No significant differences in LVT were identified for the SpeechVive group for any statistical comparison. The single-subject effect sizes, reported in Table 4, indicate that 16 speakers exhibited a significant effect size for LVT for one or more session comparisons: Nine speakers (five LSVT LOUD and four SpeechVive) decreased LVT, four speakers (one LSVT LOUD and three SpeechVive) increased LVT, and three speakers (two LSVT LOUD and one SpeechVive) showed a variable response in LVT at mid- and posttreatment.

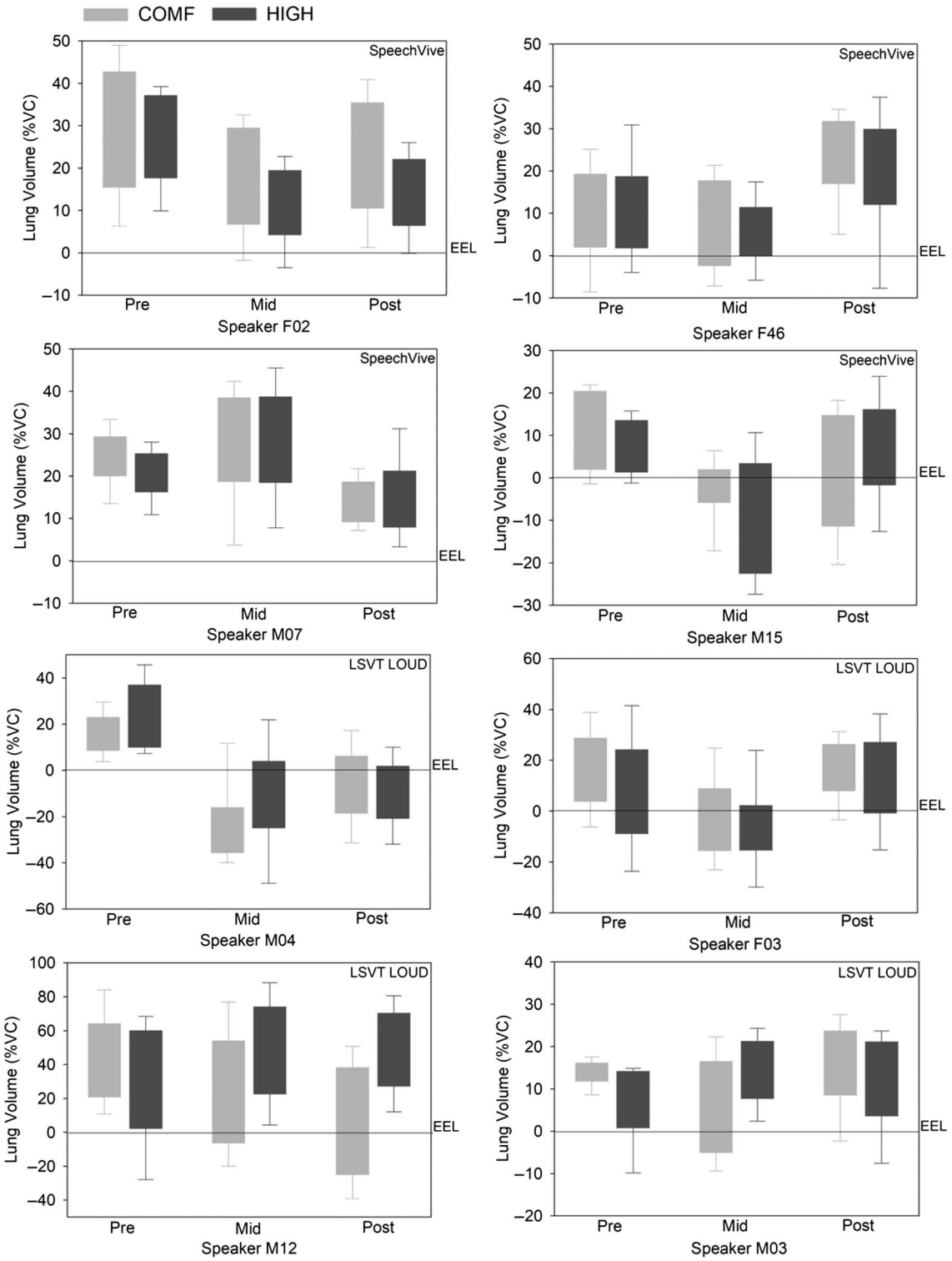
For LVE, there was no significant group ($p = .103$) or condition ($p = .814$) effect. A significant session effect was identified ($p < .001$), with significantly larger LVE values observed posttreatment, $t(38) = 3.18$, $p = .008$, $d = 0.23$ ($M = 19.30$, $SD = 15.56$), and midtreatment, $t(38) = 4.24$, $p < .001$, $d = 0.21$ ($M = 19.00$, $SD = 14.99$), as compared to pretreatment ($M = 16.14$, $SD = 12.16$). A significant Group × Session effect was also observed ($p = .027$). For the LSVT LOUD participants, significantly larger LVE values were observed posttreatment ($M = 24.19$, $SD = 18.99$), as compared to pretreatment, $t(38) = 3.37$, $p = .02$, $d = 0.36$ ($M = 18.20$, $SD = 14.44$). For the SpeechVive participants, significantly larger LVE values were observed at midtreatment, $t(38) = 3.82$, $p = .006$, $d = 0.30$ ($M = 17.91$, $SD = 14.53$), as compared to pretreatment ($M = 14.28$, $SD = 9.29$). No other interaction effects were found to be statistically significant ($p > .05$). Examination of the single-subject effect sizes indicates that nine speakers (three LSVT LOUD and six SpeechVive) exhibited a significant and positive effect size for LVE for one or more session comparisons. One LSVT LOUD participant showed a significant and negative effect size for LVE.

Discussion

The LSVT LOUD and SpeechVive participants significantly increased SPL as a result of treatment, with no change in SPL for the clinical controls. The observed increase in SPL for the experimental participants supports a positive intervention effect. The present SPL data are consistent with prior studies reporting vocal intensity gains in monologue speech after completing LSVT LOUD therapy (Ramig et al., 2018; Ramig, Sapir, Countryman, et al., 2001; Ramig, Sapir, Fox, & Countryman, 2001) or while wearing the SpeechVive prosthesis (Richardson et al., 2014; Stathopoulos et al., 2014).

Alterations in utterance length can be observed when a speaker adjusts vocal intensity. As the LSVT LOUD and SpeechVive participants increased their vocal intensity in response to treatment, utterance length

Figure 2. Lung volume initiation, termination, and excursion are shown for select SpeechVive and LSVT LOUD participants by condition (comfortable intensity [COMF] and high intensity [HIGH]) and session (pretreatment [Pre], midtreatment [Mid], and posttreatment [Post]). Top of bars represent initiation. Bottom of bars represent termination. Vertical distance between initiation and termination represent excursion. Error bars indicate standard deviation for initiation and termination. All data are measured in percent vital capacity (%VC), and initiation and termination are reported relative to end-expiratory level (EEL).



significantly increased at midtreatment by 1.48 syllables per breath group and at posttreatment by 1.05 syllables per breath group, as compared to pretreatment. Despite its statistical significance, the magnitude of the increase in utterance length is not considered clinically meaningful by the authors. The observed increase in utterance length may be attributed to improved respiratory patterns resulting in increased LVE or other physiological forces, such as increased laryngeal airway resistance at higher speech intensities. The utterance length means reported in Table 3 are congruent with prior data reported for individuals with PD (Darling-White et al., 2022; Huber & Darling, 2011), which provides further evidence of decreased utterance length in persons with PD, as compared to previously published normative data (Darling-White et al., 2022; Huber & Darling, 2011).

The present respiratory kinematic data are in accordance with prior studies reporting aberrations in speech breathing for persons with PD (Sadagopan & Huber, 2007; Solomon & Hixon, 1993; Stathopoulos et al., 2014). Although a healthy control group was not included for study, a comparison with published data indicates that the participants with PD demonstrate lower mean LVI values than previously described for healthy older adults (Huber & Darling, 2011). Initiating speech at a lower-than-normal lung volume increases the speakers' reliance on active expiratory muscle forces to generate the subglottal pressure necessary for speech production, thus increasing the work of breathing. Although there were no significant group-level effects for LVI at mid- or posttreatment, the single-subject effect sizes highlight the variability in LVI across participants. At mid- and posttreatment, eight speakers (four LSVT LOUD and four SpeechVive) decreased LVI, whereas five speakers (one LSVT LOUD and four SpeechVive) increased LVI at higher SPLs. Inhaling to a higher lung volume allows speakers to utilize the passive recoil forces of the chest wall system to produce adequate driving pressure for speech. Increasing LVI when speaking more loudly is the typical respiratory pattern demonstrated by both typical young and older adults (Hixon et al., 1973; Huber, 2007, 2008; Huber & Spruill, 2008; Stathopoulos & Sapienza, 1993) and has been interpreted as a more efficient pattern to support louder speech than using lower lung volumes and more expiratory muscle force.

In accordance with the study hypothesis, an effect of cue type was identified for speech breathing patterns in persons with PD after treatment. The LSVT LOUD participants terminated lung volume at a significantly lower level at mid- and posttreatment, as compared to pretreatment, when using the internal cue "Think Loud" to increase vocal intensity. Terminating speech at a lower-than-normal lung volume requires active engagement of the expiratory muscles, specifically the internal intercostal and abdominal muscles, in order to maintain adequate subglottal pressure as lung volume declines (Draper et al., 1959; Watson & Hixon, 1985) and may increase work of breathing or negatively affect the length of utterances. However, Bunton (2005) postulated that the use of positive abdominal pressure at lower-than-normal lung volumes may offer respiratory efficiency in the form of mechanical tuning of the RC. The significant decrease in LVT observed at mid- and posttreatment for the LSVT LOUD speakers contrasts the results of Huber et al. (2003), who found no significant difference in respiratory kinematic patterns pre– to post–LSVT LOUD. These incongruent findings may be attributed to the smaller sample size (n = 6), differences in disease severity, and the high degree of variability observed within and across the Huber et al. study participants. In contrast, the group-level data reported for the SpeechVive users indicated no significant change in respiratory patterns pre- to posttreatment.

The differences in speech breathing reported for the LSVT LOUD speakers, as compared to the SpeechVive users, support an effect of cue strategy on respiratory mechanics. The effect of cue strategy on speech breathing may be explained, in part, by the neural processes underpinning goal-directed behavior. Previous studies have implicated the role of the dorsomedial striatum, a known site of impairment in PD, in mediating goal-directed behaviors (Haruno & Kawato, 2006; Levy & Dubois, 2006), such as the online monitoring and adjustment of vocal intensity in the LSVT LOUD program. Although cue strategy has received some attention in the rehabilitation literature in studies of locomotion (Harrison et al., 2018; Park et al., 2021; Rubinstein et al., 2002), the mechanisms through which cue strategy impacts motor function are not well understood.

There is speculation that the use of external cues reduces the attentional and cognitive resources necessary to complete a task (Brouwers et al., 2016). This viewpoint is not without controversy, however, as it has been countered that the use of external cues may impart a cognitive load that requires additional attentional resources (Nanhoe-Mahabier et al., 2012). Furthermore, it has been suggested that internally generated cues function as dual-task interference resulting in a decline in task performance (Chawla et al., 2014; O'Shea et al., 2002). This notion of dual-task interference is consistent with prior studies of persons with PD, which have reported a significant and adverse effect of cognitive load on postural stability (Holmes et al., 2010), gait performance (Penko et al., 2018), and foot tapping (Brown & Marsden, 1984) under dual-task conditions. In accordance with the capacity-sharing model (Friedman et al., 1982), these study findings suggest that persons with PD have reduced central processing resources, and when demand exceeds capacity, there is a concomitant decline in task performance. The LSVT LOUD program aims to

reduce the cognitive load of therapy by having the patients overlearn routine phrases, which can then serve as an automatic cue for vocal intensity regulation. If motor automaticity can be achieved by LSVT LOUD patients, then vocal intensity regulation can be performed with minimal cognitive resources and interference. It is possible that the LSVT LOUD speakers, in this study, did not achieve motor automaticity at the end of treatment and therefore continued to require attentional and neural resources during vocal intensity adjustments. A recent report from this study found that speakers with PD reported significantly higher levels of mental demand after 4 and 8 weeks of LSVT LOUD treatment, as compared to the SpeechVive users (Richardson et al., 2022). These data are consistent with prior neuroimaging studies that have reported impaired motor automaticity in many patients with PD, even at a relatively early disease stage (Wu et al., 2010, 2015; Wu & Hallett, 2005). A deeper understanding of shifts in attentional load and prioritization during cued speech is of particular importance for individuals with PD, given the widely reported cognitive deficits (Aarsland et al., 2017; Brown & Marsden, 1984; Litvan et al., 2011; Richards et al., 1993; Van Spaendonck et al., 1996). Future studies may consider examining changes in speech breathing under internal and external cueing conditions in a group of patients with diverse cognitive profiles to better elucidate the underlying mechanisms of cognitive interference.

Last, the authors would like to note that the variability in speech breathing patterns used to support louder speech, as reflected in Figure 2 and Table 4, is consistent with prior studies of persons with PD (Bunton, 2005; Huber et al., 2003; Stathopoulos et al., 2014) and healthy young adults (Stathopoulos & Sapienza, 1993). The individual differences in respiratory strategies used to increase vocal intensity further highlight the importance of examining single-subject responses in intervention studies and considering patient-specific variables when designing voice rehabilitation programs.

Limitations

Several study limitations are noted. First, this study assigned a relatively small sample of 12 persons with hypophonia to each intervention group. We must therefore exercise caution in generalizing the results to a larger clinical population. Furthermore, the use of auditory-perceptual judgments to match groups for hypophonia severity level resulted in vocal intensity differences for the controls and the LSVT LOUD speakers. Although the effect of hypophonia severity on treatment outcomes was mitigated by the use of a within-subject statistical design, future studies may consider the use of a quantitative metric or consensus ratings to report hypophonia severity. Also, given the known heterogeneity of PD symptomology and prior reports of variability (Huber et al., 2003; Richardson et al., 2014), an extended baseline period should be considered in future studies. Last, although respiratory kinematic data are reported for lung volume displacement, abdominal volume contributions to lung volume changes remain unknown for the present group of speakers. During pilot testing, the participants with PD were unable to consistently complete the abdominal calibration maneuvers necessary for reporting abdominal volume contributions. The participants had difficulty coordinating the required parts of the maximum abdominal in and out tasks while also holding their breath. For many participants, the maximum abdominal out maneuver was particularly difficult and resulted in maximum outward movements that were smaller than those achieved during tidal breathing. It is likely they were contracting their abdominal muscles in an attempt to expand their AB, but the muscle contraction worked against the expansion.

Future studies may also consider including a temporal measure of coordination between the thoracic and abdominal systems to further elucidate the respiratory strategies used by speakers with PD during vocal intensity regulation. Thoraco-abdominal asynchrony has been extensively reported in studies of persons with PD (Florêncio et al., 2019; Huber et al., 2003; Solomon & Hixon, 1993), but the effect of voice intervention on RC–abdominal coupling remains unknown. Furthermore, although adjustments in vocal intensity are primarily mediated through respiratory drive in older adults, vocal intensity changes are also supported through the mechanistic action of the laryngeal system. Laryngeal aerodynamic data are not reported in this article, so the relative contribution of the laryngeal system in supporting vocal intensity increases remains unknown. Last, given the progressive and degenerative nature of PD, future studies may consider investigating longer term respiratory changes in response to voice intervention. Behavioral voice interventions are designed to promote maintenance of vocal intensity gains after therapy, but it is unclear if the respiratory strategies used by speakers change with continued vocal practice and how disease progression affects these clinical gains.

Conclusions

This study provides the first comparative evaluation of two voice intervention approaches on speech breathing for persons with PD. Although healthy speakers typically increase vocal intensity by increasing LVI and LVT to take advantage of higher recoil forces, our findings suggest that persons with PD may have difficulty with this type of respiratory adjustment. This is consistent with the longitudinal data in PD showing that LVI and LVT decline as the disease progresses (Darling-White et al., 2022; Huber &

Darling, 2017). The LSVT LOUD speakers significantly decreased LVT while speaking at their new habitual speech volume, thus suggesting reliance on expiratory muscle strength and increased work of breathing when speaking at a higher vocal intensity. In contrast, participants in the SpeechVive group did not alter their respiratory strategies in response to voice intervention. The present data suggest the LSVT LOUD and SpeechVive therapies elicit different respiratory adjustments in persons with PD. The increased respiratory effort, observed in the LSVT LOUD group, may have consequences for the long-term maintenance of therapy outcomes and therapeutic adherence for patients with more compromised muscle function. In addition, the individual subject data are consistent with prior reports of variability in treatment response for persons with PD. The single-subject variability highlights the need for customization of voice intervention approaches based on the clinical presentation of motor and nonmotor deficits. These study findings have important implications for the clinical management of hypophonia. Given the extensive use of inspiratory and expiratory muscles to support louder speech, clinicians may need to consider integrating direct treatment of respiratory muscle strength into their plan of care.

Author Contributions

Kelly Richardson: Conceptualization, Funding acquisition, Methodology, Supervision, Writing – original draft, Formal analysis, Writing – review & editing. **Jessica E. Huber:** Conceptualization, Funding acquisition, Methodology, Supervision, Writing – original draft, Formal analysis, Writing – review & editing. **Brianna Kiefer:** Data curation, Writing – reviewing & editing. **Caitlin Kane:** Formal analysis, Writing – review & editing. **Sandy Snyder:** Data curation, Supervision, Writing – review & editing.

Data Availability Statement

De-identified data are available on request from the authors.

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Appendix

Subject-Specific Calibration Factors and Fit Statistics

		Pre session				Mid session				Post session			
ID	Group	Calibration type	RC factor	AB factor	Mean error	Calibration type	RC factor	AB factor	Mean error	Calibration type	RC factor	AB factor	Mean error
F03	LSVT LOUD	RC&AB	0.652	0.451	0.077	RC&AB	0.075	1.023	0.069	RC&AB_abs	1.212	0.573	0.235
F04	LSVT LOUD	RC&AB	0.926	0.473	0.082	RC&AB	1.089	0.183	0.067	RC&AB	1.489	0.343	0.060
F05	LSVT LOUD	RC&AB	0.920	0.837	0.098	RC&AB	1.135	0.624	0.125	—	—	—	—
M02	LSVT LOUD	RC&AB_abs	1.695	0.027	0.100	RC&AB_abs	1.289	0.194	0.097	—	—	—	—
M03	LSVT LOUD	RC&AB	0.842	2.604	0.052	RC&AB	1.085	1.475	0.053	RC&AB	2.061	0.754	0.080
M04[a]	LSVT LOUD	RC&AB	2.433	0.200	0.081	RC&AB_abs	1.048	2.122	0.080	RC&AB	0.302	1.317	0.069
M08	LSVT LOUD	RC&AB	1.393	0.654	0.087	RC&AB	1.142	0.870	0.086	RC&AB_abs	1.243	0.301	0.125
M09	LSVT LOUD	RC&AB	0.727	0.887	0.095	RC&AB	1.424	0.147	0.091	RC&AB	1.214	0.281	0.112
M11	LSVT LOUD	RC&AB	0.691	0.885	0.080	RC&AB	1.331	0.009	0.080	RC&AB_abs	1.718	0.250	0.134
M12	LSVT LOUD	RC&AB	2.147	0.935	0.099	RC&AB_abs	2.811	3.030	0.144	RC&AB	2.124	0.729	0.092
M13	LSVT LOUD	RC&AB_abs	1.330	2.667	0.385	RC&AB	1.009	0.079	0.112	—	—	—	—
M14	LSVT LOUD	RC&AB	0.396	0.880	0.070	RC&AB	1.232	0.412	0.070	—	—	—	—
F01	SpeechVive	RC_only	1.622	1.000	0.180	RC&AB	0.483	0.692	0.052	RC&AB_abs	1.585	0.347	0.081
F02	SpeechVive	RC&AB	0.930	0.376	0.141	RC&AB	1.172	0.466	0.064	RC&AB	0.539	0.734	0.117
F46	SpeechVive	RC&AB	1.577	0.978	0.052	RC&AB	1.358	0.963	0.044	RC&AB_abs	2.079	0.035	0.042
M01	SpeechVive	RC&AB	1.226	1.957	0.072	RC&AB_abs	2.809	0.467	0.098	RC&AB	1.106	3.062	0.087
M05	SpeechVive	RC&AB	0.979	0.236	0.076	RC&AB	1.327	0.033	0.092	RC&AB_abs	1.159	0.986	0.124
M06	SpeechVive	—	—	—	—	RC&AB	0.816	0.222	0.115	RC&AB	0.489	0.557	0.093
M07	SpeechVive	RC&AB_abs	3.192	1.039	0.241	RC&AB_abs	2.00	0.303	0.094	RC&AB	2.159	0.192	0.076
M10	SpeechVive	RC&AB	2.180	1.053	0.179	RC&AB	1.767	1.253	0.119	RC&AB	2.059	1.184	0.167
M15	SpeechVive	RC&AB	1.785	0.679	0.073	RC&AB	0.695	1.301	0.067	RC&AB	0.812	1.416	0.075
M43	SpeechVive	RC&AB	1.949	0.675	0.037	RC&AB	1.459	0.726	0.054	RC&AB	1.045	1.640	0.059
M45[a]	SpeechVive	RC&AB	2.603	0.259	0.112	RC&AB	2.141	2.346	0.088	RC&AB	2.531	1.257	0.072
M48	SpeechVive	RC&AB	0.916	0.186	0.177	RC&AB	0.914	0.662	0.045	—	—	—	—

Note. Mean error values are expressed in liters. Em dashes indicate session data are unavailable due to SARS-CoV-2 pandemic and/or data could not be reliably measured. The first character in the ID column denotes participant sex (M = male; F = female). Pre = pretreatment; Mid = midtreatment; Post = posttreatment; RC = rib cage; AB = abdomen; RC&AB = calibration of both the rib cage and abdomen; RC&AB_abs = absolute values from the calibration of the rib cage and abdomen; RC_only = calibration of the rib cage only, with abdomen held at 1.

[a]Participant received deep brain stimulation to the subthalamic nucleus.