Title: Longitudinal effects of Parkinson disease on speech breathing during an extemporaneous connected speech task

Authors: Meghan Darling-White, Ph.D.[a], Zeina Anspach, M.S.[b] and Jessica E. Huber, Ph.D.[b]

Affiliation:

[a]University of Arizona, Department of Speech, Language, and Hearing Sciences

Tucson, AZ 85721

[b]Purdue University, Department of Speech, Language, and Hearing Sciences

West Lafayette, IN 47907

Corresponding author:

Meghan Darling-White

University of Arizona

1131 E. 2nd Street

Tucson, AZ 85704

Email: darlingwhite@email.arizona.edu

Phone: (520) 626-6460

Running title (not exceeding 45 letters and spaces): Longitudinal Changes to Speech Breathing in PD

Abstract

Purpose: A critical component to the development of any type of intervention to improve speech production in individuals with Parkinson disease is a complete understanding of the speech impairments present at each stage of the disease and how these impairments change with disease progression. The purpose of the current longitudinal study was to examine the impact of disease on speech production and speech breathing during an extemporaneous speech task in individuals with Parkinson disease over the course of approximately 3.5 years.

Method: Eight individuals with Parkinson disease and 8 age- and sex- matched control participants produced an extemporaneous connected speech task on two occasions (Time 1 and Time 2) an average of 3 years and 7 months apart. Dependent variables included: sound pressure level, utterance length, speech rate, lung volume initiation, termination, and excursion, and percent vital capacity per syllable.

Results: From Time 1 to Time 2, individuals with Parkinson disease demonstrated decreased utterance length and lung volume initiation, termination, and excursion and increased speech rate. Control participants demonstrated decreased utterance length and lung volume termination and increased lung volume excursion and percent vital capacity per syllable from Time 1 to Time 2.

Conclusions: Changes in speech production and speech breathing variables experienced by individuals with PD over the course of several years are related to their disease process and not typical aging. Changes to speech breathing highlight the need to provide intervention focused on increasing efficient respiratory patterning for speech production.

Key words: longitudinal, Parkinson's disease, speech, breathing, respiratory kinematics

Introduction

Speech impairment is a hallmark characteristic of Parkinson disease (PD), with 70-90% of individuals demonstrating speech impairment at some point in the disease process (Ho et al., 1999; Logemann et al., 1978). Many of the most common speech impairments associated with PD, including reduced vocal loudness, short utterances, and long pauses can be attributed to deficits in speech breathing due to disease-related changes to the respiratory system. Disease-related respiratory impairments include pulmonary function restrictions (De Pandis et al., 2002), increased chest wall rigidity (Sabaté et al., 1996; Solomon & Hixon, 1993), and reductions in respiratory muscle strength and coordination (De Bruin et al., 1993; Haas et al., 2004; Pitts et al., 2008; Weiner et al., 2002). Individuals with PD produce lower subglottal pressures (Hammer & Barlow, 2010; Matheron et al., 2017) which are likely to result from both reduced respiratory force and reduced vocal fold closure and may result in lower vocal intensity. Reduced expiratory muscle strength can result in longer pauses since these muscles are important for holding the diaphragm at rest length, allowing for a quick and deep inspiration (Hixon et al., 1973). As a result of these physiologic changes, individuals with PD rely more on active respiratory muscle forces than passive recoil forces to produce speech which increases the work of speech breathing and causes fatigue (Bunton, 2005; Huber & Darling, 2011; Huber & Darling-White, 2017; Solomon & Hixon, 1993).

Despite these well-established respiratory impairments, the vast majority of literature concerning speech production and interventions designed to improve speech production in individuals with PD have focused on the articulatory and laryngeal systems. Given that these systems are heavily influenced by the performance of the respiratory system, the development and validation of interventions specifically designed to improve speech breathing in individuals

with PD is imperative (Darling-White & Huber, 2017; Huber & Darling-White, 2017). A critical component to the development of any type of intervention to improve speech production in individuals with PD is a complete understanding of the speech impairments present at each stage of the disease and how these impairments change across time. Even though a longitudinal research design is best suited for this task, the majority of research regarding speech production in individuals with PD is cross-sectional. This is likely due to the inherent challenges with the execution of longitudinal work (Darling-White & Huber, 2020a). However, the cross-sectional approach often leads to variable findings across studies potentially due to differences in disease progression between samples. For example, individuals with PD may initiate and terminate speech at higher than normal or lower than normal lung volumes (Bunton, 2005; Darling-White & Huber, 2017; Huber & Darling, 2011; Sadagopan & Huber, 2007; Solomon & Hixon, 1993). Vocal loudness in individuals with PD, as measured by sound pressure level (SPL), may by reduced or equivalent to healthy older adults (Canter, 1963; Ho et al., 2000; Huber & Darling, 2011; Ramig et al., 2001). Utterance length, the number of syllables produced on one breath, in individuals with PD may be shorter than or equivalent to healthy older adults (Bunton, 2005; Huber et al., 2012; Huber & Darling, 2011; Solomon & Hixon, 1993). Given that the development of intervention approaches requires an understanding of habitual speech production and speech breathing patterns, this variability is problematic.

The small number of longitudinal studies examining speech production in individuals with PD consistently demonstrate worsening impairment in the acoustic and perceptual aspects of speech production over time (Darling-White & Huber, 2020a; Huber & Darling-White, 2017; Miller et al., 2011; Skodda et al., 2009, 2011, 2013). However, only two longitudinal studies have included speech breathing variables (Darling-White & Huber, 2020a; Huber & Darling-

White, 2017), both examining a set of individuals with PD and age- and sex-matched control participants producing a reading passage at two time points. Individuals with PD spoke with faster speech rates but, equivalent vocal loudness and utterance length, at Time 2 as compared to control participants (Huber & Darling-White, 2017). Individuals with PD also initiated and terminated speech at lower lung volumes as compared to control participants, particularly at Time 2 (Huber & Darling-White, 2017). Given that passive recoil forces are lower at lower lung volumes, initiating and terminating speech at lower lung volumes requires greater active muscle forces to generate the necessary pressure to produce speech. Thus, individuals with PD expend more effort during speech production, particularly as the disease progresses. Additionally, individuals with PD produced fewer breath pauses at major syntactic boundaries and periods and more breath pauses at locations with no punctuation, particularly at Time 2. Changes to breath pause patterns were significantly related to ratings of speech impairment such that fewer breath pauses at major syntactic boundaries and more breath pauses at locations unrelated to syntax were related to ratings of more severe speech impairment (Darling-White & Huber, 2020a).

While the inclusion of speech breathing variables within a longitudinal study is an important step toward the development of interventions that improve speech impairments in individuals with PD at each stage of the disease process, these variables have only been examined within the context of a reading passage. The cognitive-linguistic demands of a speech task, however, differentially impact speech breathing in healthy individuals as well as individuals with PD (Huber & Darling, 2011; Mitchell et al., 1996). In comparison to a reading task, an extemporaneous speech task has greater language formulation demands and a lack of visual cues provided by punctuation to help plan appropriate breath pauses. Speech breathing impairments observed in individuals with PD are larger during an extemporaneous speech task due to the

simultaneous demands of planning and coordinating respiratory support and language for each utterance (Huber & Darling, 2011). Further, it is important to examine the impact of disease progression during an extemporaneous speech task since this task is more ecologically valid.

The purpose of this longitudinal study was to examine the impact of disease on speech production and speech breathing during an extemporaneous speech task in individuals with PD. We examined patterns of change over time in age- and sex-matched control participants as well to differentiate disease-related changes from age-related ones. We addressed the following research questions.

1. Does speech production during an extemporaneous speech task change over time as a result of PD? We expected disease-related changes to speech production, specifically decreased SPL, decreased utterance length, and increased speech rate.
2. Do speech breathing patterns during an extemporaneous speech task change over time as a result of PD? We predicted that disease-related changes to speech breathing would manifest as decreases in lung volume initiation and termination.

These data will be crucial to the development of interventions specifically designed to improve speech breathing in individuals with PD. At this time, classification of motor symptoms in relation to disease progression focuses almost exclusively on limb motor function. However, declines in the speech production system of individuals with PD do not correlate with declines in limb motor function (Ash et al., 2017; Skodda et al., 2009, 2011, 2013). These data may also assist with the establishment of markers that healthcare providers can use to classify disease progression in the speech production system.

Methods

Research Design

This longitudinal study included acoustic and respiratory kinematic data from two data collection sessions, hereafter referred to as Time 1 and Time 2, that occurred in the Motor Speech Laboratory at Purdue University an average of 3 years; 7 months (SD = 6.5 months) apart. Data collection procedures were approved by the Purdue University Institutional Review Board. Informed consent was obtained from each participant following verbal and written presentation of study procedures at both time points. Data from Time 1, excluding F07PD, were included in previously published data sets as these data were initially collected as part of a larger cross-sectional study (Huber, 2008; Huber & Darling, 2011). Monologue data from Time 2 have not been published. Data from a reading task produced by these participants at both Time 1 and 2 have been published previously (Darling-White & Huber, 2020b; Huber & Darling-White, 2017).

Participants

Participants included eight individuals (4 men, 4 women) diagnosed with idiopathic PD by a neurologist and eight age- and sex-matched healthy adults. F07PD was initially recruited as a control participant, but her data were atypical for the healthy adult group and were excluded from analyses. At Time 2, F07PD disclosed that she had been diagnosed with PD. The pathophysiologic changes characteristic of PD begin years prior to clinically noticeable symptoms (Bernheimer et al., 1973). In fact, changes to speech production have been noted up to 5-years prior to diagnosis (Harel et al., 2004). Thus, we chose to include F07PD in the longitudinal data set presented in Huber and Darling-White (2017), Darling-White and Huber (2020), and the current study.

Individuals with PD were paired with an age- and sex-matched control participant at Time 1. Only those pairs for which we obtained acoustic and respiratory kinematic data at a

second data collection point (i.e., Time 2) for both the individual with PD and the age- and sex-matched control participant were included in this longitudinal study. At Time 1, the mean age of the participants with PD was 72;9 (years;months; *SD* = 4;1) and the mean age of the control participants was 72;10 (*SD* = 4;7). At Time 2, the mean age of the participants with PD was 76;3 (*SD* = 4;0) and the mean age of control participants was 76;7 (*SD* = 4;6). All participants reported that they were Caucasian and non-Hispanic except one participant who chose not to report race or ethnicity. Participants with PD were tested during the self-reported "on" state of their medication cycle (within 1-3 hours of taking their PD medication). Demographic information for both groups is presented in Table 1.

At Time 1, no participants reported a history of respiratory problems (including asthma) or neurological disease (except PD), head, neck, or chest cancer or surgery, or formal training in singing or speaking. All participants had been nonsmokers for at least the past 5 years, except M10PD who ceased smoking 1 year prior to Time 1, were ambulatory and living independently in the community, demonstrated adequate cognition as measured by a score of 24 or above on the Mini-Mental State Examination (Folstein et al., 1975), passed a bilateral hearing screening at 40dB HL for 500 Hz, 1000 Hz, and 2000 Hz (Ventry & Weinstein, 1983), except for M04PD who did not pass at 40dB HL for 2000 Hz in the right ear, and were free of infections, colds, and allergies. Additionally, control participants demonstrated normal speech, language, and voice as determined by participant self-report and the investigators. Only one participant reported receiving speech-language therapy services within 20 years of Time 1. M09PD participated in weekly group speech therapy with a focus on speaking more clearly for 2 years prior to Time 1.

At Time 2, most participants met the original inclusion criteria, with exceptions noted below. M04PD underwent deep brain stimulation (DBS). F01PD had a possible transient

ischemic attack one year prior, but diagnosis was not confirmed. Two participants with PD (M09PD and M10PD) demonstrated cognitive decline from Time 1 to Time 2 based on the Cognitive-Linguistic Quick Test (Helm-Estabrooks, 2001) composite score. M07OC demonstrated losses at 40dB HL for 1000 Hz and 2000 Hz in the right ear. No control participants reported receiving speech-language therapy services between Time 1 and Time 2. Three participants with PD received some speech-language therapy between Time 1 and Time 2; F01PD had some therapy for her possible TIA; F02PD and M04PD had therapy but could not describe the goals of the therapy except it was to help their speech. .

Using a visual analog scale, with one end labeled “normal” and the other end labeled “very severe”, two certified speech-language pathologists, who were not affiliated with the study, completed ratings of speech impairment for both groups at each time point using the middle 30 s of the extemporaneous speech task. Samples started and stopped at sentence boundaries. Ratings were completed with no knowledge of disease state or time point. The speech samples were intensity normalized and presented over headphones. The SLPs listened to each sample one time and rated speech severity using a visual analog scale with one end marked “normal” and one end marked “very severe.” Samples were blocked by speaker sex and randomized such that no two samples from the same participant were presented consecutively. Percent of speech impairment was calculated by measuring the distance from “normal” to the rating mark in millimeters, then dividing by the total length of the line in millimeters and multiplying by 100. Ratings from the two speech-language pathologists were averaged. If the difference between the two ratings was greater than 20%, a third rating by the third author was obtained and the two closest ratings were than averaged. This occurred in seven instances.

Ratings of speech impairment serve as a descriptive measure and were not included in any statistical comparisons.

Equipment and Data Collection Procedure

Acoustic data were collected using high-quality condenser microphones. The mouth-to-microphone distance was 6-in at Time 1 and 6-cm at Time 2. At time 2, the microphone was head worn, and at both time points, the mouth-to-microphone distance was checked several times during the session to ensure that it was held constant. The microphone signal was recorded to a digital audiotape and then digitized at 44.1 kHz, resampled at 18 kHz, and low-pass filtered at 9 kHz via Praat (Boersma & Weenink, 2003). The microphone was calibrated by collecting a signal of known intensity (94 dB) emitted from a pure tone generator with the microphone and digital audio recorder used to collect acoustic data at the same gain levels used in the data collection sessions. The microphone was calibrated before each participant. The difference between the measured intensity of the calibration signal in TF32 (Milenkovic, 2003) and the known intensity was calculated and added to the intensity measures for the speech samples collected in the session associated with each calibration. The differences in mouth-to-microphone distance were corrected using the Inverse Square Law so that data reflected a 6-cm distance.

Respiratory kinematic data were recorded via respiratory inductive plethysmography (Inductotrace system, Ambulatory Monitoring, Ardley, NY). Two elastic bands were placed on the participants to transduce the movement of the rib cage and abdomen. Rib cage movement was captured via an elastic band placed around the rib cage right under the axilla. Abdominal movement was captured via an elastic band placed around the abdomen below the last rib at the level of the belly button.

Participants performed maneuvers necessary to calibrate the respiratory kinematic signals for lung volume estimation and to determine vital capacity (VC) prior to performing any speech tasks. For the lung volume estimation calibration, participants completed two tasks: rest breathing and speech-like breathing with respitrace bands in place and while breathing into a digital spirometer (VacuMed Universal Ventilation Meter). For the speech-like breathing task, participants read a short sentence silently to themselves one time per breath. The purpose of the speech-like breathing task was to elicit larger lung volume ranges, one similar to speech utterances, to increase the accuracy of the estimates of lung volume. Correction factor (k1 in formula 1 below) for the rib cage was computed using the following formula from data from the rest breathing and speech-like breathing tasks. The correction factor for the abdomen was set to 1.

$$\text{Lung volume (from spirometer)} = \text{Rib Cage}*k1 + \text{Abdomen} \quad [1]$$

Estimated lung volume was calculated using a variant of the least squares method that has been validated for older adults and individuals with PD (Chadha et al., 1982; McKenna & Huber, 2019). During speech tasks, the estimated lung volume was calculated using the rib cage and abdominal movement and the correction factors using the following formula.

$$\text{Estimated lung volume} = \text{Rib Cage}*k1 + \text{Abdomen} \quad [2]$$

These methods have been published elsewhere (e.g., Huber & Darling-White, 2017; McKenna & Huber, 2019; Stathopoulos et al., 2014).

Participants completed vital capacity tasks both with and without the respiratory bands in place. Prior to band placement, participants completed a slow vital capacity and a forced vital capacity maneuver using a VacuMed Discovery Handheld Spirometer. In the slow vital capacity task, participants were cued to inspire to the top of their lung volume and then expire as much as

possible. In the forced vital capacity task, they were cued inspire to the top of their lung volume and then expire as quickly and forcefully as possible. The slow vital capacity task was completed first and then the forced vital capacity task. The purpose of these tasks was to measure lung function. Vital capacity, forced vital capacity, and forced expiratory volume in one second are reported in Appendix 1. With the bands in place, three more slow vital capacity maneuvers were completed. Slow vital capacity for this purpose was estimated from the rib cage and abdominal signals, using the same formula for estimating lung volume during the speech tasks. The largest of the three was used to normalize the lung volume measures, such that lung volume initiation, termination, excursion were expressed as a percent of vital capacity.

Speech Task

Using their comfortable pitch and loudness, participants spoke about a topic of their choice for approximately two minutes. Participants were asked to complete several different speech tasks during each data collection session. The extemporaneous speech task described in the current study was typically produced in the middle of each data collection session.

Measurements

The acoustic signal obtained during each extemporaneous speech task was orthographically transcribed by two research team members. Discrepancies were resolved via consensus. An utterance was defined as the speech produced during one breath. The average SPL, in decibels (dB), of each utterance was calculated using TF32 (Milenkovic, 2003). Utterance length was defined as the number of syllables produced during one breath. Speech rate was calculated by dividing the number of syllables in each utterance by its duration.

Respiratory kinematic measurements were made using custom algorithms written to run in MATLAB (The MathWorks). Using the time-locked acoustic signal, lung volume initiation

and lung volume termination were defined as the points in the lung volume signal where speech started and stopped for each utterance. Lung volume excursion was defined as the amount of lung volume expired during an utterance and was calculated by subtracting lung volume termination from lung volume initiation. Lung volume measurements were expressed as a percentage of VC and relative to end expiratory level, the rest position of the respiratory system. End expiratory level was measured as the average trough value of at least three rest breaths prior to the start of the task.

The %VC per syllable was calculated by dividing lung volume excursion by utterance length. Occasionally, participants exhaled when no speech was being produced within an utterance (i.e. pausing and exhaling within a breath group, before the utterance is completed). Periods of exhalation while no speech was being produced were visually evident in the respiratory kinematic signal and were verified by the acoustic signal. These utterances were excluded when measuring data for %VC per syllable because it would have skewed the calculation, but were included for all other measures. Approximately 15 – 20% of the utterances from each group were excluded.

Statistical Analysis

A linear mixed model analysis of variance was used with the factors: group (PD vs. control participant) as the between factor, time (Time 1 vs. Time 2) as the within factor, and participant as a random factor. Tukey's honestly significant difference post-hoc tests were used to determine statistically significant interaction effects. The alpha level for all tests was set at $p < .05$. Individual effect sizes (d_2) for each participant were calculated to determine how many participants demonstrated the same pattern of behavior as group-level statistics. The d_2 statistic was calculated by dividing the mean difference between Time 1 and Time 2 by the pooled

standard deviation across Time 1 and Time 2 (Busk & Serlin, 1992). Positive effect sizes indicated that the variable increased from Time 1 to Time 2. Negative effect sizes indicated that the variable decreased from Time 1 to Time 2. The strength of the effect size based on the *d* statistic was interpreted as very small below .2, small from .2-.5, medium from .5-.8, large from .8-1.2, very large from 1.2-2.0, and huge over 2.0 (Sawilowsky, 2009). For the purposes of determining individual-level differences in patterns, with respect to the group-level statistics, we considered effect sizes over .2.

Intermeasurer reliability for Time 1 was reported in previous publications (Huber, 2008; Huber & Darling, 2011). Intermeasurer reliability for Time 2 data was determined from three participants randomly chosen for remeasurement. Interclass Correlation Coefficients (ICC) were used to assess inter-measurer reliability. ICC were calculated in SPSS using a two-way mixed model, assessing absolute agreement for single point measures. ICC values ranged from .611 to .906, suggesting good inter-measurer reliability (see Appendix 2) (Koo & Li, 2016).

Results

Statistical analyses were conducted on a total of 397 utterances for Time 1 (PD = 187 utterances, control participants = 210 utterances) and 417 utterances for Time 2 (PD = 204 utterances, control participants = 213 utterances). Individuals with PD produced an average of 23 utterances at Time 1 and 25 utterances at Time 2. Control participants produced an average of 26 utterances at Time 1 and Time 2. Statistical analyses for main and interaction effects are presented in Table 2 for each dependent variable. Pairwise comparisons for significant interaction effects are presented in Table 3. Group means and standard errors for each dependent variable are presented by time in Table 4. Individual effect sizes are presented in Table 5. Figure 1 provides a graphic representation of the speech breathing data.

SPL

There was a significant main effect of time ($F = 17.15$, $p < .001$) and a significant interaction effect of group by time ($F = 11.85$, $p = .001$). There was no significant effect of group. There was a significant increase in SPL from Time 1 to Time 2. This was driven by a significant increase in SPL for control participants from Time 1 to Time 2. SPL increased significantly from Time 1 to Time 2 for the controls ($p < .001$), but there was no significant change in the PD group ($p = .960$). Four of the eight control participants followed this trend, all with effect sizes over 2.0. For individuals with PD, there were a mix of increases and decreases, mostly in the small to medium effect size range. M04PD, who had deep-brain stimulator implantation surgery between Time 1 and 2, had a large effect size decrease in SPL.

Utterance Length

There was a significant main effect of time ($F = 4.83$, $p = .030$), but no significant main effect of group or interaction effect of group by time. Utterance length significantly decreased from Time 1 to Time 2. Mean data indicate this decrease occurred in both groups over time, with a greater mean decrease in individuals with PD. Four of the eight control participants and five of the eight individuals with PD followed this trend. For the individuals with PD, effect sizes were mostly small and all effect sizes demonstrated a reduction in utterance length. For the control participants, the effect sizes were mostly small and only one participant (M07OC) demonstrated an increase in utterance length.

Speech Rate

There was a significant main effect of time ($F = 4.29$, $p = .039$) and a significant interaction effect of group X time ($F = 20.18$, $p < .001$). There was no significant effect of group. There was a significant increase in speech rate from Time 1 to Time 2. This effect was primarily

driven by the individuals with PD. Speech rate was significantly faster for individuals with PD at Time 2 as compared to Time 1 ($p < .001$), but there were no significant changes for control participants ($p = .300$). There were no differences in speech rate between the groups at Time 1 ($p = .970$). Individuals with PD produced a significantly faster speech rate than control participants at Time 2 ($p = .020$). Five of the eight individuals with PD increased their speech rate, mostly with medium to very large effect sizes, but one person with PD (F04PD) decreased speech rate across sessions. For the control participants, there were a range of effect size values, but only 2 of the 8 control participants increased speech rate.

Lung Volume Initiation

There was a significant main effect of time ($F = 54.43$, $p < .001$) and a significant interaction effect of group by time ($F = 17.97$, $p < .001$). There was no significant effect of group. Individuals with PD initiated speech at significantly lower lung volumes at Time 2 as compared to Time 1 ($p < .001$), but there were no significant changes for control participants ($p = .110$). Seven of the eight individuals with PD followed this trend with mostly medium to large effect sizes; one participant with PD (F02PD) increased LVI. There was a mix of changes in the control participants with three increasing LVI and three decreasing LVI.

Lung Volume Termination

There was a significant main effect of time ($F = 59.55$, $p < .001$), but no significant main effect of group or interaction effect of group by time. Both individuals with PD and control participants terminated speech at significantly lower lung volumes at Time 2 as compared to Time 1, with a greater mean decrease in individuals with PD. Five of the eight individuals with PD followed this trend with mostly very large effect sizes; three participants (F02PD, F07PD,

and M09PD) had increases in LVT. Four of the eight control participants decreased LVT mostly with medium to large effect sizes; two control participants increased LVT.

Lung Volume Excursion

There were no significant main effects of group or time, but there was a significant interaction effect of group by time ($F = 21.21$, $p < .001$). Individuals with PD used a significantly smaller amount of lung volume at Time 2 as compared to Time 1 ($p = .004$). Seven of the eight individuals with PD followed this trend, mostly with medium to large effect sizes; one participant increased LVE (M10PD). In contrast, control participants used a significantly larger amount of lung volume at Time 2 as compared to Time 1 ($p = .010$). Five of the eight control participants followed this trend, mostly with small effect sizes; only two control participants decreased LVE.

%VC per syllable

There were no significant main effects of group or time, but there was a significant interaction effect of group by time ($F = 16.31$, $p < .001$). Control participants expended more lung volume per syllable at Time 2 as compared to Time 1 ($p = .001$), but there were no significant changes for individuals with PD ($p = .220$). Six of the eight control participants followed this trend, mostly with medium to large effect sizes. For individuals with PD, five participants decreased %VC/syllable and two increased, with a range of effect sizes.

Discussion

The purpose of this longitudinal study was to examine the impact of PD on speech production and speech breathing during an extemporaneous speech task. Differences in the patterns of change across time between individuals with PD and control participants indicate that individuals with PD experience disease-related changes to their speech production and breathing.

Specifically, there were differences between the groups in the patterns of change from Time 1 to Time 2 for SPL, speech rate, lung volume initiation and excursion, and %VC per syllable.

Several of the disease-related changes, including increased speech rate and decreased lung volume initiation and termination, are consistent with our previous longitudinal work (Huber & Darling-White, 2017), suggesting that these changes are present in individuals with PD regardless of speech task. It is unsurprising that speech rate increased regardless of speech task as a fast rate of speech is a common auditory-perceptual feature of speech impairment in individuals with PD (Darley et al., 1969). However, the finding that decreased lung volume initiation and termination is present in both reading and extemporaneous speech is significant. This confirms that individuals with PD expend more effort during speech production, an issue that seems to worsen over time. These findings were very consistent across participants in the PD group. Seven of the eight participants with PD decreased lung volume initiation from Time 1 to Time 2, one with a small effect size, two with medium effect sizes, two with large effect sizes, and two with huge effect sizes. Five of the eight participants with PD demonstrated decreased lung volume termination, one with a small effect size, one with a large effect size, two with very large effect sizes, and one with a huge effect size. These individual data support the group-level statistics, suggesting that most individuals with PD are using lower lung volumes than age- and sex-matched individuals during speech production.

Efficient speech breathing patterns rely on a balance between passive recoil and active muscle forces. To reduce the work of speech breathing, the balance of these forces tends to skew in favor of the use of more passive recoil than active muscle forces during expiration. Since passive recoil forces are higher at higher lung volume, the use of lower lung volume initiations and terminations, as seen in individuals with PD, results in an overreliance on active muscle

forces, resulting in increased work of speech breathing. While control participants also demonstrated decreased lung volume termination, increasing the use of active muscle forces to produce speech likely impacts individuals with PD more negatively given the disease-related decrements in force and coordination of the respiratory muscles (De Bruin et al., 1993; Haas et al., 2004; Pitts et al., 2008; Weiner et al., 2002). More importantly, this finding sheds light on the underlying reason behind changes to speech breathing patterns in individuals with PD. Two primary theories have emerged to explain disease-related downward shifts in lung volume initiation and termination: 1) changes to respiratory physiology, like increased chest wall rigidity, and 2) difficulty planning and coordinating respiratory support and language for each utterance (Huber & Darling-White, 2017). Downward shifts in lung volume initiation and termination regardless of speech task suggest that changes to respiratory physiology drive changes to speech breathing patterns. Very few participants had pulmonary function test results outside of normal limits (see Appendix 1), so it is unlikely that reduced capacity drove changes to speech breathing patterns. Increased chest wall rigidity has been hypothesized before as a driver of speech breathing changes in PD (Solomon & Hixon, 1993). If chest wall rigidity increased for individuals with PD from Time 1 to Time 2, it may have become more difficult to expand the chest wall, leading to reduced lung volume initiations and terminations.

Decreases in utterance length and lung volume excursion at Time 2 in individuals with PD are unique to the extemporaneous speech task. Both control participants and individuals with PD demonstrated decreases in utterance length over time. In fact, seven of the eight individuals with PD demonstrated a reduction in utterance length from Time 1 to Time 2, although only five of them had changes that reflected a small effect size or larger. Age-related decreases in utterance length from young adulthood to older adulthood are well-documented (Hoit & Hixon,

1987; Huber, 2008; Sperry & Klich, 1992). However, this is the first study to demonstrate that age-related decreases in utterance length occur across a relatively short amount of time (3-4 years). Six of the eight control participants demonstrated a reduction in utterance length from Time 1 to Time 2, although only four of them had changes that reflected a small effect size or larger. Given that Huber and Darling-White (2017) did not find significant decreases in utterance length at Time 2 for either group during a reading task, our findings suggest that decreases in utterance length are influenced by the task itself. Healthy older adults and individuals with PD produce longer utterances during extemporaneous speech tasks than during reading tasks (Huber & Darling, 2011). When comparing the mean data for utterance length from this study and Huber and Darling-White (2017), the mean differences between extemporaneous speech and reading were smaller at Time 2 than at Time 1. It is possible that the utterance lengths produced during the extemporaneous speech task at Time 1 were too physiologically taxing at Time 2 given the age- and disease-related changes to respiratory physiology. Thus, utterance length decreased at Time 2 to fit within the constraints of the respiratory system.

Given that utterance length decreased across time in individuals with PD, it is not surprising that lung volume excursion decreased. Individual difference data supports this interpretation since all participants that had a reduction in utterance length also showed a reduction in lung volume excursion. Two of the control participants followed the trend of reduced utterance length coupled with reduced lung volume excursion. In both healthy adults and individuals with PD, lung volume excursion is positively correlated with utterance length (Huber & Darling, 2011). However, for the control participants, utterance length decreased but lung volume excursion increased, possibly due to age-related reductions in vocal fold valving, allowing more pressure and airflow to leak through the vocal folds (as indexed by increased

%VC per syllable) (Hoit & Hixon, 1987; Sperry & Klich, 1992). Two of the control participants showed decreased utterance lengths coupled with increased lung volume excursion and increased %VC/syllable.

Vocal loudness, as measured by SPL, was not significantly impacted by disease over time. It is possible that the lack of change was the result of the difference in mouth-to-microphone distance in wave 2 relative to wave 1, despite the correction we applied based on the inverse square law. However, this finding is consistent with a growing body of evidence that PD does not necessarily result in decreased SPL (Holmes et al., 2000; Huber & Darling-White, 2017; Ramig et al., 2001). Lack of change in SPL across several years may seem counterintuitive to reports that decreased vocal loudness is a commonly reported auditory-perceptual characteristic of the speech produced by individuals with PD, particularly in later stages of the disease (Ho et al., 1999; Logemann et al., 1978). In fact, the majority of interventions to improve speech production in individuals with PD focus on increasing vocal loudness (Fox et al., 2002; Stathopoulos et al., 2014). Further research is needed to interrogate the relationship between subjective ratings of reduced vocal loudness and objective measurements of SPL. It is possible that other common speech/voice characteristics such as breathiness contribute more to the perception of reduced vocal loudness than the raw intensity of the signal in individuals with PD.

Clinical Implications

The findings of reduced lung volume initiations, terminations, and excursions and reduced utterance length over time due the disease-process highlight the need for speech-language pathologists to consider the respiratory system as a treatment target when working with individuals with PD. The respiratory system produces the steady, driving pressure necessary for speech production. The use of lower lung volumes during speech production results in an

overreliance on active muscle forces during speech production. This coupled with reduced respiratory muscle strength results in increased effort and fatigue during speech production. Since fatigue is a major contributor to decreased satisfaction with communicative participation in individuals with motor speech disorders (Yorkston et al., 2012), it is crucial individuals with PD receive intervention directly related to improving speech breathing patterns. Unfortunately, our field has a long way to go in this area. The most commonly used speech intervention for individuals with PD is the Lee Silverman Voice Treatment (LSVT) program. The only study of speech breathing patterns pre-post LSVT did not demonstrate improvements to speech breathing after intervention, although this study was small (Huber et al., 2003). The intervention with the most data to support its use for improving respiratory function in individuals with PD is expiratory muscle strength training which has been shown to normalize speech breathing patterns (Darling-White & Huber, 2017) and to result in improved cough strength (Sapienza et al., 2011; Troche et al., 2010).

Limitations

The primary limitation of this work is the small sample size. Longitudinal studies that involve individuals experiencing a degenerative disease are particularly difficult to implement in a large-scale, controlled manner given the likelihood of death and/or significant disability and the inability to control variables related to disease management (e.g., type of medication, surgery, participation in rehabilitation services). Small, fairly heterogenous cohorts are to be expected in this type of work. While this may limit the generalizability of a single longitudinal study, the more longitudinal work that is conducted the better able the field will be to develop a complete picture of the disease process.

Another limitation of this work is that one individual with PD had DBS implanted between the two data collection sessions. Given that only one patient had this surgery in the cohort, it is difficult to determine how much of the change seen for that participant was due to PD progression versus DBS. However, individual effect sizes demonstrate changes were quite large and negative. Thus, it is likely that DBS did have a significant and detrimental effect on speech production and speech breathing for this participant. Future longitudinal studies examining the effects of DBS on speech breathing would be beneficial.

Conclusions

Disease-related changes to speech production and speech breathing are evident individuals with PD over 3-4 years. In general, the extemporaneous speech task did not appear to exaggerate disease-related changes as was originally hypothesized. Consistency in the results across reading and extemporaneous speech tasks for both groups suggests that age- and disease-related changes are driven predominately by physiologic changes to the respiratory system as opposed to the coordination between the respiratory and cognitive-linguistic systems. This longitudinal study provides further evidence for the potential benefit of interventions specifically designed to improve speech breathing in individuals with PD, especially during later stages of the disease. These findings also suggest that speech breathing patterns in individuals with PD should be frequently assessed to monitor the need for direct intervention.

Acknowledgements

We would like to thank the participants involved in this study and Jay Wolstencroft, Meghan MacPherson, and Bharath Chandrasekaran for assistance with data collection. Research reported in this publication was supported by Grant R03DC05731 from the National Institutes of Health, National Institute on Deafness and Other Communication Disorders, a Research Support Incentive Grant from the Center on Aging and the Life Course at Purdue University, and a Summer Faculty Support Grant from Purdue University all of which were awarded to the second author (Jessica E. Huber). Darling-White was supported by the National Institutes of Health, National Institute on Deafness and Other Communication Disorders under Award T32DC000030 (awarded to Elizabeth A. Strickland). 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, the Center on Aging and the Life Course, or Purdue University.

References

Ash, S., Jester, C., York, C., Kofman, O. L., Langey, R., Halpin, A., Firn, K., Dominguez Perez, S., Chahine, L., Spindler, M., Dahodwala, N., Irwin, D. J., McMillan, C., Weintraub, D., & Grossman, M. (2017). Longitudinal decline in speech production in Parkinson's disease spectrum disorders. *Brain and Language*, *171*, 42–51. https://doi.org/10.1016/j.bandl.2017.05.001

Bernheimer, H., Birkmayer, W., Hornykiewicz, O., Jellinger, K., & Seitelberger, F. (1973). Brain dopamine and the syndromes of Parkinson and Huntington Clinical, morphological and neurochemical correlations. *Journal of the Neurological Sciences*, *20*(4), 415–455. https://doi.org/10.1016/0022-510X(73)90175-5

Boersma, P., & Weenink, D. (2003). *Praat* (Version 4.1) [Computer software]. Phonetic Sciences, University of Amsterdam. http://www.praat.org/

Bunton, K. (2005). Patterns of lung volume use during an extemporaneous speech task in persons with Parkinson disease. *Journal of Communication Disorders*, *38*(5), 331–348. https://doi.org/10.1016/j.jcomdis.2005.01.001

Busk, P. L., & Serlin, R. (1992). Meta-analysis for single case research. In *Single-case research design and analysis: New directions for psychology and education* (pp. 187–212). Lawrence Erlbaum Associates.

Canter, G. J. (1963). Speech characteristics of patients with Parkinson's disease: I. Intensity, pitch, and duration. *Journal of Speech and Hearing Disorders*, *28*(3), 221–229.

Chadha, T., Watson, H, Birch, S, Jenouri, GA, Schneider, AW, Cohn, MA, & Sackner, MA. (1982). Respiratory inductive plethysmographyusing different calibration procedures. *The American Review of Respiratory Disease*, *125*(6), 644–649.

Darley, F. L., Aronson, A. E., & Brown, J. R. (1969). Differential diagnostic patterns of dysarthria. *Journal of Speech, Language, and Hearing Research*, *12*(2), 246–269. https://doi.org/10.1044/jshr.1202.246

Darling-White, M., & Huber, J. E. (2017). The impact of expiratory muscle strength training on speech breathing in individuals with Parkinson's disease: A preliminary study. *American Journal of Speech-Language Pathology*, *26*(4), 1159–1166. https://doi.org/10.1044/2017_AJSLP-16-0132

Darling-White, M., & Huber, J. E. (2020a). The impact of Parkinson's disease on breath pauses and their relationship to speech impairment: A longitudinal study. *American Journal of Speech-Language Pathology*, 1–13. https://doi.org/10.1044/2020_AJSLP-20-00003

Darling-White, M., & Huber, J. E. (2020b). The Impact of Parkinson's Disease on Breath Pauses and Their Relationship to Speech Impairment: A Longitudinal Study. *American Journal of Speech-Language Pathology*, *29*(4), 1910–1922. https://doi.org/10.1044/2020_AJSLP-20-00003

De Bruin, P. F. C., De Bruin, V. M. S., Lees, A. J., & Pride, N. B. (1993). Effects of treatment on airway dynamics and respiratory muscle strength in Parkinson's disease. *American Review of Respiratory Disease*, *148*(6_pt_1), 1576–1580. https://doi.org/10.1164/ajrccm/148.6_Pt_1.1576

De Pandis, M. F., Starace, A., Stefanelli, F., Marruzzo, P., Meoli, I., De Simone, G., Prati, R., & Stocchi, F. (2002). Modification of respiratory function parameters in patients with severe Parkinson's disease. *Neurological Sciences*, *23*(Suppl 2), S69–S70. https://doi.org/10.1007/s100720200074

Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). “Mini-mental state”: A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research*, *12*(3), 189–198. https://doi.org/10.1016/0022-3956(75)90026-6

Fox, C. M., Morrison, C. E., Ramig, L. O., & Sapir, S. (2002). Current perspectives on the Lee Silverman Voice Treatment (LSVT) for individuals with idiopathic Parkinson disease. *American Journal of Speech-Language Pathology*, *11*(2), 111–123. https://doi.org/10.1044/1058-0360(2002/012)

Haas, B. M., Trew, M., & Castle, P. C. (2004). Effects of respiratory muscle weakness on daily living function, quality of life, activity levels, and exercise capacity in mild to moderate Parkinson’s disease. *American Journal of Physical Medicine & Rehabilitation*, *83*(8), 601–607. https://doi.org/10.1097/01.PHM.0000133436.61009.02

Hammer, M. J., & Barlow, S. M. (2010). Laryngeal somatosensory deficits in Parkinson’s disease: implications for speech respiratory and phonatory control. *Experimental Brain Research*, *201*(3), 401–409. https://doi.org/10.1007/s00221-009-2048-2

Harel, B., Cannizzaro, M., & Snyder, P. J. (2004). Variability in fundamental frequency during speech in prodromal and incipient Parkinson’s disease: A longitudinal case study. *Brain and Cognition*, *56*(1), 24–29. https://doi.org/10.1016/j.bandc.2004.05.002

Helm-Estabrooks, N. (2001). *Cognitive Linguistic Quick Test*. Harcourt Assessment.

Hixon, T. J., Goldman, M. D., & Mead, J. (1973). Kinematics of the Chest Wall during Speech Production: Volume Displacements of the Rib Cage, Abdomen, and Lung. *Journal of Speech Language and Hearing Research*, *16*(1), 78. https://doi.org/10.1044/jshr.1601.78

Ho, A. K., Bradshaw, J. L., & Iansek, R. (2000). Volume perception in Parkinsonian speech. *Movement Disorders*, *15*(6), 1125–1131. https://doi.org/10.1002/1531-8257(200011)15:6<1125::AID-MDS1010>3.0.CO;2-R

Ho, A. K., Iansek, R., Marigliani, C., Bradshaw, J. L., & Gates, S. (1999). Speech impairment in a large sample of patients with Parkinson's disease. *Behavioural Neurology*, *11*(3), 131–137. https://doi.org/10.1155/1999/327643

Hoit, J. D., & Hixon, T. J. (1987). Age and speech breathing. *Journal of Speech and Hearing Research*, *30*(3), 351–366. https://doi.org/10.1044/jshr.3003.351

Holmes, R. J., Oates, J. M., Phyland, D. J., & Hughes, A. J. (2000). Voice characteristics in the progression of Parkinson's disease. *International Journal of Language & Communication Disorders*, *35*(3), 407–418. https://doi.org/10.1080/136828200410654

Huber, J. E. (2008). Effects of utterance length and vocal loudness on speech breathing in older adults. *Respiratory Physiology & Neurobiology*, *164*(3), 323–330. https://doi.org/10.1016/j.resp.2008.08.007

Huber, J. E., & Darling, M. (2011). Effect of Parkinson's disease on the production of structured and unstructured speaking tasks: Respiratory physiologic and linguistic considerations. *Journal of Speech, Language, and Hearing Research*, *54*(1), 33–46. https://doi.org/10.1044/1092-4388(2010/09-0184)

Huber, J. E., Darling, M., Francis, E. J., & Zhang, D. (2012). Impact of typical aging and Parkinson's disease on the relationship among breath pausing, syntax, and punctuation. *American Journal of Speech-Language Pathology*, *21*(4), 368–379. https://doi.org/10.1044/1058-0360(2012/11-0059)

Huber, J. E., & Darling-White, M. (2017). Longitudinal changes in speech breathing in older adults with and without Parkinson's disease. *Seminars in Speech and Language*, *38*(3), 200–209. https://doi.org/10.1055/s-0037-1602839

Huber, J. E., Stathopoulos, E. T., Ramig, L. O., & Lancaster, S. L. (2003). Respiratory function and variability in individuals with Parkinson disease: Pre- and post-Lee Silverman Voice Treatment. *Journal of Medical Speech-Language Pathology*, *11*(4), 185–201.

Koo, T. K., & Li, M. Y. (2016). A Guideline of Selecting and Reporting Intraclass Correlation Coefficients for Reliability Research. *Journal of Chiropractic Medicine*, *15*(2), 155–163. https://doi.org/10.1016/j.jcm.2016.02.012

Logemann, J. A., Fisher, H. B., Boshes, B., & Blonsky, E. R. (1978). Frequency and cooccurrence of vocal tract dysfunctions in the speech of a large sample of Parkinson patients. *Journal of Speech and Hearing Disorders*, *43*(1), 47. https://doi.org/10.1044/jshd.4301.47

Matheron, D., Stathopoulos, E. T., Huber, J. E., & Sussman, J. E. (2017). Laryngeal Aerodynamics in Healthy Older Adults and Adults With Parkinson's Disease. *Journal of Speech Language and Hearing Research*, *60*(3), 507–524. https://doi.org/10.1044/2016_JSLHR-S-14-0314

McKenna, V. S., & Huber, J. E. (2019). The accuracy of respiratory calibration methods for estimating lung volume during speech breathing: A comparison of four methods across three adult cohorts. *Journal of Speech, Language, and Hearing Research*, *62*(8), 2632–2644. https://doi.org/10.1044/2019_JSLHR-S-18-0478

Milenkovic, P. (2003). *Time-Frequency Analysis (TF32)*.

Miller, N., Andrew, S., Noble, E., & Walshe, M. (2011). Changing perceptions of self as a communicator in Parkinson's disease: A longitudinal follow-up study. *Disability and Rehabilitation*, *33*(3), 204–210. https://doi.org/10.3109/09638288.2010.508099

Mitchell, H. L., Hoit, J. D., & Watson, P. J. (1996). Cognitive-linguistic demands and speech breathing. *Journal of Speech and Hearing Research*, *39*(1), 93–104. https://doi.org/10.1044/jshr.3901.93

Pitts, T., Bolser, D., Rosenbek, J., Troche, M., & Sapienza, C. (2008). Voluntary cough production and swallow dysfunction in Parkinson's disease. *Dysphagia*, *23*(3), 297–301. https://doi.org/10.1007/s00455-007-9144-x

Ramig, L. O., Sapir, S., Fox, C., & Countryman, S. (2001). Changes in vocal loudness following intensive voice treatment (LSVT) in individuals with Parkinson's disease: A comparison with untreated patients and normal age-matched controls. *Movement Disorders*, *16*(1), 79–83. https://doi.org/10.1002/1531-8257(200101)16:1<79::AID-MDS1013>3.0.CO;2-H

Sabaté, M., González, I., Ruperez, F., & Rodríguez, M. (1996). Obstructive and restrictive pulmonary dysfunctions in Parkinson's disease. *Journal of the Neurological Sciences*, *138*(1–2), 114–119. https://doi.org/10.1016/0022-510X(96)00003-2

Sadagopan, N., & Huber, J. E. (2007). Effects of loudness cues on respiration in individuals with Parkinson's disease. *Movement Disorders*, *22*(5), 651–659. https://doi.org/10.1002/mds.21375

Sapienza, C., Troche, M., Pitts, T., & Davenport, P. (2011). Respiratory Strength Training: Concept and Intervention Outcomes. *Seminars in Speech and Language*, *32*(01), 021–030. https://doi.org/10.1055/s-0031-1271972

Sawilowsky, S. S. (2009). New Effect Size Rules of Thumb. *Journal of Modern Applied Statistical Methods*, *8*(2), 597–599. https://doi.org/10.22237/jmasm/1257035100

Skodda, S., Flasskamp, A., & Schlegel, U. (2011). Instability of syllable repetition as a marker of disease progression in Parkinson's disease: A longitudinal study. *Movement Disorders*, *26*(1), 59–64. https://doi.org/10.1002/mds.23382

Skodda, S., Grönheit, W., Mancinelli, N., & Schlegel, U. (2013). Progression of voice and speech impairment in the course of Parkinson's disease: A longitudinal study. *Parkinson's Disease*, *2013*, 1–8. https://doi.org/10.1155/2013/389195

Skodda, S., Rinsche, H., & Schlegel, U. (2009). Progression of dysprosody in Parkinson's disease over time-A longitudinal study. *Movement Disorders*, *24*(5), 716–722. https://doi.org/10.1002/mds.22430

Solomon, N. P., & Hixon, T. J. (1993). Speech breathing in Parkinson's disease. *Journal of Speech and Hearing Research*, *36*(2), 294–310. https://doi.org/10.1044/jshr.3602.294

Sperry, E. E., & Klich, R. J. (1992). Speech breathing in senescent and younger women during oral reading. *Journal of Speech, Language, and Hearing Research*, *35*(6), 1246–1255. https://doi.org/10.1044/jshr.3506.1246

Stathopoulos, E. T., Huber, J. E., Richardson, K., Kamphaus, J., DeCicco, D., Darling, M., Fulcher, K., & Sussman, J. E. (2014). Increased vocal intensity due to the Lombard effect in speakers with Parkinson's disease: Simultaneous laryngeal and respiratory strategies. *Journal of Communication Disorders*, *48*, 1–17. https://doi.org/10.1016/j.jcomdis.2013.12.001

Troche, M. S., Okun, M. S., Rosenbek, J. C., Musson, N., Fernandez, H. H., Rodriguez, R., Romrell, J., Pitts, T., Wheeler-Hegland, K. M., & Sapienza, C. M. (2010). Aspiration and

swallowing in Parkinson disease and rehabilitation with EMST: A randomized trial. *Neurology*, *75*(21), 1912–1919. https://doi.org/10.1212/WNL.0b013e3181fef115

Ventry, I. M., & Weinstein, B. E. (1983). *Identification of elderly people with hearing problems*. *25*(7), 37–42.

Weiner, P., Inzelberg, R., Davidovich, A., Nisipeanu, P., Magadle, R., Berar-Yanay, N., & Carasso, R. L. (2002). Respiratory muscle performance and the perception of dyspnea in Parkinson's disease. *Canadian Journal of Neurological Sciences / Journal Canadien Des Sciences Neurologiques*, *29*(1), 68–72. https://doi.org/10.1017/S031716710000175X

Yorkston, K. M., Bamer, A., Johnson, K., & Amtmann, D. (2012). Satisfaction with participation in multiple sclerosis and spinal cord injury. *Disability and Rehabilitation*, *34*(9), 747–753. https://doi.org/10.3109/09638288.2011.619615

Table 1. Participant demographic information

Pair	Participant	Age at Time 1 (years; months)	Age at Time 2 (years; months)	Years Since Diagnosis (Time 1)	Medications at Time 1	Medications at Time 2	CLQT at Time 2	Speech Impairment at Time 1 (%)	Speech Impairment at Time 2 (%)
1	F01PD	72;5	76;3	0.75	Mirapex, Prozac	Sinemet, Mirapex, Prozac, Buspirone, Aspirin	WNL	8.5	50.5
	F13OC	73;5	76;9	n/a	Atenolol, Norvasc, Lipitor	Atenolol, Norvasc, Lipitor, Fosamax	WNL	4.5	2.5
2	F02PD	69;9	72;2	9.0	Sinemet, Eldepryl, Clinoril, Zoloft, Welbutrin, Maxide, Tylenol	Sinemet, Eldepryl, Clinoril, Zoloft, Lipitor, Avapro, Inderal, Detrol, Aspirin	WNL	12.0	43.0
	F07OC	65;8	69;7	n/a	None	Estrace	WNL	1.0	1.0
3	F07PD	72;2	75;11	-3.0^	None	Prilosec	WNL	3.5	10.0
	F02OC	74;5	78;6	n/a	Procardia, Avapro, Amaryl, Glucophage	Procardia, Avapro, Amaryl, Glucophage	WNL	1.3	4.7

Note. F = female, M = male; PD = Parkinson disease, OC = control participant; CLQT = composite score of the Cognitive-Linguistic Quick Test (Helm-Estabrooks, 2001); WNL = within normal limits; ^ diagnosed with PD three years after Wave 1; higher numbers indicate more severe speech ratings for speech severity.

Table 1. Participant demographic information, continued

Pair	Participant	Age at Wave 1 (years; months)	Age at Wave 2 (years; months)	Years Since Diagnosis (Wave 1)	Medications at Wave 1	Medications at Wave 2	CLQT at Wave 2	Speech Impairment at Wave 1 (%)	Speech Impairment at Wave 2 (%)
4	F04PD	74;3	76;11	5.0	Sinemet, Eldepryl, Bromocriptine	Sinemet, Bromocriptine, Zelapar	WNL	2.8	11.4
	F05OC	73;1	77;1	n/a	Lipitor	Lipitor, Diazepam	WNL	3.5	7.1
5	M04PD	68;9	73;5	3.5	Stalevo, Permax	Sinemet, Sinemet CR, Requip, Flomax	WNL	37.5	82.0
	M07OC	70;6	74;0	n/a	None	Eye drops for Glaucoma	WNL	3.5	7.5
6	M09PD	72;8	76;8	9.0	Sinemet, Lipitor, Prozac, Metoprolol	Sinemet, Aricept, Lipitor, Metoprolol, Tylenol, Ibuprofen	Moderate	35.5	73.0
	M11OC	73;5	77;3	n/a	Aspirin	None	WNL	0.3	3.0

Note. F = female, M = male; PD = Parkinson disease, OC = control participant; CLQT = composite score of the Cognitive-Linguistic Quick Test (Helm-Estabrooks, 2001); WNL = within normal limits; ^ diagnosed with PD three years after Wave 1; higher numbers indicate more severe speech ratings for speech severity.

Table 1. Participant demographic information, continued

Pair	Participant	Age at Wave 1 (years; months)	Age at Wave 2 (years; months)	Years Since Diagnosis (Wave 1)	Medications at Wave 1	Medications at Wave 2	CLQT at Wave 2	Speech Impairment at Wave 1 (%)	Speech Impairment at Wave 2 (%)
7	M10PD	70;0	73;7	4.5	Sinemet	Sinemet, Aricept, Mirtazapine, Donepezil	Mild	3.0	8.9
	M06OC	70;6	74;1	n/a	None	None	WNL	0.6	2.0
8	M11PD	82;0	85;2	3.75	Amantadine, Sinemet, Carvedilol, Flomax	Amantadine, Sinemet, Lodosyn, Carvedilol, Provigil, Clonazepam, Fosamax, Flomax	WNL	43.0	35.5
	M09OC	82;0	85;6	n/a	Lipitor	Lipitor	WNL	7.5	0.3

Note. F = female, M = male; PD = Parkinson disease, OC = control participant; CLQT = composite score of the Cognitive-Linguistic Quick Test (Helm-Estabrooks, 2001); WNL = within normal limits; ^ diagnosed with PD three years after Wave 1; higher numbers indicate more severe speech ratings for speech severity

Table 2: Statistical summary for main and interaction effects

	Group (df = 1)		Time (df = 1)		Group X Time (df = 1)	
Measure	***F***	***p***	***F***	***p***	***F***	***p***
Sound pressure level (dB)	0.02	0.896	17.15	<0.0001*	11.85	0.0006*
Utterance length (syllables)	1.21	0.272	4.83	0.03*	1.15	0.284
Speech rate (syllables/second)	3.06	0.081	4.29	0.039*	20.18	<0.0001*
Lung volume initiation (%VC)	0.04	0.846	54.43	<0.0001*	17.97	<0.0001*
Lung volume termination (%VC)	0.18	0.672	59.55	<0.0001*	1.02	0.312
Lung volume excursion (%VC)	0.31	0.58	0.10	0.753	21.21	<0.0001*
%VC per syllable	0.05	0.823	1.62	0.204	16.31	<0.0001*

Note. df = degrees of freedom; dB = decibels; %VC = percent vital capacity; *$p < .05$

Table 3: Pairwise comparisons for significant group by time interaction effects

Measure	Contrast	*p*
Sound pressure level (dB)	CP – Time 1 vs. CP – Time 2	<.0001*
	PD – Time 1 vs. PD – Time 2	.96
	CP – Time 1 vs. PD – Time 1	.74
	CP – Time 2 vs. PD – Time 2	.87
Speech rate (syllables/second)	CP – Time 1 vs. CP – Time 2	.30
	PD – Time 1 vs. PD – Time 2	<.0001*
	CP – Time 1 vs. PD – Time 1	.97
	CP – Time 2 vs. PD – Time 2	.02*
Lung volume initiation (%VC)	CP – Time 1 vs. CP – Time 2	.11
	PD – Time 1 vs. PD – Time 2	<.0001*
	CP – Time 1 vs. PD – Time 1	.82
	CP – Time 2 vs. PD – Time 2	.96
Lung volume excursion (%VC)	CP – Time 1 vs. CP – Time 2	.01*
	PD – Time 1 vs. PD – Time 2	.004*
	CP – Time 1 vs. PD – Time 1	.84
	CP – Time 2 vs. PD – Time 2	.23
%VC per syllable	CP – Time 1 vs. CP – Time 2	.0008*
	PD – Time 1 vs. PD – Time 2	.22
	CP – Time 1 vs. PD – Time 1	.62
	CP – Time 2 vs. PD – Time 2	.86

Note. dB = decibels; CP = control participants; PD = Parkinson disease; %VC = percent vital capacity; *$p < .05$

Table 4: Means and standard deviations (in parentheses) for each group and time

Measure	Time 1	Time 2
Sound pressure level (dB)		
PD	76.30 (4.04)	76.46 (5.53)
Control Participants	75.59 (3.08)	77.41 (4.28)
Utterance length (syllables)		
PD	14.04 (8.96)	12.33 (6.89)
Control Participants	14.46 (8.70)	13.90 (7.97)
Speech rate (syllables/second)		
PD	4.20 (1.09)	4.68 (1.27)
Control Participants	4.05 (0.93)	3.88 (0.93)
Lung volume initiation (%VC)		
PD	40.34 (16.98)	25.53 (17.45)
Control Participants	36.01 (28.03)	31.60 (24.30)
Lung volume termination (%VC)		
PD	18.69 (19.94)	8.44 (17.91)
Control Participants	16.18 (25.89)	8.61 (18.42)
Lung volume excursion (%VC)		
PD	21.65 (13.37)	17.09 (10.54)
Control Participants	19.83 (13.15)	22.99 (15.35)
%VC per syllable		
PD	1.74 (1.74)	1.52 (1.52)
Control Participants	1.51 (1.51)	1.83 (1.83)

Note. Positive values for lung volume initiation, termination, and excursion indicate lung volumes above end expiratory level; PD = Parkinson disease; dB = decibels; VC = vital capacity

Table 5: Individual effect sizes

Participant	SPL (dB)	Utterance Length	Speech Rate	LVI (%VC)	LVT (%VC)	LVE (%VC)	%VC per Syllable
F01PD	0.33^{S}	-0.52^{M}	1.72^{VL}	-0.83^{L}	-0.16^{S}	-0.51^{M}	0.14^{VS}
F02PD	-0.16^{VS}	-0.01^{VS}	0.84^{L}	0.56^{M}	1.16^{VL}	-0.56^{M}	-1.00^{L}
F04PD	0.62^{M}	-0.24^{S}	-0.23^{S}	-1.66^{VL}	-1.60^{VL}	-0.17^{S}	0.36^{S}
F07PD	0.65^{M}	-0.16^{S}	0.70^{M}	-0.45^{M}	0.26^{S}	-0.63^{M}	-0.68^{M}
M04PD*	-1.24^{VL}	-0.09^{VS}	0.11^{VS}	-4.17^{H}	-3.55^{H}	-0.81^{L}	-1.30^{VL}
M09PD	-0.38^{S}	-0.31^{S}	0.30^{S}	-0.50^{M}	0.18^{S}	-0.85^{L}	-1.00^{L}
M10PD	0.76^{L}	0.01^{VS}	0.77^{M}	-0.17^{S}	-0.84^{L}	0.77^{L}	0.91^{L}
M11PD	-0.32^{S}	-0.41^{S}	-0.07^{VS}	-1.97^{H}	-1.29^{VL}	-0.44^{S}	-0.37^{S}
F02OC	2.02^{H}	-0.27^{S}	0.59^{M}	-0.50^{M}	0.13^{VS}	-0.58^{M}	-0.78^{L}
F05OC	-0.64^{M}	-0.01^{VS}	0.06^{VS}	0.29^{S}	-0.03^{VS}	0.27^{S}	0.27^{S}
F07OC	0.13^{VS}	-0.19^{S}	-1.22^{VL}	1.50^{VL}	0.86^{L}	0.23^{S}	0.86^{L}

Note. Negative value means that the outcome measure decreased from Time 1 to Time 2; Positive values means that the outcome measure increased from Time 1 to Time 2; * indicates the participant underwent deep brain stimulation surgery between Time 1 and Time 2. Effect size strength based on Sawilowsky (2009): VS = very small, S = small; M = medium; L = large; VL = very large; and H = huge.

Table 5: Individual effect sizes

Participant	SPL (dB)	Utterance Length	Speech Rate	LVI (%VC)	LVT (%VC)	LVE (%VC)	%VC per Syllable
F13OC	2.76^{H}	-0.28^{S}	-0.39^{S}	-0.85^{L}	-0.83^{L}	-0.05^{VS}	0.51^{M}
M06OC	-0.56^{M}	-0.01^{VS}	-0.82^{L}	0.37^{S}	-0.47^{M}	0.83^{L}	1.43^{VL}
M07OC	-0.31^{S}	0.81^{L}	0.23^{S}	2.60^{H}	1.50^{VL}	1.24^{VL}	0.59^{M}
M09OC	2.14^{H}	0.10^{VS}	-0.12^{VS}	-0.34^{S}	-0.45^{M}	0.20^{S}	-0.22^{S}
M11OC	2.57^{H}	-0.75^{L}	0.04^{VS}	-3.20^{H}	-2.81^{H}	-0.18^{S}	0.31^{S}

Note. Negative value means that the outcome measure decreased from Time 1 to Time 2; Positive values means that the outcome measure increased from Time 1 to Time 2; * indicates the participant underwent deep brain stimulation surgery between Time 1 and Time 2. Effect size strength based on Sawilowsky (2009): VS = very small, S = small; M = medium; L = large; VL = very large; and H = huge.

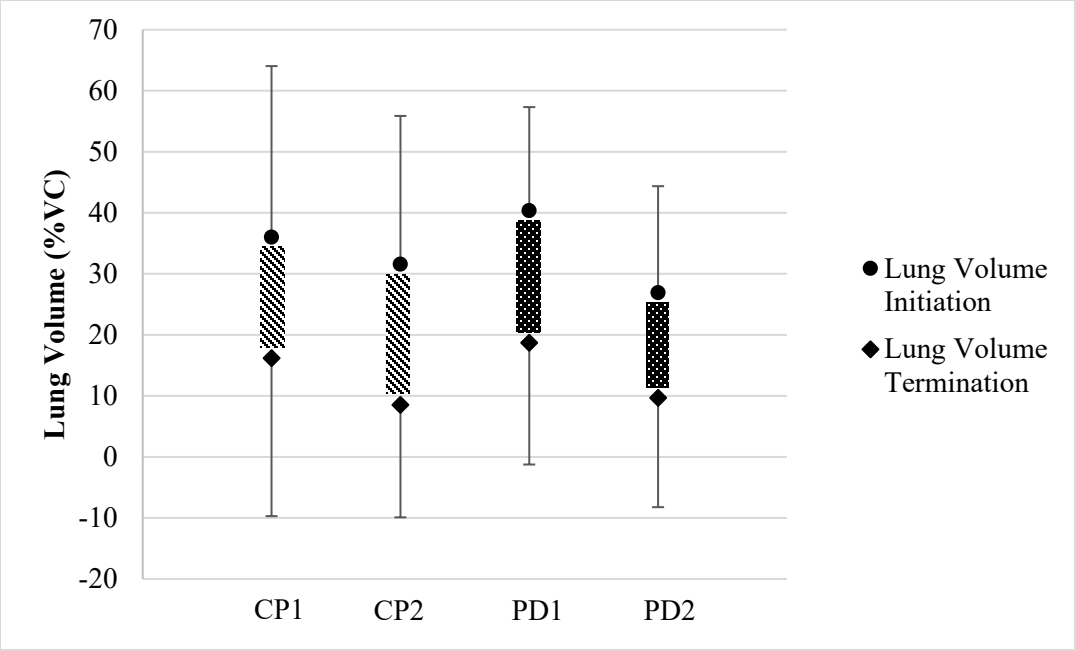

Figure 1: Lung volume for group and time. Bars represent excursions. Lines represent standard deviations of initiations and terminations. 0%VC represents end expiratory level. CP1 and CP2, control participants at each time point; PD1 and PD2, individuals with Parkinson disease at each time point.

Appendix 1. Participant Vital Capacity Data (in liters)

Participant	VC at Time 1	VC at Time 2	FVC at Time 1	FVC at Time 2	$FEV_{1.0}$ at Time 1	$FEV_{1.0}$ at Time 2
F01PD	2.43 WNL	2.18 WNL	2.40 WNL	2.37 WNL	2.07 WNL	2.02 WNL
F02PD	2.40 WNL	2.15 Low	2.29 WNL	2.18 Low	1.99 WNL	1.77 WNL
F04PD	2.54 WNL	2.29 WNL	2.24 WNL	2.18 WNL	1.99 WNL	1.77 WNL
F07PD	Unable to test*	2.35 WNL	2.59 WNL	2.32 WNL	2.18 WNL	1.68 Low
F02OC	2.87 WNL	2.40 WNL	2.87 WNL	2.68 WNL	2.57 WNL	2.40 WNL
F05OC	2.54 WNL	2.40 WNL	2.51 WNL	2.37 WNL	2.18 WNL	1.77 WNL
F07OC	2.68 WNL	2.37 WNL	2.65 WNL	2.51 WNL	2.26 WNL	2.07 WNL
F13OC	2.79 WNL	2.24 Low	2.65 WNL	2.59 WNL	2.1 WNL	2.02 WNL
M04PD	5.08 WNL	4.72 WNL	4.64 WNL	4.67 WNL	3.67 WNL	3.48 WNL
M09PD	2.59 WNL	2.93 WNL	2.93 WNL	2.68 WNL	2.46 WNL	2.35 WNL
M10PD	2.98 Low	2.54 Low	2.24 Low	2.98 WNL	1.82 Low	2.21 WNL
M11PD	3.31 WNL	2.98 WNL	3.12 WNL	2.93 WNL	2.21 WNL	2.10 WNL
M06OC	4.14 WNL	3.98 WNL	4.14 WNL	4.00 WNL	3.23 WNL	2.98 WNL
M07OC	3.78 WNL	3.45 WNL	3.75 WNL	3.37 WNL	3.01 WNL	2.65 WNL
M09OC	4.61 WNL	4.53 WNL	4.69 WNL	4.39 WNL	3.48 WNL	3.37 WNL
M11OC	3.28 WNL	3.01 WNL	3.17 Low	2.95 WNL	2.18 Low	2.18 Low

Note. F = female; M = male; PD = person with Parkinson disease; OC = control participants; VC = slow vital capacity in liters; FVC = forced vital capacity in liters; FEV1.0 = forced expiratory volume in 1 second in liters; WNL = within normal limits for age, sex, weight, height, and ethnicity; *participant could not complete the slow vital capacity within the time limit for the spirometer

Appendix 2: Inter-Class Correlations

Dependent Variable	**Mean Difference between Measurers**	**ICC**	**Level of Agreement**
Utterance Length	.73 syllables	.611	Moderate
Speech Rate	.18 syllables per second	.766	Good
Lung Volume Initiation	.08 %VC relative to EEL	.906	Excellent
Lung Volume Termination	.44 %VC relative to EEL	.683	Moderate
Lung Volume Excursion	.51 %VC	.622	Moderate

Note: %VC = percent vital capacity, EEL = end expiratory level, level of agreement based on Koo and Li (2016)