

Research Article

Longitudinal Effects of Parkinson's Disease on Speech Breathing During an Extemporaneous Connected Speech Task

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ABSTRACT

Purpose: A critical component to the development of any type of intervention to improve speech production in individuals with Parkinson's disease (PD) 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 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 over the course of approximately 3.5 years.

Method: Eight individuals with PD and eight 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 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 PD 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.

Speech impairment is a hallmark characteristic of Parkinson's 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 physiological changes, individuals with PD rely more on active respiratory muscle forces than passive recoil forces to produce speech, which increases the work

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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, 2020). 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 be 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 & Darling, 2011; Huber et al., 2012; 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, 2020; 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, 2020; 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 with control participants (Huber & Darling-White, 2017). Individuals

with PD also initiated and terminated speech at lower lung volumes as compared with 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, 2020).

Although 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 and 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). Furthermore, 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 health care providers can use to classify disease progression in the speech production system.

Method

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). Data from the extemporaneous speech task from Time 2 have not been published. Data from a reading task produced by these participants at both Time 1 and Time 2 have been published previously (Darling-White & Huber, 2020; Huber & Darling-White, 2017).

Participants

Participants included eight individuals (four men and four 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 pathophysiological 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 the studies of Huber and Darling-White (2017), Darling-White and Huber (2020), and this 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 hr 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 40 dB HL for 500, 1000, and 2000 Hz (Ventry & Weinstein, 1983), except for M04PD, who did not pass at 40 dB 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 (TIA) 1 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 40 dB HL for 1000 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

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
3	F07OC	65;8	69;7	n/a	None	Estrace	WNL	1.0	1.0
	F07PD	72;2	75;11	-3.0 ^a	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
4	F04PD	74;3	76;11	5.0	Sinemet, Eldepryl, Bromocriptine	Sinemet, Bromocriptine, Zelapar	WNL	2.8	11.4
5	F05OC	73;1	77;1	n/a	Lipitor	Lipitor, Diazepam	WNL	3.5	7.1
	M04PD	68;9	73;5	3.5	Stalevo, Permax	Sinemet, Sinemet CR, Requip, Flomax	WNL	37.5	82.0
6	M07OC	70;6	74;0	n/a	None	Eye drops for Glaucoma	WNL	3.5	7.5
	M09PD	72;8	76;8	9.0	Sinemet, Lipitor, Prozac, Metoprolol	Sinemet, Aricept, Lipitor, Metoprolol, Tylenol, Ibuprofen	Moderate	35.5	73.0
7	M11OC	73;5	77;3	n/a	Aspirin	None	WNL	0.3	3.0
	M10PD	70;0	73;7	4.5	Sinemet	Sinemet, Aricept, Mirtazapine, Donepezil	Mild	3.0	8.9
8	M06OC	70;6	74;1	n/a	None	None	WNL	0.6	2.0
	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. CLQT = composite score of the Cognitive Linguistic Quick Test (Helm-Estabrooks, 2001); F = female; PD = Parkinson's disease; WNL = within normal limits; M = male; OC = control participant; n/a = not applicable.

^aDiagnosed with PD 3 years after Wave 1; higher numbers indicate more severe speech ratings for speech severity.

certified speech-language pathologists (SLPs) 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 1 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. Percentage 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 SLPs 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 then 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). 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 speechlike breathing with Respitrace bands in place and while breathing into a digital spirometer (VacuMed Universal Ventilation Meter). For the speechlike breathing task, participants read a short sentence silently to themselves 1 time per breath. The purpose of the speechlike 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 (k_1 in Formula 1 below) for the rib cage was computed using the following formula from data from the rest breathing and speechlike breathing tasks. The correction factor for the abdomen was set to 1.

$$\text{Lung Volume (from spirometer)} = \text{RibCage} * k_1 + \text{Abdomen} \quad (1)$$

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.

$$\text{Estimated Lung Volume} = \text{RibCage} * k_1 + \text{Abdomen} \quad (2)$$

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

Participants completed VC tasks both with and without the respiratory bands in place. Prior to band placement, participants completed a slow VC and a forced VC maneuver using a VacuMed Discovery Handheld Spirometer. In the slow VC task, participants were cued to inspire to the top of their lung volume and then expire as much as possible. In the forced VC task, they were cued to inspire to the top of their lung volume and then expire as quickly and forcefully as possible. The slow VC task was completed first followed by the forced VC task. The purpose of these tasks was to measure lung function. VC, forced VC, and forced expiratory volume in 1 s are reported in Appendix A. With the bands in place, three more slow VC maneuvers were completed. Slow VC 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, and excursion were expressed as a percentage of VC.

Speech Task

Using their comfortable pitch and loudness, participants spoke about a topic of their choice for approximately 2 min. Participants were asked to complete several different speech tasks during each data collection session. The extemporaneous speech task described in this 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 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 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 if below 0.2, small if from 0.2 to 0.5, medium if from 0.5 to 0.8, large if from 0.8 to 1.2, very large if from 1.2 to 2.0, and huge if 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 0.2.

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 (ICCs) were used to assess intermeasurer reliability. ICCs 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 intermeasurer reliability (see Appendix B; 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 \times Time ($F = 11.85$, $p = .001$). There was no significant effect of group. There was a significant increase in SPL

Table 2. Statistical summary for main and interaction effects

Measure	Group (<i>df</i> = 1)		Time (<i>df</i> = 1)		Group × Time (<i>df</i> = 1)	
	<i>F</i>	<i>p</i>	<i>F</i>	<i>p</i>	<i>F</i>	<i>p</i>
Sound pressure level (dB)	0.02	.896	17.15	< .0001*	11.85	.0006*
Utterance length (syllables)	1.21	.272	4.83	.03*	1.15	.284
Speech rate (syllables/s)	3.06	.081	4.29	.039*	20.18	< .0001*
Lung volume initiation (%VC)	0.04	.846	54.43	< .0001*	17.97	< .0001*
Lung volume termination (%VC)	0.18	.672	59.55	< .0001*	1.02	.312
Lung volume excursion (%VC)	0.31	.58	0.10	.753	21.21	< .0001*
%VC per syllable	0.05	.823	1.62	.204	16.31	< .0001*

Note. *df* = degrees of freedom; %VC = percent vital capacity.

**p* < .05.

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 control participants (*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 DBS implantation surgery between Time 1 and Time 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 × 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

Table 3. Pairwise comparisons for significant Group × Time interaction effects.

Measure	Contrast	<i>p</i>
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/s)	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. CP = control participants; PD = Parkinson's 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/s)		
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 (0.95)	1.52 (0.81)
Control participants	1.51 (1.39)	1.83 (0.93)

Note. Positive values for lung volume initiation, termination, and excursion indicate lung volumes above end expiratory level. PD = Parkinson's disease; %VC = percent 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 ^a	-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
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	0.23 ^S	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. Effect size strength was based on Sawilowsky (2009): VS = very small; S = small; M = medium; L = large; VL = very large; and H = huge. SPL = sound pressure level; LVI = lung volume initiation; LVT = lung volume termination; LVE = lung volume excursion; %VC = percent vital capacity; F = female; PD = Parkinson's disease; M = male; OC = control participant.

^aThe participant underwent deep brain stimulation surgery between Time 1 and Time 2.

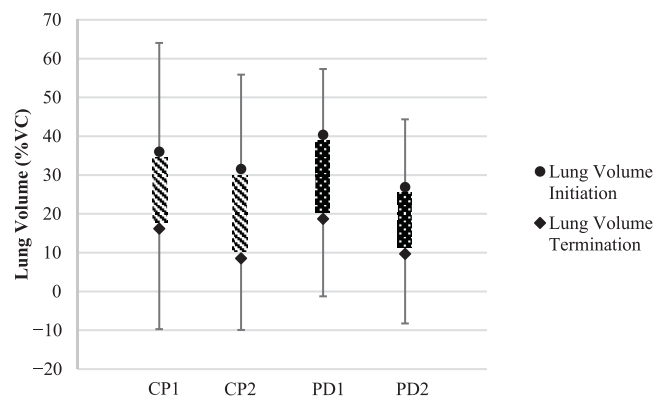
Group \times Time ($F = 20.18$, $p < .001$). There was no significant effect of group. There was a significant increase in speech rate from Time 1 to Time 2. This effect was primarily driven by the individuals with PD. Speech rate was significantly faster for individuals with PD at Time 2 as compared with 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 two of the eight 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 \times 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 with 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

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's disease at each time point. %VC = percent vital capacity.



(F02PD) increased lung volume initiation. There was a mix of changes in the control participants with three increasing lung volume initiation and three decreasing lung volume initiation.

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 \times Time. Both individuals with PD and control participants terminated speech at significantly lower lung volumes at Time 2 as compared with 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 lung volume termination. Four of the eight control participants decreased lung volume termination mostly with medium to large effect sizes; two control participants increased lung volume termination.

Lung Volume Excursion

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

%VC per Syllable

There were no significant main effects of group or time, but there was a significant interaction effect of Group \times Time ($F = 16.31, p < .001$). Control participants expended more lung volume per syllable at Time 2 as compared with 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 per 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. Although 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: (a) changes to respiratory physiology, like increased chest wall rigidity, and (b) 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 normal limits (see Appendix A), 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's study, 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 support

this interpretation since all participants who 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 per 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 to the disease process highlight the need for SLPs 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 that 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, and participation in rehabilitation services). Small, fairly heterogeneous cohorts are to be expected in this type of work. Although 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 in 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 physiological 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.

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Appendix A

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 ^a	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. VC = slow vital capacity in liters; FVC = forced vital capacity in liters; FEV_{1.0} = forced expiratory volume in 1 second in liters; F = female; PD = person with Parkinson's disease; WNL = within normal limits for age, sex, weight, height, and ethnicity; OC = control participants; M = male.

^aParticipant could not complete the slow vital capacity within the time limit for the spirometer.

Appendix B

Interclass Correlations

Dependent variable	Mean difference between measurers	ICC	Level of agreement
Utterance length	0.73 syllables	.611	Moderate
Speech rate	0.18 syllables/s	.766	Good
Lung volume initiation	0.08 %VC relative to EEL	.906	Excellent
Lung volume termination	0.44 %VC relative to EEL	.683	Moderate
Lung volume excursion	0.51 %VC	.622	Moderate

Note. ICC = interclass correlation coefficient; %VC = percent vital capacity; EEL = end expiratory level, level of agreement based on Koo and Li (2016).