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Longitudinal Changes in Speech Breathing in Older Adults with and without Parkinson's Disease

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Abstract

This longitudinal study examines changes to speech production and speech breathing in older adults with and without Parkinson's disease (PD). Eight participants with PD and 8 age- and sex-matched older adults participated in two data collection sessions, separated by 3.7 years on average. Speech severity and speech rate increased for people with PD. Vital capacity decreased for both groups. Older adult control participants displayed significant increases in lung volume initiation and excursion and percent vital capacity expended per syllable. These changes allow older adults to utilize higher recoil pressures to generate subglottal pressure for speech production, potentially reducing work of breathing. Participants with PD displayed significant decreases in lung volume initiation and termination. Thus, unlike older adults, people with PD exert more expiratory muscle pressure during speech production, leading to increased effort. Speech-language pathologists need to consider direct treatment of respiratory patterns for speech to reduce effort and fatigue.

Keywords

Parkinson's disease; Aging; Respiratory function

INTRODUCTION

While there have been a large number of investigations of speech production in people with Parkinson's disease (PD), most have focused on articulatory and voice. Less is known about the changes to speech breathing in PD. And even less attention has been paid to understanding the effects of disease progression on speech production and speech breathing.

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FINANCIAL DISCLOSURE

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People with PD demonstrate consistent respiratory physiologic differences as a result of their disease, including smaller vital capacity (VC)[1–3] and weaker inspiratory and expiratory muscles[2,4,5] than age- and sex-matched older adults. However, findings regarding patterns of speech breathing in PD have been relatively varied. Studies have found a large number of changes to speech breathing, including shorter utterances, both higher and lower lung volume initiations (LVI) and terminations (LVT), and higher variability of respiratory movements[6–10]. In general, these findings suggest that, for individuals with PD, speech breathing patterns reflect a stiffer or more rigid chest wall, weaker respiratory muscles, and/or reduced coordination between speech production and breathing. Unfortunately, due to the variability of findings related to speech breathing, it is difficult to develop an understanding of the importance of direct treatment of the respiratory system to improve speech production and to identify potential respiratory treatment targets. It may be that disease progression, or where a participant is in their disease progression, is a significant factor in the variability across studies[10]. There is no gold-standard method for obtaining the progression of changes to the respiratory muscles as a result of PD and assuming that axial muscles change at the same rate or in the same manner as appendicular muscles may not be valid. Longitudinal studies of speech production changes associated with Parkinson's disease provide the information necessary to fully understand the effects of disease progression and to develop effective interventions for each stage of disease progression.

Despite the value of longitudinal data, there have been very few longitudinal studies of speech production changes associated with PD and none of speech breathing. One study examined changes to speech production, motor function, cognition, and mood over two data collection points, separated by approximately 3 years, and found significant increase in speech severity and declines in speech intelligibility, motor function, and cognition[11]. In a second study, dysprosody associated with PD and motor function were examined at two time points[12]. The range of time between the two data collection points was wide (~.5 years to 6.5 years). Speech rate decreased in men with PD and pitch variability declined in women with PD, but motor function was stable[12]. The paucity of longitudinal data relative to speech production changes in PD, and the complete lack of longitudinal data on speech breathing in PD, are significant gaps in the literature. A better understanding of disease progression relative to speech production and speech breathing would advance treatment for dysarthria in PD significantly.

Since PD is a disease that is more common in older adults[13], it is also important to consider the changes that would be expected as a result of typical aging when examining disease progression. Reductions in VC with typical aging are well-established in the literature[14–17]. Lung elasticity and chest wall compliance decrease with typical aging, reducing the passive pressures that can be generated in the respiratory system[18,19]. As compared to young adults, older adults use higher LVI, LVT, lung volume excursion (LVE), and percent VC expended per syllable (%VC/syl)[20–23]. Recoil pressure is higher at higher lung volumes, even in older adults[24,25]. Therefore, beginning speech at a higher lung volume allows older adults to use that higher recoil pressure to generate subglottal pressure for speech, potentially reducing work of breathing. Increased LVE is often thought to be related to decreased vocal fold closure resulting in a greater %VC/syl[21,23]. While a number of studies have examined

changes to speech breathing in aged populations, no longitudinal studies of speech breathing are available in the literature.

The purpose of this longitudinal study was to examines changes to speech production and speech breathing in older adults with and without PD over two data collection sessions, separated by 3.7 years on average (hereafter called Wave 1 and Wave 2). Global speech outcomes (speech severity, sound pressure level (SPL), utterance length, and speech rate) were assessed along with speech breathing outcomes (VC, LVI, LVT, LVE, and %VC/syl). We predict that typically aging older adults will increase LVI, LVT, LVE, and %VC/syl and decrease SPL, utterance length, and VC from Wave 1 to 2. However, people with PD will increase speech severity and speech rate and decrease SPL, utterance length, VC, LVI, LVT, LVE, and %VC/syl from Wave 1 to 2 As a result of disease progression, we expect that differences between the two groups will be larger in Wave 2 than in Wave 1.

METHODS

This longitudinal study consisted of two data collection sessions (Wave 1 and Wave 2). Wave 1 was part of a larger study designed to examine the impact of different types of cues to increase loudness on speech production patterns. The participants presented in this paper are a small subset of the total number of participants who took part in the original study. Only those participants from Wave 1 for whom both the individual with PD and the age- and sex-matched older adult were able to participate in Wave 2 were included in this study. The average time between waves was 3 years; 7 months (SE = 1 year; 8 months). The data from the Wave 1 participants have been included in previously published datasets[8,10,20]. The data from Wave 2 have not been previously published.

Participants

Eight individuals diagnosed with idiopathic PD by a neurologist and 8 age- and sex-matched older adults participated in this study. There were 4 males and 4 females in each group. Table 1 provides the demographic participant data. Participants with PD were free of any other neurological issues. Typical older adults had no neurologic diagnoses or speech impairments. Participants were closely age- and sex-matched. A t-test on the ages of the participants at each wave showed no significant differences in age across the groups (Wave 1: t=−.06, p=955; Wave 2: t=.16, p=.879).

Longitudinal research poses many challenges. For example, F07PD was originally seen in Wave 1 as a control participant. However, her data from Wave 1 were excluded due to significant differences from the rest of the female control group. In the years between Waves 1 and 2, F07PD was diagnosed with PD. Another challenge is the fact that treatment changes occur between sessions 1 and 2 for some participants with PD. One participant (M04PD) had undergone deep-brain stimulation surgery, and this surgery occurred between Waves 1 and 2. One participant received speech therapy for PD (M09PD), weekly group therapy for two years, before for Wave 1. No other participants reported engaging in speech therapy within 20 years of Wave 1. Between Waves 1 and 2, F01PD reported therapy to improve speech intensity and rate, F02PD and M04PD reported speech therapy to increase loudness, M09PD reported therapy to improve speech clarity, and M10PD reported therapy to improve

word finding. None of the older control participants reported speech or language therapy between Waves 1 and 2.

All participants were free from colds, infections, and allergy symptoms on test days. All of the participants were non-smokers for at least five years prior to Wave 1, except M10PD who stopped smoking 1 year prior to Wave 1. Non-smoking status was maintained through Wave 2 for all participants. All participants passed the Mini-Mental State Exam[26] in Wave 1. The Cognitive-Linguistic Quick Test[27] was given in Wave 2 to index cognition, but participants were not required to obtain a score within normal limits to continue in the study (see Table 1). All participants were living in the community and ambulatory, although some required assistance.

All participants passed a hearing screening at 40 dB at .5, 1, and 2 kHz in Wave 1 and Wave 2 except M04PD at both waves (higher than 40 dB at 2 kHz in right ear) and M07OC at Wave 2 (higher at all frequencies in right ear). Vital capacity, forced vital capacity, and forced expiratory volume in one second were tested in both waves (VacuMed Discovery Handheld Spirometer).

Two speech-language pathologists, with experience treating patients with motor speech disorders and uninvolved in the study, rated speech severity for all participants. Speech severity was rated on a Visual Analog Scale (VAS) with one end marked “normal” and one end marked “very severe.” Ratings were made after listening to a 30 second sample of a monologue. Samples were taken from the approximate middle of the monologue, without starting or ending during a sentence. The speech samples were intensity normalized and presented over headphones. The raters listened to each sample one time. Samples were blocked by speaker sex so that all the female samples were presented first and then all the male samples. Samples were randomized such that no two samples from the same participant were presented consecutively. Ratings were completed without knowledge of the speaker’s group or wave.

The distance from “normal” to the rating mark on the VAS was measured in mm and converted to a percent by dividing by the length of the line in mm. The higher numbers indicated more severe the speech impairment. For most of the samples, the speech severity rating was computed by averaging the ratings of the two speech-language pathologists. When the difference between the two raters was greater than 20%, a third speech-language pathologist (the first author) rated the speech sample. This occurred in 7 instances. For these samples, the third rating was less than 20% from one of the two original ratings, and the speech severity rating was computed by averaging the third rating and the rating which was closest to the third rating. Speech severity ratings are presented in Table 1.

Equipment

High quality condenser microphones, with flat frequency response from 50 to 18,000 Hz, were used to transduce the acoustic signal. The acoustic signal was recorded to digital audiotape. The acoustic signal was digitized at 44.1 kHz and then resampled at 18 kHz and low-pass filtered at 9 kHz in Praat[28]. A sound level meter provided amplification to the

acoustic signal, and gain was factored in for calibration. The mouth-to-microphone distance was 6 in.

The respiratory kinematic data were recorded by using respiratory inductive plethysmography (Respitrace system, Ambulatory Monitoring). Two elastic bands were placed on the participants, one around the rib cage (RC) under the axilla to transduce RC movement and the other around the abdomen (AB) below the last rib and approximately at the level of the navel to transduce AB movement. Sound from the room was transduced by a second microphone and was digitized in synchrony with the Respitrace signals.

Procedures and Stimuli

Speech Task: Participants produced two trials of the Papa Passage[29] at comfortable loudness and pitch per session. While data from a variety of speech tasks were collected at each session, the reading passage was always collected early in the data collection session, shortly after the respiratory calibration tasks and the maximum capacity tasks.

Respiratory Calibration for Lung Volume: Calibration of the respiratory kinematic signals for lung volume estimation was completed using a variant of the least squares method and has been described in detail previously[10,30,31]. Participants also produced three trials of the vital capacity maneuver while wearing the Respitrace bands.

Measurements

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

Sound pressure level (SPL): Average SPL was measured for each breath group in Praat[28].

Utterance length: The number of syllables produced during each breath was counted.

Speech Rate: Computed by dividing the utterance length by the duration of each breath group.

Lung Volume: All of the lung volume measurements were made as a percent of VC. Lung volume initiation (LVI) was defined as the lung volume at which participants began speaking and was measured at the onset of the acoustic signal for each breath group. Lung volume termination (LVT) was defined as the lung volume at which participants stopped speaking and was measured at the offset of the acoustic signal for each breath group. LVI and LVT were measured relative to end expiratory level which was determined from at least three rest breaths prior to the start of the trial. Thus positive numbers are above end expiratory level and negative numbers are below end expiratory level. Lung volume excursion (LVE) was defined as the lung volume expended during the breath group and was measured by subtracting LVT from LVI. The percent VC per syllable (%VC/syl) was measured by dividing the LVE by the utterance length.

Statistical Analysis

A mixed model, repeated measures Analysis of Variance was computed for each dependent variable. Group (PD or OC) was a between-subject factor. Wave (1 and 2) was a within-subject factor. Participant was included as a random factor. Tukey honestly significantly different (HSD) tests were computed when the interaction between group and wave was significant. The alpha level was set to $p \leq 0.01$ for all tests.

Reliability

One male and one female participant in each wave were randomly chosen and remeasured by a second measurer. A t-test was used to determine if the two measurements were significantly different. No significant differences were present for any of the dependent variables indicating adequate inter-measurer reliability.

RESULTS

See Table 2 for means and standard errors for all dependent variables for each group and wave.

Speech Severity:

Group (F=11.42, p=.005), wave (F=9.27, p=.009), and the interaction between group and Wave (F=8.06, p=.013) were all significant. Speech severity increased from Wave 1 to Wave 2 for participants with PD (p=.005) but not for the typical older adults (p=.999). Participants with PD had higher speech severity scores than typical older adults in Wave 2 (p=.004) but not in Wave 1 (p=.295).

SPL:

Wave (F=155.0,3 p<.001) and the interaction between group and Wave (F=15.96, p<.001) were significant, but group was not significant (F=.20, p=.654). SPL significantly increased from Wave 1 to Wave 2 for both groups (p<.001).

Utterance length:

There were no significant main effects of Wave (F=.18, p=.676) or group (F=.18, p=.674) and no significant interaction effects (F=.85, p=.175).

Speech rate:

Wave (F=17.25, p<.001) and the interaction between group and Wave (F=16.30, p<.001) were significant, but group was not significant (F=1.19, p=.275). Speech rate significantly increased from Wave 1 to Wave 2 for participants with PD (p<.001) but there were no significant changes for typical older adults (p=.999).

VC:

Wave (F=25.12, p<.001) was significant, but group (F=.56, p=.467) and the interaction between group and Wave (F=.42, p=.527) were not significant. VC significantly decreased from Wave 1 to Wave 2 for both groups.

LVI:

There were no significant main effects of group (F=.04, p=.836) or wave (F=1.08, p=.300), but there was a significant interaction between group and Wave (F=85.88, p=<.001). LVI increased significantly from Wave 1 to Wave 2 for typical older adults (p<.001). However, for the participants with PD, LVI decreased significantly from Wave 1 to Wave 2 (p<.001).

LVT:

Wave (F=12.53, p<.001) and the interaction between group and Wave (F=19.57, p<.001) were significant, but group was not significant (F=.29, p=.587). LVT significantly decreased from Wave 1 to Wave 2 for participants with PD (p<.001), but there were no significant changes for typical older adults (p=.924).

LVE:

Wave (F=9.42, p=.002) and the interaction between group and Wave (F=42.59, p<.001) were significant, but group was not significant (F=.49, p=.485). LVE increased from Wave 1 to Wave 2 for typical older adults (p<.001), but there were no significant changes for those with PD (p=.070).

%VC/syl:

Wave (F=27.61, p<.001) and the interaction between group and Wave (F=42.59, p<.001) were significant, but group was not significant (F=.49, p=.485). %VC/syl significantly increased from Wave 1 to Wave 2 typical older adults (p<.001), but there were no significant changes for those with PD (p=.079).

DISCUSSION

The purpose of this longitudinal study was to examine changes to speech production and speech breathing in people with PD and age- and sex-matched older adults over two data collection sessions. Global speech production measurements changed for the participants with PD, but not for the typical older adults. Participants with PD experienced a significant worsening of their speech severity ratings, as would be expected given the progressive nature of PD. Speech rate also significantly increased for the participants with PD. Increased rate is a common perceptual characteristic in dysarthria associated with PD[32]. Our data suggest that faster speech rate is more likely to occur as PD progresses.

Interestingly, even though speech rate increased for the participants with PD, utterance length (the number of syllables said on one breath) did not change. Thus, increased speech rate in the participants with PD did not result in more being said on one breath. Utterance length in typical older adults did not significantly change either. Several published studies have reported that utterance length is shorter in typical older adults, as compared to young adults[20–22], but the findings are more mixed for the comparison of people with PD and age- and sex-matched individuals with some, but not all, finding shorter utterances in people with PD[6,9,10,33]. The current study suggests that while utterance length may change across large swaths of time (e.g., from 20-year olds to 70 year olds), utterance length does not change over a relatively short amount of time for older adults with and without PD.

There were small but significant increases in SPL across the two waves for both groups. While speech breathing is altered when a speaker changes SPL, all of the samples were collected at the participant's self-chosen comfortable loudness level. The increase from Wave 1 to Wave 2 was small enough (~1.5 dB) that it would be unlikely to drive the changes in speech breathing.

Turning to the changes in respiration that occurred across the waves, both groups demonstrated a significant decrease in VC across time. These results fit with previous data for the most part, except there was no significant difference between those with PD and those without[2,3,14–17,34]. Most of the previous studies were not longitudinal in nature, but did, generally, involve larger samples of people with PD; thus, it is more likely that decreased VC relative to age- and sex-matched controls is the more common finding for people with PD. It is clear that the pattern of changes in speech breathing differed in the two groups. Typical older adults displayed significant increases in LVI, LVE, and %VC/syl. Participants with PD displayed significant decreases in LVI and LVT with no changes in LVE or %VC/syl.

The patterns demonstrated by the typical older adults in the current study are in agreement with what has been previously reported for older adults as compared to young adults[20–23], suggesting age-related changes occur in an ongoing fashion. Initiating speech at higher lung volumes allows older adults to utilize higher recoil pressures to generate subglottal pressure for speech production, potentially reducing their work of breathing. Increased LVE is the result of the participants using a higher LVI but not LVT. Increased LVE is often suggested to be related to decreased vocal fold closure resulting in a loss of air volume (greater %VC/syl)[21,23]. The present study demonstrates that age-related changes can be seen even over short periods of time (less than 4 years).

In contrast to the typical older adults, the participants with PD decreased LVI and LVT across the study period. Significant differences in speech breathing as compared to the typical older adults were only present in Wave 2. These data suggest that speech breathing changes as PD progresses, opposite of what is expected in typical aging, highlighting the importance of disease progression in the examination of speech breathing in PD. Initiating and terminating speech at lower lung volumes will have the effect of increasing muscular effort. Less recoil pressure is available at lower lung volumes. Thus, expiratory muscular pressure has to be used to generate subglottal pressure for speech. As a result, speech breathing may be more fatiguing for people with PD as the disease progresses, and the residual function for meeting more taxing speech demands (like speaker louder or longer) may not be available[35,36]. These findings strongly suggest the need to specifically target speech breathing in the treatment of dysarthria for people with PD. More specifics linking physiologic findings to treatment directions are described below.

There are at least two potential reasons why people with PD use lower lung volumes for speech, rather than higher ones. These changes may be due to chest wall rigidity. Chest wall rigidity could be expected to worsen with disease progression; however, no measures of rigidity were made in the current study. Solomon and Hixon (1993) suggested that the rib cage was less compliant than the abdomen in people with PD[9]. If rib cage rigidity did

worsen, it could make it difficult for people with PD to inspire to higher lung volumes. This hypothesis suggests the need to strengthen inspiratory and expiratory muscles in treatment to overcome chest wall rigidity. Expiratory muscle strength training has been shown to improve cough[37] and respiratory patterns during speech[38] in people with PD.

Alternatively, lower LVI and LVT may reflect issues with respiratory planning. Previous literature has suggested that some people with PD have motor programming or cognitive issues that impact speech production[39]. Huber and Darling (2011) found that the relationships between LVI, utterance length, and inspiratory duration were weaker for people with PD than they were for typical older adults and hypothesized that the weaker relationship may be due to cognitive-linguistic or planning issues[10]. Relatedly, Huber et al (2012) found that the people with PD took more breaths at boundaries unrelated to syntax in a reading passage than typical older adults, supporting the interpretation that individuals with PD have difficulty with linguistic and respiratory planning[33]. This hypothesis suggests that behavioral therapy be focused on improving speech planning and attention to speech breathing. Future research should try to elucidate the effects of muscle tone (like rigidity) as opposed to cognitive/motor planning issues in PD.

The major limitation of the present study is the small sample size. Due to death and significant disability, approximately 50% of the participants were lost from Wave 1 to Wave 2. Losses were sustained in both the control and the PD groups. Thus, the data set likely reflects participants starting in the mild or mild to moderate stages of PD and those who aged well. Unfortunately, we lost another 50% of the participants in the next 3–4 years and so could not examine further aging and disease progression with adequate sample sizes.

In summary, the current study is the first longitudinal study of speech breathing. Speech severity ratings worsened and speech rate increased for the participants with PD, but not the typical older adults. The typical older adults displayed expected speech breathing changes, increases in LVI, LVE, and %VC/syl. The participants with PD displayed the opposite pattern, decreases in LVI and LVT with no changes in LVE or %VC/syl. These data suggest that, unlike older adults, people with PD exert more expiratory muscle pressure during speech production. This may cause people with PD to expend more effort during speech production. The reasons for the decreased LVI and LVT in people with PD are not clear, but the two most likely interpretations are that increased chest wall rigidity makes increasing LVI and LVT difficult and/or that speech planning or cognitive-linguistic impairments results in poor coordination in speech production.

These results demonstrate the critical need for speech-language pathologists to understand how both aging and disease impact respiratory mechanics during speech and highlight the need to provide intervention that targets direct improvement of speech breathing. Further, these results suggest that the effects on respiration be considered when implementing therapy with people with PD. The most common therapeutic strategy used with people with PD is teaching them to increase vocal intensity. Typical speakers increase vocal intensity by increasing lung volume initiation. However, our results suggest that some people with PD may have significant difficulty making this type of respiratory adjustment. Thus, the respiratory changes described here are likely to result in a reduced capacity to increase vocal

intensity without significant effort. Understanding the effects of therapy strategies on respiration will allow clinicians to choose therapies that do not increase effort or fatigue in speaking and instead to choose therapies that train more efficient respiratory patterns.

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BIOSKETCHES FOR AUTHORS

Jessica E. Huber, Ph.D., CCC-SLP, is a Professor of Speech, Language, and Hearing Sciences at Purdue University. The aim of Dr. Huber's research program is to develop a theoretical account of the multiple factors that influence speech production and cognitive change in individuals with Parkinson's disease (PD) and to translate findings to clinical treatment resulting in improvements in communication. Dr. Huber's research is funded by the National Institutes of Health. She is the inventor of a small wearable device, the SpeechVive device, to treat communication impairments in people with PD. The device elicits the Lombard effect that can be exploited to improve speech clarity in individuals with PD while bypassing cognitive and sensory impairments. Her current research continues to examine the effects a number of speech therapy techniques including respiratory muscle strength training and using the SpeechVive device, and collaborating to develop a new balance-training paradigm.

Meghan Darling-White, Ph.D., CCC-SLP, is an Assistant Professor of Speech, Language, and Hearing Sciences at The University of Arizona. Her long-term research goal is to develop and validate theoretically driven, evidence-based interventions that have an impact on speech production skills as well as communicative participation in individuals with motor speech disorders. Her current work focuses on children with developmental dysarthria, particularly cerebral palsy. The goal of Dr. Darling-White's research, funded by the National Institutes of Health, is to quantify respiratory function during speech production in children with cerebral palsy and assess the effects of common intervention techniques, loudness and rate manipulations, on respiratory function and global speech outcomes. Dr. Darling-White is also embarking on the development of a tool to measure communicative participation in developmental dysarthria.

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CEU QUESTIONS

1. As people age, which of the following changes to speech breathing occur?

- **a.** Lung volume initiation decreases and lung volume termination increases.
- **b.** Lung volume initiation increases and lung volume termination decreases.
- **c.** Lung volume initiation and termination decrease.
- **d.** Lung volume initiation and excursion increase.
- **e.** Lung volume termination and excursion decrease.

Answer: D

1. As PD progresses, which of the following changes to speech breathing occur?

- **a.** Lung volume initiation decreases and lung volume termination increases.
- **b.** Lung volume initiation increases and lung volume termination decreases.
- **c.** Lung volume initiation and termination decrease.
- **d.** Lung volume initiation and excursion increase.
- **e.** Lung volume termination and excursion decrease.

Answer: C

1. As PD progresses, which of the following changes occur to speech production?

- **a.** Speech rate and utterance length decrease.
- **b.** Speech severity and speech rate increase.
- **c.** Speech severity and utterance length increase.
- **d.** Speech rate and utterance length increase.
- **e.** Speech severity increases and speech rate decreases.

Answer: B

1. As PD progresses,

- **a.** Speech breathing becomes more effortful.
- **b.** Speech breathing becomes more efficient.
- **c.** Speech breathing becomes more like that of young adults.
- **d.** Speech breathing becomes more like that of age- and sex-matched older adults.

e. Speech breathing doesn't change.

Answer: A

1. As the typical older adults aged, which of the following changes occur to speech production?

a. Speech rate and utterance length decrease.

b. Speech severity and speech rate increase.

c. Speech severity and utterance length increase.

d. Speech rate and utterance length increase.

e. None of the above.

Answer: E

LEARNING OBJECTIVES

As a result of this activity, the reader will be able to:

1. Describe changes to speech breathing that occur in typical older adults as they age.
2. Describe changes to speech breathing that occur in older adults with Parkinson's disease as the disease progresses.
3. Describe differences in changes to speech breathing with aging as opposed to changes as a result of Parkinson's disease.

Table 1:

Participant Demographic Information. Data are organized in pairs of participants with PD and age- and sex-matched typical older adults.

Pair	Subject	Age at Wave 1 (years; months)	Age at Wave 2 (years; months)	Years Since Diagnosis (Wave 1)	CLQT at Wave 2	Speech Severity at Wave 1	Speech Severity at Wave 2
1	M04PD	68;9	73;5	3.5	WNL	37.5%	82.0%
	M07OC	70;6	74;0	n/a	WNL	3.5%	7.5%
2	M09PD	72;8	76;8	9.0	Moderate	35.5%	73.0%
	M11OC	73;5	77;3	n/a	WNL	0.3%	3.0%
3	M10PD	70;0	73;7	4.5	Mild	3.0%	8.9%
	MO6OC	70;6	74;1	n/a	WNL	0.6%	2.0%
4	M11PD	82;0	85;2	3.75	WNL	43.0%	35.5%
	M090C	82;0	85;6	n/a	WNL	7.5%	0.3%
5	F01PD#	72;5	76;3	0.75	WNL	8.5%	50.5%
	F130C	73;5	76;9	n/a	WNL	4.5%	2.5%
6	F02PD	69;9	72;2	9.0	WNL	12.0%	43.0%
	F070C	65;8	69;7	n/a	WNL	1.0%	1.0%
7	F07PD	72;2	75; 11	−3.0 ^	WNL	3.5%	10.0%
	F020C	74;5	78;6	n/a	WNL	1.3%	4.7%
8	F04PD	74;3	76; 11	5.0	WNL	2.8%	11.4%
	F050C	73; 1	77;1	n/a	WNL	3.5%	7.1%

M = male, F = female; PD = Parkinson's disease, OC = age- and sex-matched control;

^ diagnosed with PD three years after participating in Wave 1;

possible TIA 1 year before Wave 2 data collection, but not definitively diagnosed; L=Liters; WNL=within normal limits. Higher numbers for speech severity indicate more severe speech ratings.

Table 2:

Means and Standard Errors (in parentheses) by Wave and Group

Dependent Variable	Older Adults Wave 1	Parkinson's Disease Wave 1	Older Adults Wave 2	Parkinson's Disease Wave 2
Sp Sev (percent)	2.78 (.87)	18.23 (6.13)	3.5 (.95)	39.3 (10.07)
SPL (decibels)	75.5 (.26)	76.9 (.23)	77.9 (.25)	77.8 (.22)
Utt Length (syl)	12.8 (.43)	13.0 (.48)	13.2 (.42)	12.0 (.40)
Rate (syl/sec)	4.6 (.04)	4.8 (.06)	4.6 (.04)	5.1 (.08)
LVI (%VC rEEL)	18.9 (.66)	23.0 (.79)	24.4 (1.11)	16.2 (.80)
LVT (%VC rEEL)	4.8 (.63)	3.8 (.94)	5.4 (.98)	−.72 (.76)
LVE (%VC)	14.1 (.45)	19.3 (.65)	19.0 (.62)	16.9 (.54)
%VC/syl (%VC)	1.18 (.03)	1.65 (.05)	1.54 (.04)	1.54 (.04)
VC (liters)	3.34 (.27)	2.99 (.32)	3.05 (.30)	2.77 (.30)

Sp Sev=speech severity, SPL=sound pressure level; Utt=utterance; syl=syllables; sec=second; LVI=lung volume initiation; LVT=lung volume termination; LVE=lung volume excursion; %VC=percent vital capacity; rEEL=relative to end expiratory level;