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Effects of Loudness Cues on Respiration in Individuals with Parkinson's disease

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Abstract

Individuals with Parkinson's disease (PD) demonstrate low vocal intensity (hypophonia) which results in reduced speech intelligibility. We examined the effects of three cues to increase loudness on respiratory support in individuals with PD. Kinematic data from the rib cage and abdomen were collected using respiratory plethysmography while participants read a short passage. Individuals with PD and normal age- and sex-matched controls (OC) increased sound pressure level (SPL) to a similar extent. As compared to OC, individuals with PD used larger rib cage volume excursions in all conditions. Further, they did not slow their rate of speech in noise as OC speakers did. Respiratory strategies used to support increased loudness varied with the cue, but the two groups did not differ in the strategies used. When asked to target a specific loudness, both groups used more abdominal effort than at comfortable loudness. Speaking in background noise resulted in the largest increase in SPL with the most efficient respiratory patterns, suggesting natural or implicit cues may be best when treating hypophonia in individuals with PD. Data demonstrate the possibility that both vocal loudness and speech rate are impacted by cognitive mechanisms (attention or self-perception) in individuals with PD.

Keywords

Parkinson's disease; hypophonia; respiratory kinematics; increasing vocal loudness

INTRODUCTION

Reduced speech intensity (hypophonia) is one of the cardinal features of the speech dysfunction associated with Parkinson's disease (PD) (1–3). Hypophonia in PD, an important contributor to decreased intelligibility, has been ascribed to glottal incompetence (4–6) and reduced respiratory support, related primarily to increased chest wall rigidity (7). In support of this hypothesis, studies of pulmonary function in individuals with PD have demonstrated disease-related reductions in forced vital capacity and forced expiratory volume in one second, even under the effects of anti-Parkinsonian medications (8, 9). Abnormal movements of the rib cage and abdomen during the production of speech in individuals with PD, including increased abdominal and decreased rib cage volume initiations, abdominal paradoxing, a smaller contribution of the rib cage to overall lung volume change, and increased variability of respiratory movements compared to healthy older adults, have been reported (7, 10–13).

Although it is clear that individuals with PD use different respiratory mechanisms than healthy older adults, it is not well-understood how individuals with PD utilize the respiratory system to support louder speech. Given the previously reported differences, it is likely that

people with PD use different respiratory strategies than healthy controls when speaking loudly. Huber, Stathopoulos, Ramig, and Lancaster (11) found that individuals with PD relied more on abdominal movement to make changes to lung volume while increasing loudness. Further, they documented individual differences in the strategies used to increase sound pressure level (SPL) in these patients. Young adults have also been shown to use a variety of strategies to increase loudness (14).

The respiratory strategy used to increase loudness may be affected by the cue used to elicit loud speech. Cues can vary by the degree of cognitive, emotional, and physical demands they place on the individual. Broadly, the cues used in studies of changes to respiratory kinematics with increased loudness in young adults can be categorized along the lines of being explicit cues ("say the sentence at a level that feels like twice as loud as normal" ((15); p. 1006) or targeting a specific SPL while using a meter for feedback (14, 16)) or implicit cues (talking in noise (17)). Explicit cues incorporate clear directions and instructions that require the individual to consciously manipulate his or her system to reach a desired goal (18). Implicit cues, sometimes referred to as "self-cues," are based on information that the individual is not consciously aware of, such as reactions to sensations or habits (18). Compared to explicit cues, implicit cues are more natural and can occur either with or without the individual's direct conscious awareness.

Studies utilizing explicit cues have reported that young adults begin to talk at higher lung volumes when they increase loudness (14–16). Winkworth and Davis (17) used speaking in noise to elicit louder speech and demonstrated a large amount of within and across participant variability in respiratory patterns. These authors hypothesized that under natural speaking conditions the mechanisms used to increase loudness were more variable than those reported for loudness changes in response to explicit cues.

Huber, Chandrasekaran, and Wolstencroft (19) examined how respiratory patterns are affected by cues to increase loudness in young adults. Three cues were used to elicit an increase in loudness: 1) targeting a loudness level 10 dB SPL higher than the individual's comfortable SPL using a sound level meter for feedback, 2) speaking at what the individual perceived to be twice his/her comfortable loudness, and 3) speaking in multi-talker noise. In the last two cueing conditions (twice comfortable loudness and noise), there was with no external feedback provided about loudness. Using these three cues, these authors demonstrated that the respiratory mechanisms supporting increased SPL did vary depending on the cue used to elicit the louder speech. These authors explained these findings by relating the cues to speakers' internal targets. According to this explanation, for any desired movement goal, there is an internal representation of the target. Cue-related changes to the internal target may result in different respiratory strategies used to support loud speech.

Such a suggestion is particularly relevant in the study of speech breathing in individuals with PD. Recent studies of both speech and limb movements have suggested that the reduced amplitude of movements and hypophonia associated with PD may be due to sensory deficits (20–23). This may cause individuals with PD to have an impaired perception of their own speech volume (21), resulting in inaccurate internal representations of movement targets and affecting how patients with PD respond to cues to increase loudness. In contrast, some studies of limb movement have shown individuals with PD to have adequate amplitude or force targets, but difficulty producing the appropriate force or amplitude suggesting the problem is related to motor execution as opposed to the accuracy of internal targets (24–26).

Whatever the underlying cause of the movement disorder associated with PD, cueing or bringing conscious attention to a motor task has been shown to result in improved motor

performance in individuals with PD in both limb movements and in speech production (25, 27–29). In the context of the current study, this may mean that explicit cues will result in greater loudness changes than implicit cues. The specificity of the target or the amount of feedback provided by the cue may influence the amount of attention required and the extent to which the targets are met. For example, asking a participant to speak at a particular SPL (and providing an SPL meter for feedback) may be more effective in eliciting a louder response than a cue that is less specific or one in which feedback is not objective (speaking “twice as loud”). The increased attentional load associated with an explicit cue may function as a “concurrent-task” which has been shown to have a negative impact on speech intensity (30). Therefore, it is possible that loudness changes as a result of a more natural, implicit cue (speaking in noise) may be achieved more easily and effectively than in explicit cues that require greater attention.

The question of whether different cues result in different respiratory strategies to support louder speech has significant clinical implications. Many individuals with PD are taught to use a louder voice in speech therapy. The most well known example of this type of therapy is the Lee Silverman Voice Treatment (LSVT) program (31, 32). However, many different cues are used in LSVT and other therapies to train the individuals to increase their loudness (33). The current study is relevant since one cue may be better than other cues in eliciting louder speech and more normal respiratory kinematic patterns in individuals with PD. If so, use of such a cue may improve therapy for hypophonia. It is also important to understand what respiratory strategies are used by individuals with PD and how they differ from those used by healthy older adults.

The purpose of this study was two-fold. First, we were interested in examining the respiratory mechanisms used by individuals with PD as they get louder. We hypothesized that individuals with PD would use different respiratory mechanisms to support louder speech than healthy older adults due to the changes to the speech system associated with PD. Second, we wanted to determine if persons with PD, like normal younger adults (19), utilize different strategies to increase loudness depending on how they are cued. The current study uses the same cues as were used by Huber, Chandrasekaran, and Wolstencroft (19). We hypothesized that there would be differences in resulting SPL and respiratory strategies employed for loudness increases depending on the cue.

METHODS

Participants

Fourteen individuals with idiopathic Parkinson’s disease (PD) by a neurologist and 14 age- and sex-matched healthy older adults (OC) participated in the study. Individuals with parkinsonian symptoms as a result of Parkinson plus syndromes were not included. There were 5 women and 9 men in each group. The mean age of the OC women was 71 years ($SD = 4$ years, range: 66–74 years), the women with PD was 69 years ($SD = 11$ years, range: 51–80 years), the OC men was 72 years ($SD = 6$ years, range: 66–81 years), and the men with PD was 76 years ($SD = 7$ years, range: 69–90 years). Individuals with PD were tested within 1–3 hours of taking their anti-Parkinsonian medications, even though it is not likely that drug cycles alter respiratory function in PD (7, 9). Detailed information about the participants with PD is provided in Table 1.

All participants reported no history of respiratory problems (including asthma), no head or neck cancer or surgery, and no formal training in singing or speaking. At the time of experimental screening, participants were free from infections, colds and allergies, had been non-smokers for at least 5 years, and spoke a North American dialect of English. All participants were required to be living independently in the community and ambulatory,

demonstrate normal hearing (34), and pass the Mini-Mental State Exam (35). Additionally, OC participants reported no history of neurological disease, were determined by the experimenter to have normal speech, language, and voice. Older control participants demonstrated normal lung function by producing vital capacity (VC), forced VC, and forced expiratory volume in one second at > 80% of expected values based on age, sex, height, weight, and ethnicity (VacuMed Discovery Handheld Spirometer).

Ratings of reduced loudness, breathiness and hoarseness were made for each participant with PD by three experienced speech-language pathologists. Reduced loudness was rated from a sentence, presented without equalizing intensity. Breathiness and hoarseness were rated from sustained vowel productions, presented at equal intensity. On average, individuals with PD had mild to moderate speech difficulties (Table 1). For the characteristic of reduced loudness, 7 of the 14 participants were rated as being mild, 2 moderate, and 1 severe. Four of the 14 were rated as having normal loudness. For the characteristic of breathiness, 8 of the 14 participants were rated as moderate and 1 severe. Five of the 14 were rated as having no breathiness. For the characteristic of hoarseness, 5 of the 14 participants were rated as mild, 1 moderate, and 3 severe. Five of the 14 were rated as having no hoarseness, and 4 of these 5 were not rated as breathy either. These ratings suggest that two-thirds of the individuals with Parkinson's disease had some changes to vocal fold vibratory patterns or glottal configurations, as evidenced by the changes to perceptual voice characteristics.

Equipment

A microphone, held at a constant 6-inch mouth-to-microphone distance, was used to transduce the acoustic signal. The microphone signal was recorded to a digital audiotape and later digitized using a computer software program, Praat (36). An SPL meter, coupled to the microphone, provided feedback to the participants when they were asked to target a specific SPL and amplified the microphone signal during the study. The gain provided by the SPL meter to the microphone signal was factored in when calibrating the acoustic signal. The SPL meter provided a digital, numerical display of average SPL and was projected on a television screen placed next to the display of the reading passage.

Respiratory kinematic data was transduced with the Resptrace (Ambulatory Monitoring, Inc.). Rib cage displacement was transduced by an elastic band placed around the rib cage just under the axilla. Similarly, abdominal movement was tracked by a second band placed around the abdomen below the last rib, at the level of the participant's belly button. Signals from the Resptrace bands were digitized through the analog-to-digital converter in the Optotrak system (Northern Digital Inc.) with input from a second "room" microphone.

Procedures and Speech Stimuli

With the Resptrace bands in place, all participants performed maximum capacity tasks which were used to estimate maximum lung (VC), rib cage (RCC), and abdominal (ABC) volume capacities for each participant (37).

The combined effect of changes in the rib cage (RC) and abdominal (AB) volumes reflect change in lung volume (LV) (38). The sum of RC and AB movement signals were compared to a digital spirometer signal (SP), all collected during rest breathing and "speech-like breathing" (19). A least squares analysis was used to determine the best correction factors (k_1 and k_2) for the RC and AB signals in the formula (19):

$$SP = k_1(RC) + k_2(AB)$$

Estimated lung volume was computed during all speech tasks by summing the RC and AB volumes after the correction factors were applied.

During data collection, the reading passage was displayed on the computer screen in front of the participant. Participants were instructed to read the “Papa passage” (39) such they were clear and audible to an experimenter sitting about four feet away. The passage was read twice in each of the following four experimental conditions (19):

- COMF: Participants were asked to read the passage at comfortable loudness and pitch.
- COMF+10: Participants were instructed to read the passage while maintaining SPL at 10 dB (+/- 2 dB) above their comfortable SPL. An SPL meter was used for feedback about SPL.
- 2XCOMF: Participants were told to read the passage at a level they felt was “twice as loud” as their comfortable loudness. No feedback was given to the participants regarding how loud they were. When this condition did not directly follow COMF, participants were acclimated to their comfortable loudness before giving the “twice as loud” cue.
- NOISE: Background multi-talker noise (AUDiTEC of St. Louis) was introduced at 70 dBA through free-field speakers positioned in front of the participant at a distance of approximately 40 inches. The participants were then instructed to read the passage.

The COMF condition was always performed first, and the order of the other three loudness conditions was counterbalanced across participants.

Measurements

The average SPL, in decibels (dB), for each sentence of the reading passage was measured using Praat (36). The speech rate was measured in syllables per second by dividing the number of syllables produced in each breath group by its duration.

The respiratory kinematic measurements were made from each breath group in the passage using algorithms written to run in Matlab (Mathworks, Inc.). They were expressed as a percentage of capacity (VC, RCC, and ABC) and relative to the end expiratory level (EEL). The EEL for each trial of the reading passage was computed as an average of at least three steady troughs of rest breathing preceding that trial. For measurement of respiratory kinematics, the respiratory signals were displayed along with a time-locked acoustic signal.

1. Speech initiation measurements were made from the lung volume (LVI), rib cage (RCVI), and abdominal volume (ABVI) signals at the point where voicing started in the acoustic signal.
2. Speech termination measurements were made from the lung volume (LVT), rib cage (RCVT), and abdominal volume (ABVT) signals at the point that voicing ended in the acoustic signal.
3. Speech excursions (lung volume (LVE), rib cage (RCVE), and abdominal (ABVE)) were calculated by subtracting the volume at termination from the volume at initiation.

Accuracy of selection of speech initiation and termination points was verified by listening to the audio signal.

Statistical Analysis

Means were computed for each participant in each condition. A two-factor repeated measures analysis of variance (ANOVA) was used with condition (loudness cue) as the within factor and group (PD vs. OC) as the between factor. Tukey's honestly significantly different (HSD) tests were used to assess difference within factors for any significant results from the ANOVA. The alpha level for all tests was set at $p < 0.05$. Hedge's bias-corrected effect size tests were completed for all significant pairwise comparisons (40).

Data from 5 randomly selected participants (2 OC and 3 PD) were re-analyzed by a second individual in the laboratory. Inter-measurer reliability was evaluated by computing Pearson Product Moment Correlations between the original measurements and reliability measurements. The correlations for all measurements fell in the range of 0.89 to 0.98, indicating good inter-measurer reliability.

RESULTS

The statistical results for the ANOVA for the main effects and interactions are presented in Table 2. Means and standard deviations for group and condition are presented in Table 3. Effect sizes (d) for the significant pairwise comparisons are given in the text.

For SPL, there was a significant condition effect but no significant group or interaction effects. SPL was significantly higher for the three loud conditions than in COMF (COMF +10 [$d = 1.48$], 2XCOMF [$d = 1.26$], and SPL in the NOISE was significantly greater than in 2XCOMF [$d = -0.78$] and COMF+10 [$d = -0.57$] (see Figure 1).

For rate of speech, there was a significant group effect and a significant group by condition interaction. The OC participants produced fewer syllables per second in NOISE than in COMF [$d = 1.06$], but there were no differences across conditions for the individuals with PD [$d = -0.01$]. Means and standard deviations (in parentheses) were as follows: OC COMF: 4.7 (0.36), COMF+10: 4.5 (0.39), 2XCOMF: 4.6 (0.32), NOISE: 4.4 (0.34); PD COMF: 4.6 (0.76), COMF+10: 4.8 (0.73), 2XCOMF: 4.8 (0.91), NOISE: 4.7 (0.83).

For LVI, RCVI, LVT, and RCVT, no significant group, condition, or interaction effects were observed (see Figure 2). For ABVI and ABVT, there were significant condition effects but no group or interaction effects. Both ABVI and ABVT were significantly lower in COMF+10 than in COMF (ABVI [$d = 0.58$], ABVT [$d = 0.71$]) and NOISE (ABVI [$d = 0.64$], ABVT [$d = 0.63$]) (see Figure 3).

For LVE, RCVE, and ABVE, there were significant condition effects. All excursions were significantly larger for the three loud conditions as compared to COMF (LVE: COMF+10 [$d = -0.46$], 2XCOMF [$d = -0.52$], NOISE [$d = -0.56$]; RCVE: COMF+10 [$d = -0.30$], 2XCOMF [$d = -0.35$], NOISE [$d = -0.34$]; ABVE: COMF+10 [$d = -0.34$], 2XCOMF [$d = -0.26$], NOISE [$d = -0.32$]). For RCVE, there was also a significant group effect. Significantly smaller RCVE were observed in OC as compared to patients with PD [$d = -0.81$] (see Figure 4).

DISCUSSION

Given a particular cueing condition, both groups (OC and PD) increased SPL to a similar extent (see Figure 1). Differences in respiratory kinematics were demonstrated across the cueing conditions for both groups and between the individuals with PD and the normal control speakers. The laryngeal system was likely to be involved in the changes in loudness across the three conditions; however, the current paper focused on changes to the respiratory

system. The differences in respiratory kinematics clearly indicate that the cues did affect how the respiratory system was used in the louder speech conditions.

Individuals with PD used respiratory strategies similar to those used by OC participants to increase loudness, increasing LVE, RCVE, and ABVE for all loud conditions. This observation is consistent with aerodynamic requirements of loud speech. Increases in loudness were associated with increased volume expenditure due to the need for a higher driving pressure regardless of group or cueing condition.

An interesting difference between the two groups was noted in RCVE. Parkinson's disease has been hypothesized to result in rigidity of the chest wall (7, 8). This has been thought to result in decreased overall chest wall movement and hence, decreased respiratory support for speech. Given this, our finding that individuals with PD demonstrated increased RCVE compared to OC participants was a surprising one. Closer examination of the data revealed that individuals with PD did not increase RCVI but reached lower RCVT levels, resulting in a larger RCVE (see Figure 4). This effect was not condition specific; the magnitude of the effect is about the same, regardless of how the individuals were cued.

We propose an explanation for this observation that is related to the changes that occur to the physiology of the speech system in individuals with PD. Rigidity of the chest wall associated with PD would make it difficult for these individuals to increase RCVI. In addition, individuals with PD usually have associated laryngeal impairment, including decreased synergy and activation of the laryngeal muscles, glottal incompetence, and fatigue (1, 2, 6, 7). Due to the reduced glottal efficiency, individuals with PD need to produce greater respiratory drive as compared to healthy older adults. This active respiratory drive may have been generated by the rib cage in participants with PD by increasing RCVE, in particular by lowering RCVT.

Based on findings with young adults (19), we hypothesized that the type of cue used to elicit loud speech would influence the respiratory strategies used to increase loudness in individuals with PD. We also speculated that some cues may be more difficult for individuals with PD. The results demonstrated that respiratory strategies used to increase loudness do depend on the cue used to elicit changes in loudness; however, the differences among the cues were the same for OC participants and individuals with PD for the most part.

The NOISE condition was the most effective in eliciting increased SPL in both groups. This cueing condition produced the greatest increase in SPL, and the mean SPL levels for individuals with PD were closest to the OC participants in NOISE (see Figure 1). In addition, NOISE appeared to elicit the most efficient respiratory strategies in increasing loudness in both groups. The mid-lung volume range is used by most speakers in normal comfortable level speaking conditions (16, 41). At the mid-lung volume range, less muscular effort is required to generate pressure for speech since the recoil pressure available is close to the amount of subglottal pressure required for speech (42). Both groups utilized volumes in the mid-lung volume range during the NOISE condition, suggesting speaking loudly in this condition was less effortful than the other two loud conditions. Lung volumes used by individuals with PD are most like those used by OC participants in NOISE (see Figure 2); there is a small increase in lung volume initiation in NOISE (see Figure 2), suggesting use of higher passive recoil pressures, and the least amount of abdominal muscle tension was used in NOISE, as compared to COMF+10 and 2XCOMF (see Figure 3). Therefore, the greatest loudness increase was achieved with the least respiratory effort in the NOISE condition by both groups.

The COMF+10 cue, in contrast, appeared to elicit a more effortful respiratory strategy in making changes to loudness for both groups. Both ABVI and ABVT were lower in COMF+10 than in COMF and NOISE (see Figure 3), indicating the use of expiratory muscle force to produce more driving force for speech. This finding was very different from respiratory kinematic patterns observed in COMF+10 in younger adults who used higher LVI and RCVI while not changing AB volumes during a sentence task (19).

The findings related to cues have practical implications for therapy. It appears that for individuals with PD, a more natural cue, such as the NOISE cue, is more effective in eliciting efficient respiratory strategies than a more explicit cue, such as the COMF+10 cue. The COMF+10 cue may have been particularly difficult for both OC participants and individuals with PD because it required the speakers to divide their attention between reading the paragraph and maintaining the target SPL. However, it is important to remember that mechanisms used by individuals in response to a cue may differ from those used once they have learned to cue themselves through training or treatment (11). These data may suggest that a device that provides low levels of noise during speech (along the lines of delayed auditory feedback) may be an option for individuals who are unable to learn to cue themselves.

One important difference was demonstrated in the current study between OC participants and individuals with PD. Individuals with PD did not slow their rate of speech in NOISE as older and younger adults (19) did. For individuals with PD, this may affect their intelligibility in noise. This implication is supported by earlier findings that the intelligibility of utterances produced in noise was higher when the speech samples collected in quiet and noise were presented at the same signal to noise ratios (43). Individuals with PD may not realize the need to reduce their rate of speech when speaking in noise due to problems with self-monitoring of speech (20, 21, 23, 44).

These findings have implications for hypophonia associated with PD and its treatment. Our data support the idea that hypophonia in individuals with PD does not appear to be due to disease-related physiologic limitations in increasing loudness, similar to the finding that individuals with PD can produce a normal stepping pattern with cueing (28). For all conditions, participants with PD increased their vocal intensity to a similar extent as OC and largely used similar respiratory strategies in speaking louder. These results, especially taken with the findings related to rate of speech in noise, suggest that hypophonia may be related to a cognitive or perceptual difficulty, as has been suggested by other researchers (20, 21, 23, 44–46).

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References

1. Darley FL, Aronson AE, Brown JR. Clusters of deviant speech dimensions in the dysarthria. *J Speech and Hear Res.* 1969; 12:462–496. [PubMed: 5811846]
2. Darley FL, Aronson AE, Brown JR. Differential diagnostic patterns of dysarthria. *J Speech and Hear Res.* 1969; 12:246–269. [PubMed: 5808852]
3. Logemann JA, Fisher HB, Boshes B, Blonsky ER. Frequency and cooccurrence of vocal tract dysfunctions in the speech of a large sample of Parkinson patients. *J Speech Hear Disord.* 1978; 43:47–57. [PubMed: 633872]

4. Hanson DG, Gerratt BR, Ward PH. Cinegraphic observations of laryngeal function in Parkinson's disease. *Laryngoscope*. 1984; 94:348–353. [PubMed: 6700351]
5. Perez KS, Ramig LO, Smith ME, Dromey C. The Parkinson larynx: Tremor and videostroboscopic findings. *J Voice*. 1996; 10(4):354–361. [PubMed: 8943139]
6. Smith ME, Ramig LO, Dromey C, Perez KS, Samandari R. Intensive voice treatment in Parkinson disease: Laryngostroboscopic findings. *J Voice*. 1995; 9(4):453–459. [PubMed: 8574314]
7. Solomon NP, Hixon TJ. Speech breathing in Parkinson's disease. *J Speech and Hear Res*. 1993; 36:294–310. [PubMed: 8487522]
8. De Pandis M, Staracce A, Stefanelli F, et al. Modification of respiratory function parameters in patients with severe Parkinson's disease. *Neurol Sci*. 2002; 23 (Suppl 2):S69–70. [PubMed: 12548348]
9. Weiner P, Inselberg R, Davidovich A, et al. Respiratory muscle performance and the perception of dyspnea in Parkinson's disease. *Can J Neurol Sci*. 2002; 29:68–72. [PubMed: 11858539]
10. Bunton K. Patterns of lung volume use during an extemporaneous speech task in persons with Parkinson's disease. *J Commun Disord*. 2005; 38:331–348. [PubMed: 15963334]
11. Huber JE, Stathopoulos ET, Ramig LO, Lancaster SL. Respiratory function and variability in individuals with Parkinson disease: Pre- and post-Lee Silverman Voice Treatment. *J Med Speech-Lang Path*. 2003; 11:185–201.
12. Lethlean JB, Chenery HJ, Murdoch BE. Disturbed respiratory and prosodic function in Parkinson's disease: A perceptual and instrumental analysis. *Australian Journal of Human Communication Disorders*. 1990; 18(2):83–97.
13. Murdoch BE, Chenery HJ, Bowler S, Ingram JCL. Respiratory function in Parkinson's subjects exhibiting a perceptible speech deficit: A kinematic and spirometric analysis. *J Speech Hear Disord*. 1989; 54:610–626. [PubMed: 2811341]
14. Stathopoulos ET, Sapienza CM. Respiratory and laryngeal measures of children during vocal intensity variation. *J Acoust Soc Am*. 1993; 94(5):2531–2543. [PubMed: 8270731]
15. Dromey C, Ramig LO. Intentional changes in sound pressure level and rate: Their impact on measures of respiration, phonation, and articulation. *J Speech Lang Hear Res*. 1998; 41:1003–1018. [PubMed: 9771624]
16. Stathopoulos ET, Sapienza CM. Developmental changes in laryngeal and respiratory function with variations in sound pressure level. *J Speech Lang Hear Res*. 1997; 40:595–614. [PubMed: 9210117]
17. Winkworth AL, Davis PJ. Speech breathing and the Lombard effect. *J Speech Lang Hear Res*. 1997; 40:159–169. [PubMed: 9113867]
18. Gazzaniga, MS.; Ivry, RB.; Mangun, GR. *Cognitive Neuroscience The Biology of the Mind*. New York: W.W. Norton & Company; 1998.
19. Huber JE, Chandrasekaran B, Wolstencroft JJ. Changes to respiratory mechanisms during speech as a result of different cues to increase loudness. *J Appl Physiol*. 2005; 98:2177–2184. [PubMed: 15705723]
20. Fox CM, Morrison CE, Ramig LO, Sapir S. Current perspectives on the Lee Silverman Voice Treatment Program (LSVT) for individuals with idiopathic Parkinson's disease. *Am J Speech Lang Pathol*. 2002; 11:111–123.
21. Ho AK, Bradshaw JL, Iansek R. Volume perception in Parkinsonian speech. *Mov Disord*. 2000; 15(6):1125–1131. [PubMed: 11104195]
22. Rickards C, Cody F. Proprioceptive control of wrist movements in Parkinson's disease: Reduced muscle vibration-induced errors. *Brain*. 1997; 120:977–990. [PubMed: 9217682]
23. Trail M, Fox C, Ramig LO, Sapir S, Howard J, Lai E. Speech treatment for Parkinson's disease. *Neuro Rehabilitation*. 2005; 20:205–221. [PubMed: 16340101]
24. Longstaff M, Mahant P, Stacy M, Van Gemmert A, Leis B, Stelmach G. Discrete and dynamic scaling of the size of continuous graphic movements of parkinsonian patients and elderly controls. *J Neurol Neurosurg Psychiatry*. 2003; 74:299–304. [PubMed: 12588912]
25. Morris M, Iansek R, McGinley J, Matyas T, Huxham F. Three-dimensional gait biomechanics in Parkinson's disease: evidence for a centrally mediated amplitude regulation disorder. *Mov Disord*. 2005; 20:40–50. [PubMed: 15390033]

26. Stelmach G, Teasdale N, Phillips J, Worringham C. Force production characteristics in Parkinson's disease. *Exp Brain Res*. 1989; 76:165–172. [PubMed: 2753097]
27. Ho AK, Bradshaw JL, Iansek R, Alfredson R. Speech volume regulation in Parkinson's disease: Effects of implicit cues and explicit instructions. *Neuropsychologia*. 1999; 37:1453–1460. [PubMed: 10617265]
28. Morris ME, Iansek R, Matyas TA, Summers JJ. Stride length regulation in Parkinson's disease Normalization strategies and underlying mechanisms. *Brain*. 1996; 119:551–568. [PubMed: 8800948]
29. Oliveira RM, Gurd JM, Nixon P, Marshall JC, Passingham RE. Micrographia in Parkinson's disease: the effect of providing external cues. *J Neurol Neurosurg Psychiatry*. 1997; 63:429–433. [PubMed: 9343118]
30. Ho AK, Iansek R, Bradshaw JL. The effect of a concurrent task on Parkinsonian speech. *J Clin Exp Neuropsychol*. 2002; 24:36–47. [PubMed: 11935422]
31. Ramig LO, Countryman S, Thompson LL, Horii Y. Comparison of two forms of intensive speech treatment for Parkinson Disease. *J Speech and Hear Res*. 1995; 38:1232–1251. [PubMed: 8747817]
32. Ramig LO, Sapir S, Fox C, Countryman S. Changes in vocal intensity following intensive voice treatment (LSVT) in individuals with Parkinson disease: A comparison with untreated patients and normal age-matched controls. *Mov Disord*. 2001; 16:79–83. [PubMed: 11215597]
33. Ramig LO, Verdolini K. Treatment efficacy: Voice disorders. *J Speech Lang Hear Res*. 1998; 41:S101–S116. [PubMed: 9493749]
34. Ventry IM, Weinstein BE. Identification of elderly people with hearing problems. *ASHA*. 1983; 25:37–42. [PubMed: 6626295]
35. Folstein MF, Folstein SE, McHugh PR. "Mini Mental State" A practical method for grading the cognitive state of patients for the clinician. *J Psychiat Res*. 1975; 12:189–198. [PubMed: 1202204]
36. Boersma, P.; Weenink, D. Praat. 4.1. Amsterdam: Institute of Phonetic Sciences; 2003.
37. Hoit JD, Hixon TJ. Age and speech breathing. *J Speech and Hear Res*. 1987; 30:351–366. [PubMed: 3669642]
38. Konno K, Mead J. Measurement of the separate volume changes of the rib cage and the abdomen during breathing. *J Appl Physiol*. 1967; 22(3):407–422. [PubMed: 4225383]
39. Sapienza CM, Stathopoulos ET. Speech task effects on acoustic and aerodynamic measures of women with vocal nodules. *J Voice*. 1995; 9(4):413–418. [PubMed: 8574307]
40. Hedges, L.; Olkin, I. *Statistical Methods for Meta-Analysis*. New York: Academic Press; 1985.
41. Hixon TJ, Goldman MD, Mead J. Kinematics of the chest wall during speech production: Volume displacements of the rib cage, abdomen, and lung. *J Speech and Hear Res*. 1973; 16:78–115. [PubMed: 4267384]
42. Hixon, TJ. Respiratory function in speech. In: Minifie, FD.; Hixon, TJ.; Williams, F., editors. *Normal Aspects of Speech, Hearing, and Language*. Englewood Cliffs, NJ: Prentice-Hall, Inc; 1973. p. 73-125.
43. Van Summers W, Pisoni DB, Bernacki RH, Pedlow RI, Stokes MA. Effects of noise on speech production: Acoustic and perceptual analyses. *J Acoust Soc Am*. 1988; 84(3):917–928. [PubMed: 3183209]
44. Forrest K, Nygaard L, Pisoni DB, Siemers E. Effects of speaking rate on word recognition in Parkinson's disease and normal aging. *J Med Speech-Lang Path*. 1998; 6:1–12. [PubMed: 21637728]
45. Ramig LO, Countryman S, O'Brien C, Hoehn M, Thompson L. Intensive speech treatment for patients with Parkinson's disease: Short- and long-term comparison of two techniques. *Neurology*. 1996; 47:1496–1504. [PubMed: 8960734]
46. Ramig LO, Sapir S, Countryman S, et al. Intensive voice treatment (LSVT®) for patients with Parkinson's disease: a 2 year follow up. *J Neurol Neurosurg Psychiatry*. 2001; 71:493–498. [PubMed: 11561033]

Figure 1.

Sound pressure level for group and condition. Bars represent means; lines represent standard errors. OC = older control participants, PD = individuals with Parkinson's disease, COMF = comfortable loudness condition, COMF+10 = targeting 10 dB above comfortable loudness SPL, 2XCOMF = speaking at twice comfortable loudness, NOISE = speaking in multi-talker noise.

Figure 2.

Lung volume for group and condition. Bars represent excursions. Lines represent standard errors of initiations and terminations. OC = older control participants, PD = individuals with Parkinson's disease, COMF = comfortable loudness condition, COMF+10 = targeting 10 dB above comfortable loudness SPL, 2XCOMF = speaking at twice comfortable loudness, NOISE = speaking in multi-talker noise.

Figure 3.

Abdominal volume across cues. Bars represent excursions. Lines represent standard errors of initiations and terminations. COMF = comfortable loudness condition, COMF+10 = targeting 10 dB above comfortable loudness SPL, 2XCOMF = speaking at twice comfortable loudness, NOISE = speaking in multi-talker noise.

Figure 4. Rib cage volume across groups. Bars represent excursions. Lines represent standard errors of initiations and terminations. OC = older control participants, PD = individuals with Parkinson's disease.

Table 1

Detailed Information about Participants with Parkinson's disease

PD Participant	Age (years)	Time since diagnosis	History of Speech treatment	Drugs	Perceptual Speech Severity
F01PD	73	1 yr	No	Matiptex	Mild
F02PD	70	9 yrs	No	Welbutrin Eldepryl Zoloft	Normal-Mild
F03PD	51	2 yrs	No	Sinemet Contan Elerail	Normal-Mild
F04PD	74	5 yrs	No	Sinemet Eldepryl Bromocriptine	Normal
F05PD	80	6 yrs	No	Eldpyryl Stalevo Requip	Mild
M01PD	83	5 yrs	No	None	Moderate
M02PD	76	5 yrs	No	Sinemet Requip Contan	Moderate
M03PD	69	3-4 yrs	No	Pemax Stalevo	Normal-Mild
M04PD	90	3 yrs	No	Carbidopa Amratadase	Moderate
M05PD	70	4 yrs	No	Stalevo Sinemet Mirapet	Normal-Mild
M06PD	75	3 yrs	Yes	Carbidopa Premex Contan	Moderate
M07PD	73	10 yrs	2 yrs	Sinemet Lipator Metropocol Prozac	Mild
M08PD	70	4 yrs	No	Sinemet	Mild
M09PD	82	4 yrs	No	Amantazine Sinemet Flomax	Mild

Table 2

Statistical summary for group, condition, and group by condition effects from ANOVA.

Measures	Group (df = 1)		Condition (df = 3)		Group * Condition (df = 3)	
	F	p	F	p	F	p
Sound Pressure Level	3.197	0.0859	59.6	0.0000 *	1.307	0.2783
Duration	0.0328	0.8576	1.4981	0.2216	1.4701	0.2292
Syllables per second	0.590	0.4495	2.730	0.0495 *	3.902	0.0119 *
Lung volume initiation	0.9098	0.3489	1.6101	0.1938	1.9964	0.1214
Lung volume termination	0.0010	0.9748	1.9383	0.1303	1.8279	0.1489
Lung volume excursion	3.2892	0.0813	10.9693	0.0000 *	0.3893	0.7610
Rib cage volume initiation	0.0176	0.8954	2.3815	0.0758	0.6869	0.5628
Rib cage volume termination	1.2742	0.2693	1.0526	0.3742	0.6366	0.5937
Rib cage volume excursion	5.0795	0.0329 *	7.5968	0.0002 *	2.0098	0.1194
Abdominal volume initiation	1.0718	0.3096	4.7634	0.0042 *	2.2382	0.0904
Abdominal volume termination	0.2114	0.6495	5.7297	0.0013 *	2.1368	0.1024
Abdominal volume excursion	0.3192	0.5769	5.8874	0.0011 *	0.5755	0.6328

df = degrees of freedom

* indicates factors that are statistically significant at $p < 0.05$.

Table 3

Means and standard deviations (in parentheses) for the group and condition factors.

Measures	Groups			Condition		
	OC	PD	COMF	COMF+10	2XCOMF	NOISE
Sound Pressure Level (dB)	84.3 (5.7)	81.3 (7.5)	76.2 (4.7)	84.8 (6.5)	83.5 (6.5)	87.4 (2.8)
Duration (sec)	2.8 (0.59)	2.8 (0.73)	2.8 (0.61)	2.8 (0.67)	2.9 (0.73)	2.9 (0.65)
Syllables per second	4.6 (0.37)	4.7 (0.79)	4.7 (0.59)	4.7 (0.60)	4.7 (0.67)	4.5 (0.64)
Lung volume initiation (%VC)	18.7 (15.3)	23.6 (14.8)	20.5 (11.7)	20.0 (16.1)	20.1 (16.8)	24.1 (16.1)
Lung volume termination (%VC)	1.6 (12.4)	1.8 (14.4)	3.8 (10.7)	0.0 (13.1)	-0.2 (15.4)	14.1 (3.2)
Lung volume excursion (%VC)	17.1 (5.3)	21.8 (9.0)	16.7 (5.6)	20.0 (8.0)	20.3 (7.7)	20.9 (8.8)
Rib cage volume initiation (%RCC)	26.7 (18.5)	27.6 (22.2)	22.8 (12.4)	30.0 (22.9)	26.7 (23.6)	28.9 (21.0)
Rib cage volume termination (%RCC)	13.0 (16.1)	6.2 (19.5)	7.6 (10.8)	12.0 (19.0)	8.2 (22.9)	10.6 (18.1)
Rib cage volume excursion (%RCC)	13.7 (6.5)	21.3 (11.4)	15.2 (8.0)	18.0 (10.7)	18.5 (10.6)	18.4 (10.4)
Abdominal volume initiation (%ABC)	1.2 (8.1)	3.8 (9.3)	3.9 (6.9)	-0.6 (8.4)	1.5 (10.0)	5.0 (8.8)
Abdominal volume termination (%ABC)	-8.1 (10.8)	-6.7 (8.6)	-4.5 (9.5)	-11.1 (8.5)	-8.6 (11.0)	-5.4 (9.1)
Abdominal volume excursion	9.2 (4.5)	10.5 (7.6)	8.5 (5.3)	10.5 (6.4)	10.1 (6.8)	10.4 (6.5)

OC = older control participants; PD = individuals with Parkinson's disease; COMF = at comfortable loudness; COMF+10 = targeting 10 dB above comfortable loudness SPL; 2XCOMF = speaking at twice comfortable loudness; NOISE = speaking in multi-talker noise