The Effect of Background Noise on the Speech Intensity of Individuals with Hypophonia Associated with Parkinson's Disease

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Reduced speech intensity or hypophonia often is a consequence of hypokinetic dysarthria associated with Parkinson's disease (PD). Because the impact of hypophonia often is exacerbated in noise, the introduction of background noise during conversational tasks is a particularly relevant context in which to study this speech symptom. The purpose of this study was to examine the effect of background noise on the speech intensity of 30 participants with hypophonia associated with PD and 15 control participants. Measures of maximum speech intensity and habitual conversational speech intensity were obtained for both groups. Conversational speech intensity was measured in various intensity levels of background noise in both groups. Results revealed significant differences between groups in maximum speech intensity, habitual conversational speech intensity, and conversational speech intensity in background noise. Specifically, participants with PD were significantly less intense (by ~5 dB sound pressure level [SPL]) than control participants in various intensity levels of background noise. Furthermore, participants with PD demonstrated a similar but attenuated pattern of response to the various intensity levels of background noise. This suggests that a potentially important and fundamental aspect of hypophonia in PD may be the lack of ability to accurately regulate speech intensity relative to background noise conditions.

INTRODUCTION

One of the most disabling aspects of Parkinson's disease (PD) is its effect on spoken communication. It is estimated that more than 75% of individuals with PD will present with speech and voice abnormalities related directly to PD (Logemann, Fisher, Boshes, & Blonsky, 1978; Sapir, Ramig, Hoyt, Countryman, O'Brien, & Hoehn, 2002). Reduced speech intensity, or hypophonia, often is a consequence of hypokinetic dysarthria associated with PD. Hypophonia can be a disabling aspect of hypokinetic dysarthria and a frustrating experience for speakers with PD.

Hypophonia Associated with Hypokinetic Dysarthria

Although not originally described as a distinctive feature in Darley, Aronson, and Brown's (1969) clinical characterization of hypokinetic dysarthria, hypophonia often emerges as an initial speech symptom in the beginning stages of PD (Logemann et al., 1978) but it is not present in all individuals with hypokinetic dysarthria. Ludlow and Bassich (1984) and Gamboa et al. (1997) found that hypophonia was present in 42% and 49% of individuals they studied with hypokinetic dysarthria, respectively. Anecdotally, there are reports that individuals with PD often complain that others ask for them to speak louder and to repeat themselves, and communication partners indicate the individuals with hypophonia often lack an awareness of their softer voice or reduced loudness (Adams & Dykstra, 2008; Dromey & Adams, 2000; Duffy, 1995; Ramig, 1998). Although reduced loudness is a salient perceptual characteristic in individuals with PD (Darley, Aronson, & Brown, 1975; Logemann et al., 1978), there are inconsistencies in acoustic evidence for this claim. Illes, Metter, Hanson, and Iritani (1988) demonstrated reduced loudness in individuals with PD during a reading task. There also is a small but emerging literature that has confirmed reduced speech intensity levels in speakers with PD during conversational tasks (Ho, Iansek, & Bradshaw, 1999; Moon, 2005) and monologues (Fox & Ramig, 1997). In general, however, few studies have been able to find significant acoustic differences in speech intensity between individuals with PD and healthy age-matched control participants (Canter, 1963; Kempler & Van Lancker, 2002; Metter & Hanson, 1986).

There appears to be dichotomy between clinical, perceptual impressions of hypophonia in PD and the instrumental or acoustic measures that often fail to capture this perceptual speech feature. Possible explanations could include patient compensation (i.e., speaking louder) during formal speech testing (Adams & Dykstra, 2008) and the complexity of measuring speech intensity in experimental conditions. Other variables such as speech task (i.e., imitation of phrases, reading aloud, spontaneous conversation) and the nature of the speaking environment (artificial experimental testing in a laboratory versus spontaneous conversation in one's natural environment) also are important to consider because these factors can differentially impact the speaker's speech intensity level.

To obtain valid estimates of hypophonia in PD, it may be necessary to assess speech intensity in natural speaking environments using naturalistic speech tasks such as conversational speech.

Typically, researchers who have investigated hypophonia in PD have measured speech intensity during reading tasks (e.g., Canter, 1963) and during repetition or imitation of short sentences (e.g., Ludlow & Bassich, 1984), most likely in an attempt to control for experimental variability. However, these studies have not found significant differences in speech intensity between healthy control participants and individuals with PD. The confirmation of reduced speech intensity levels in speakers with PD (Fox & Ramig, 1997; Ho et al., 1999; Moon, 2005) has emerged primarily during conversational speaking tasks. It is likely that conversational speaking tasks use greater attentional and linguistic demands of the speaker compared with other tasks such as reading or imitation, which can exacerbate hypophonic speech in PD. For example, Ho, Iansek, and Bradshaw (2002) demonstrated that in hypophonic participants with PD, a reduction in speech intensity was exacerbated during a concurrent task that involved a tracing task combined with a spontaneous conversation task. The importance of considering the speech task in the interpretation and understanding of hypophonia in hypokinetic dysarthria is an important consideration given the apparent speech task effect.

The Lombard Effect

When examining the effect of hypophonia in PD, the introduction background noise during conversational tasks is a particularly relevant context in which to study this speech symptom because the impact of hypophonia often is exacerbated in this naturalistic context. Interestingly, with the exception of a few studies, the effect of background noise on speech intensity in hypophonic speakers has not been examined or documented systematically in the research literature. To understand the relationship between speech intensity and background noise both in normal speakers and in hypophonic individuals with PD, the Lombard effect is particularly relevant to this discussion. In 1911, Etienne Lombard described a phenomenon in which an individual, when engaged in conversation and when presented with background noise, unconsciously increased his vocal intensity. Conversely, when the background noise was stopped, the individual's

vocal intensity decreased (Lane & Tranel, 1971). This phenomenon has been termed the "Lombard sign," the "Lombard effect," or the "Lombard reflex" (Lane & Tranel, 1971). It is generally agreed upon in the literature that the Lombard effect is mediated by the communicative intent of the message, that is, the function of the Lombard effect can be interpreted as a listener-centered phenomenon (Amazi & Garber, 1982; Lane & Tranel, 1971). The purpose, therefore, of the Lombard effect is to ensure accurate message delivery to the listener with increasing levels of background noise. In 1971, Lane and Tranel suggested that the magnitude of response of the Lombard sign is governed by the premium on intelligible communication. In other words, speakers increase their speech intensity in order to communicate more effectively with a listener during increasing levels of noise. Amazi and Garber (1982) demonstrated a similar finding in that the communicative task differentially affected the magnitude of response of the Lombard effect. For example, these researchers found that adults increased their speech intensity more during a storytelling task than during a word-labeling task. Amazi and Garber concluded that their results supported Lane and Tranel's finding that the Lombard sign is determined by the speaker's unconscious need to communicate effectively intelligible messages to the listener. The Lombard sign also has been described in the PD literature but with inconsistent results (Adams, Haralabous, Dykstra, Abrams, & Jog, 2005; Adams & Lang, 1992; Ho, Bradshaw, & Iansek, 2000; Ho, Bradshaw, Iansek, & Alfredson, 1999; Moon, 2005). Studying the robustness or the magnitude of the Lombard sign in PD may provide important information regarding the nature of hypophonic speech in PD. Ho and colleagues (1999) failed to demonstrate the Lombard effect in 12 individuals with hypophonia and PD. During presentation of pink noise at 10 to 30 dB sensation level (SL) above threshold, these researchers demonstrated a minimal increase in the speech intensity of the individuals they studied. Conversely, Adams and Lang (1992) established the Lombard effect in a group of 10 individuals with hypophonia and PD. These researchers demonstrated a marked increase in the speech intensity of these hypophonic individuals when 90 dB SPL of white noise was presented. An additional and more recent study also suggests that the Lombard effect can be elicited in PD. Adams et al. (2005) found that multi-talker noise, presented at 50 to 70 dB SPL in 5-dB increments, produced a

significant increase in the speech intensity of memorized sentences in 10 individuals with hypophonia and PD. When interpreting these incongruent results, it is important to consider a number of factors that may affect the robustness of the Lombard effect. These factors include the type of speech task (i.e., conversation vs. reading vs. memorized sentences), the type of background noise (i.e., white vs. pink vs. multi-talker noise), the intensity in which the background noise is presented, how intensity is measured, the method of stimulus presentation (i.e., free-field background noise vs. presentation via headphones), and the severity of hypophonia. For example, Ho et al. (1999) used pink noise as the background noise in their study. A potential limitation of using pink noise over multi-talker noise is that it may not represent a naturalistic type of background noise. Presentation of multi-talker noise, on the other hand, can simulate a more naturalistic noise that can be encountered when speakers have to deal with the background noise of competing speakers such as when speaking in a noisy restaurant. In relation to the level of background noise presented, the Ho et al. (1999) study could have indirectly demonstrated that a certain threshold of background noise is required to elicit the Lombard effect. In this study, background noise was presented to participants at +10 and +25 dB SL (referenced to each individuals threshold) during the conversational task and between +10 and +30 dB SL in 5-dB increments for the reading task. These low levels of background noise may not have been intense enough to elicit the Lombard effect in the group of individuals with PD they studied. Lane and Tranel (1971) discussed the effect of choosing very low but also very high levels of background noises and cited floor and ceiling effects. Lane and Tranel explained that one's speech intensity cannot continue to fall indefinitely at the rate dictated by the noise-competition function (1971, p. 683). Conversely, when one is driven to the limits of his or her range of response, the result can be an overall flattening of the function or a ceiling effect (Lane & Tranel, 1971). In both the Adams and Lang (1992) and Adams et al. (2005) studies, background noise was presented to participants between 50 and 90 dB SPL. These ranges of background noise levels may have been sufficient to elicit the Lombard effect without producing floor or ceiling effects. Additionally, intensities within a range of 50 to 90 dB SPL are likely comparable to what an individual may encounter in everyday communicative situations.

The Ho et al. (1999) and Adams and Lang (1992) studies also can be critiqued on the mode of stimulus presentation. Although free-field background noise was used in the Adams et al. (2005) study, presentation of background noise was delivered via headphones in the aforementioned studies. A potential risk of using headphones to deliver background noise is that headphone presentation most likely does not represent how background noise is naturally encountered. Presentation of free-field background noise likely simulates a more realistic and naturalistic experience for the listener and the speaker.

It has been suggested that individuals with hypophonia associated with PD can have difficulty identifying accurately the presence of hypophonia in their own speech because of sensory-perceptual deficits (Ho et al., 2000). These sensory-perceptual deficits often manifest as difficulties in sensory self-perception of effort or in the scaling of motor output and effort (Lewis & Byblow, 2002). It also has been suggested that individuals with PD may have auditory-motor integration deficits (Adams & Dykstra, 2008). These auditory-motor integration deficits can include deficits in the temporal discrimination of auditory stimuli (Artieda, Pastor, & Lacruz, 1992) as well as hyperactivity of the stapedial reflex (Murofushi, Yamane, & Osanai, 1992). The basal ganglia are thought to be responsible for the gating of sensory input for motor control (Kaji, 2001). Individuals with PD often demonstrate hypokinesia in a variety of complex motor movements. *Hypokinesia* refers to a reduction in movement amplitude such as small handwriting (micrographia) or the shuffling gait characteristic of many individuals with PD (Berardelli, Rothwell, Thompson, & Hallet, 2001). Hypophonia may be an analogous correlate to the hypometric movements observed in the limbs. Numerous researchers have suggested that individuals with PD can produce increased speech intensity in response to increasing interlocutor distance and background noise levels (i.e., Lombard effect), but the intensity is consistently lower by 2 to 4 dB SPL than in healthy control participants (Adams et al., 2005; Adams & Dykstra, 2008; Fox & Ramig, 1997; Ho et al., 2001). This suggests that individuals with PD can produce a normal pattern of intensity regulation but with an "overall gain reduction" for intensity (Adams & Dykstra, 2008). In addition to an overall reduction in the scaling of loudness, individuals with PD often have sensory anomalies

in that they have difficulty correctly perceiving their own speech intensity. In a study that examined speech intensity and self-perception of loudness in PD and in healthy control participants, Ho, Bradshaw, and Iansek (2000) found that the participants with PD perceived their speech intensity to be louder than the healthy control participants despite having quieter speech intensity than the control participants. Interestingly, when individuals with PD are given external cues such as visual or auditory cues or explicit instructions, they are able to normalize movement patterns (Ho et al., 1999). This suggests that providing appropriate cues to individuals with PD may have beneficial effects on the correct scaling and perception of motor movements (including speech intensity).

The purpose of this study was to examine the effect of background noise on the speech intensity of individuals with hypophonia associated with PD.

METHODS

Participants

This study included 30 participants ($n = 30$) with hypophonia as a result of mild to severe idiopathic PD. In total, there were 21 men and nine women (age range, 43–77 years; mean age, 63.26 years) tested with an average PD onset of 8.6 years (range, 2–26 years). Participants with PD were reported by a neurologist (M. Jog) to demonstrate reduced speech intensity or hypophonia. All participants with PD were stabilized on their antiparkinsonian medication and were tested at approximately 1 hour after taking their regularly scheduled antiparkinsonian medication. Table 1 provides a description of the participants with PD. The 15 control participants included eight women and seven men (age range, 56–77 years; mean age, 69.4 years)). Participants with PD and control participants were required to pass a 40 dB HL bilateral hearing screening at 500, 1000, and 2000 hertz. All participants had language and cognition skills that were sufficient to support conversation.

Procedures

Speech Intensity

All participants were tested in an audiometric booth. A loudspeaker was placed at 150 cm and 45 degrees to the left of and in front of each

TABLE 1. Description of Participants with Parkinson's Disease

Subject	Age (years)	Sex	PD Duration (years)	Occupation	Antiparkinson Medications
PD1	55	M	6	Retired engineer	Amantadine
PD2	54	F	10	Retired nurse	Mirapex, Sinemet
PD3	66	M	5	Retired engineer	Sinemet, Permex
PD4	66	M	8	Retired nurse	Sinemet
PD5	55	M	6	Factory worker	Simemet, Permax
PD6	55	M	8	Farmer	Sinemet, Permax
PD7	67	M	12	Retired factory worker	Sinemet, Mirapex
PD8	66	F	2	Retired secretary	Sinemet, Propranolol
PD9	64	F	6	Retired insurance agent	Sinemet, Permax, Mirapex
PD10	72	M	17	Retired crane operator	Sinemet
PD11	75	M	8	Retired counselor	Sinemet
PD12	68	M	3	Contractor	Sinemet
PD13	64	F	15	Retired switchboard supervisor	Sinemet
PD14	55	M	10	Sales	Sinemet
PD15	55	M	3	Retired chartered accountant	Sinemet, Requip
PD16	61	M	5	Citizenship judge	Amantadine, Mirapex
PD17	63	F	6	Retired secretary	Sinemet
PD18	53	M	6	Retired accountant	Sinemet, Requip
PD19	60	F	10	Retired jewelry store manager	Sinemet
PD20	68	M	10	Retired brick layer	Sinemet, Requip
PD21	77	M	5	Retired controller	Sinemet
PD22	61	F	11	Retired store manager	Sinemet, Permax
PD23	74	M	7	Retired school bus driver	Sinemet
PD24	70	F	11	Retired hairdresser	Sinemet
PD25	63	M	6	Physician	Lamictal, lithium, propranolol
PD26	43	M	2	Auto assembly worker	Sinemet, Mirapex
PD27	61	M	10	Retired school superintendent	Sinemet
PD28	69	M	26	Farmer	Sinemet, domperidone, propranolol
PD29	66	M	18	Priest	Sinemet, amantadine, Requip
PD30	72	F	6	Retired teacher	Sinemet

F = female; M = male; PD = Parkinson's disease.

participant. A free-field microphone (Shure SM48) attached to a floor stand was placed at 150 cm and 45 degrees to the right of and in front of each participant. Thus, the participant, floor microphone, and loudspeaker were positioned at three corners of a triangle (150 cm per side). Participants wore a headset microphone (AKG-C420) positioned at a constant 6 cm distance from the mouth. The experimenter sat 150 cm in front of the participant. The loudspeaker presented free-field multi-talker noise (Audiotech four talker noise). The intensity of the noise was adjusted via a diagnostic audiometer (GSI 10) that had been calibrated to reflect intensity in dB SPL. The speech of each participant was recorded using a dual-channel digital audio tape recorder (Tascam DA-01).

Maximum Intensity. Maximum intensity was determined by asking each participant to produce the phrase "I owe you a yo-yo, I owe you a yo-yo" (Goldinger, Pisoni, & Luce, 1996) at their maximum loudness level. Maximum intensity was calculated by determining and analyzing the loudest production of this phrase over a series of three trials. The loudest production of this phrase was analyzed using the Visipitch program. The utterance was displayed on a computer screen and isolated with user-controlled cursors. Once isolated, the Visipitch program calculated the average intensity of the utterance in dB SPL. The sentence "I owe you a yo-yo" was chosen because it is completely voiced and therefore it provides a relatively steady and continuous speech intensity contour from which to make accurate average speech intensity measures.

Habitual Intensity. The experimenter engaged all participants in approximately 2 minutes of conversation without introduced background noise at the beginning of the conversational protocol. For the 2-minute segment of conversation without added background noise, six utterances of approximately 7 to 10 seconds each were extracted and analyzed separately and the mean intensity was calculated. Each 7 to 10 seconds of utterances was analyzed separately using the Kay Pentax Visipitch program (Model 43008). The utterances were displayed on a computer screen and isolated with user-controlled cursors. Once isolated, the Visipitch program calculated the average intensity of the utterance in dB SPL.

Conversational Intensity in Multi-talker Background Noise

Multi-talker background noise was presented to each participant during the experimental protocol. Participants were engaged in 2 minutes of conversation per intensity level. In total, there were five randomly presented intensity levels (50, 55, 60, 65, and 70 dB SPL) with the total conversation time approximately 10 minutes in length. Participants were not instructed explicitly to stay above the noise during the conversational speech task. During this conversational task, the participants were asked to discuss topics such as their interests, occupation, vacations, and such. For each of the 2 minutes of conversation per intensity level (i.e., 50, 55, 60, 65, and 70 dB SPL) in multi-talker noise, six utterances of approximately 7 to 10 seconds each were extracted and analyzed separately, and the mean intensity per intensity level was calculated. Each 7 to 10 seconds of utterances was analyzed separately using the Visipitch program. The utterances were displayed on a computer screen and isolated with user-controlled cursors. Once isolated, the Visipitch program calculated the average intensity of the utterance in dB SPL. The average utterance intensity values were used as the dependent measure in the statistical analyses.

RESULTS

Maximum Intensity Comparisons

This objective addressed maximum speech intensity levels during the repetition of a phrase across both experimental groups. This analysis examined the maximum speech intensity of participants with PD and control participants while repeating the phrase "I owe you a yo-yo, I owe you a yo-yo." To evaluate maximum speech intensity between the two participant groups, an independent samples *t*-test was conducted. More specifically, the following comparison was made: *Maximum intensity: PD versus control participants*. This analysis was conducted to answer the following research question: Do participants with PD have similar maximum intensity levels as control participants? Table 2 shows the mean and standard deviation (SD) values for maximum speech intensity levels of each group. This analysis revealed a significant difference between PD and control participants

TABLE 2. Mean Maximum Speech Intensity Levels of Control and Parkinson's Disease Participants

Control	Parkinson's Disease
87.07	76.59
(3.07)	(5.44)

Speech intensity levels are in dB SPL. Standard deviations appear in parentheses below means.

$(t (43) = 6.917; P = .001)$. Participants with PD are significantly less intense (by ~10 dB SPL) than control participants in their maximum speech intensity.

Habitual Conversational Speech Intensity Comparisons

An independent samples t-test evaluated conversational speech intensity in the no added background noise condition. The means and SDs for conversational speech intensity levels between participants with PD and control participants are presented in Table 3. This analysis revealed a significant difference between PD and control participants $(t (43) = 4.909; P = .001)$. Participants with PD showed habitual conversational intensity levels that are significantly less intense (by ~5 dB SPL) than those of control participants.

Conversational Speech Intensity Comparison in Various Intensity Levels of Background Noise

A two-factor repeated measures ANOVA (analysis of variance) with one between-group and one within-group factor was performed to compare the PD and control participants' intensity values during presentation of five intensity levels of background noise. "Group" was used as the between-group independent variable with two levels (PD, control), and "noise" was the

TABLE 3. Mean Habitual Conversational Speech Intensity Levels of Control and Parkinson's Disease Participants

Control	Parkinson's Disease
71.82	66.86
(2.51)	(3.48)

Speech intensity levels are in dB SPL. Standard deviations appear in parentheses below means.

within-group, repeated measures independent variable with five levels (noise: 50, 55, 60, 65, and 70 dB SPL). The "group" main effect was significant $[F(1, 43) = 33.944; P = .001]$. These results are illustrated in Figure 1 with associated means and SDs in Table 4. There also was a significant main effect for "noise," but Mauchly's test of the sphericity assumption $[W = .414; \chi^2(9) = 36.509; P = .001]$ was not met, so the Greenhouse-Geisser correction was applied $[F_{adj}(2.831, 43) = 126; P = .001]$. The significant main effect of "noise" is illustrated in Figure 1 with associated means and SDs in Table 4. There was no significant interaction between "group" (PD, control) and "noise" levels $[F(4,43) = 1.199; P = .313]$. These results demonstrate that participants with PD were significantly less intense (by ~5 dB SPL) than control participants in various intensity levels of background noise. However, the participants with PD demonstrated a similar but attenuated pattern of response to the various intensity levels of background noise. In other words, the participants with PD were quieter on average than control participants, but the participants with PD still demonstrated a similar Lombard effect with increasing intensity levels of background noise. An additional analysis involved calculating individual regression lines for each of the 30 participants with PD and 15 control participants. Regression lines were calculated for speech intensity versus

TABLE 4. Mean Conversational Speech Intensity Levels of Control and Parkinson's Disease Participants in Different Levels of Multi-talker Background Noise

Group	Multi-talker Background Noise Level				
	50 dB SPL	55 dB SPL	60 dB SPL	65 dB SPL	70 dB SPL
Control	72.33	73.51	73.80	75.21	76.72
	(2.71)	(2.69)	(2.53)	(2.51)	(2.25)
Parkinson's disease	67.37	68.11	68.79	69.57	71.19
	(3.25)	(3.28)	(2.82)	(3.28)	(3.25)

Speech intensity levels are in dB SPL. Standard deviations appear in parentheses below means.
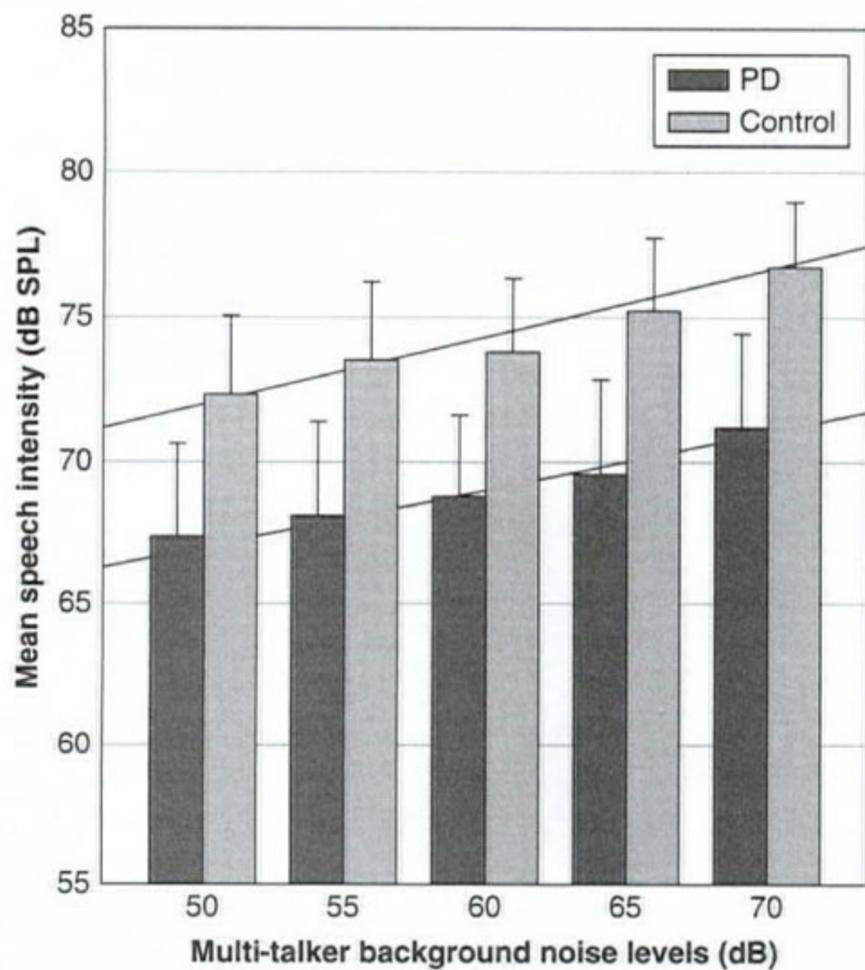
Figure 1. Mean conversational speech intensity levels in multi-talker background noise. PD = Parkinson's disease; SPL = sound pressure level.

all five intensity levels of multi-talker background noise for control participants (see Figure 2) and participants with PD (see Figure 3). Figure 2 depicts graphically individual slope values for the control participants across all five intensity levels
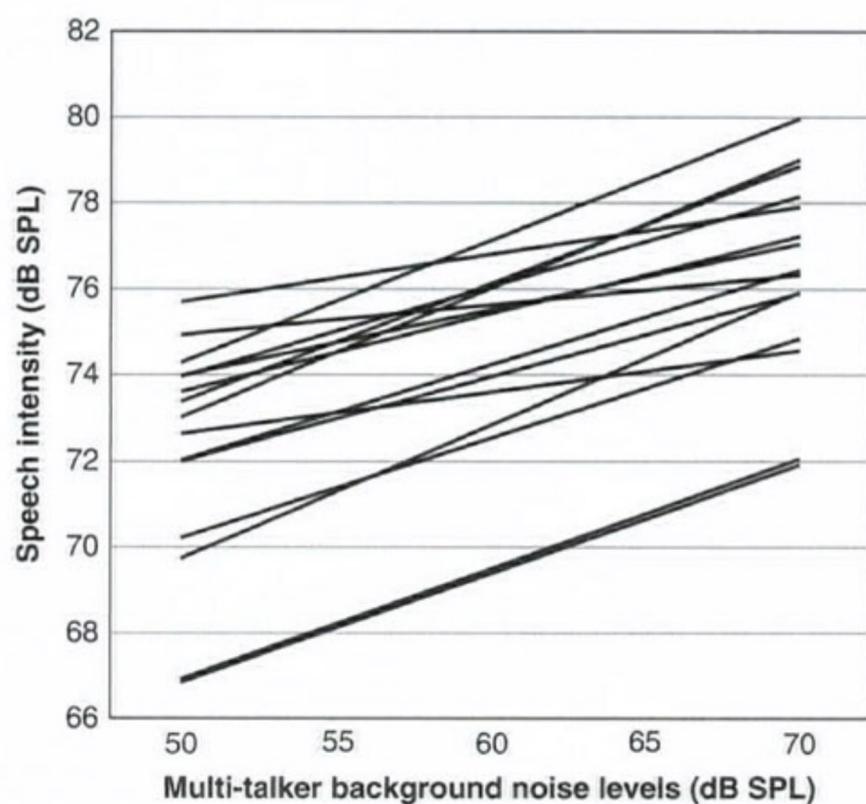

Figure 2. Individual speech intensity versus background noise regression lines for control participants. SPL = sound pressure level.
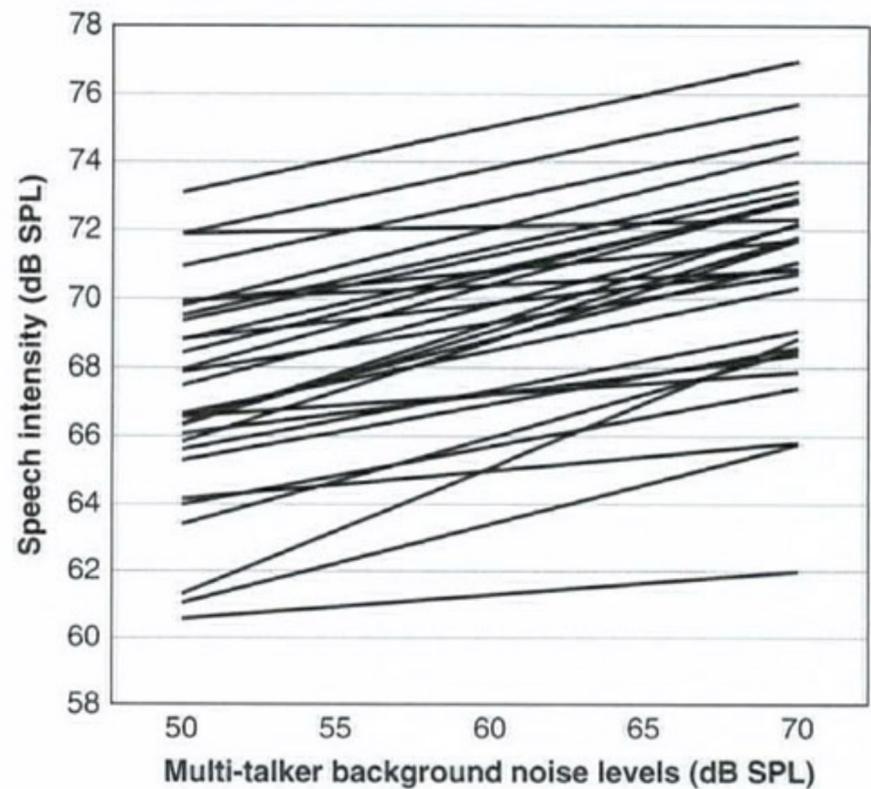

Figure 3. Individual speech intensity versus background noise regression lines for participants with Parkinson's disease. SPL = sound pressure level.

of multi-talker background noise. Upon examination of this figure, the positive and steep slopes suggest that each of the control participants demonstrated a normal and robust Lombard effect. The mean slope across all control participants was 0.21. For the participants with PD, Figure 3 demonstrates that the majority of the regression lines show a positive slope. Upon closer examination of this figure, it is of interest to note that some of the most severely hypophonic participants with PD have fairly strong positive slopes over increasing levels of background noise. The mean slope across all participants with PD was 0.18. This figure suggests that there is little to no relationship between the severity of an individual's hypophonia and the slope of his or her speech intensity versus noise regression line.

DISCUSSION

This study examined the effect of various intensities of background noise on the conversational speech intensity of individuals with hypophonia associated with PD. The first objective of this study addressed the maximum speech intensity of participants with PD and control participants while repeating the phrase "I owe you a yo-yo, I owe you a yo-yo" at their maximum loudness. Significant differences were found between groups on this task, revealing that the maximum

speech intensity of individuals with PD was approximately 10 dB SPL less than that of control participants (control participants: 87.07 dB SPL; SD 3.07 vs. PD participants: 76.59 dB SPL; SD = 5.44). Although the PD group had a larger SD than the control group, the PD group consistently produced maximum intensity levels that were less than those of the control participants.

In addition to maximum speech intensity, habitual conversational speech intensity was evaluated in both PD and control participants. Similar to the findings related to maximum speech intensity measures already presented, significant differences were also found between groups on this task. In general, the habitual conversational speech intensity values for the participants with PD was approximately 5 dB SPL less (66.86 dB SPL; SD = 3.48 vs. 71.82 dB SPL; SD = 2.51) than those of control participants.

Previous reports have suggested that individuals with hypophonia associated with PD can produce speech intensity levels comparable to those of healthy control participants (Canter, 1963; Kempler & Van Lancker, 2002; Metter & Hanson, 1986). However, growing acoustic and perceptual evidence suggests the contrary (Fox & Ramig, 1997; Ho et al., 1999; Illes et al., 1988; Moon, 2005). Fox and Ramig (1997) suggested that on average, individuals with PD have speech intensity levels 2 to 4 dB SPL lower than those of age-matched, healthy control participants.

In this examination of maximum speech intensity and habitual conversational speech intensity, the difference between PD and control participants was in the order of 10 dB SPL and 5 dB SPL, respectively. This very robust difference between groups may be capturing a subgroup of severely hypophonic individuals. Upon examination of the results for maximum intensity, there is a fairly large SD for the PD group and a large range of intensity values across participants with PD (minimum, 60.04 dB SPL to maximum, 84.87 dB SPL) and to a lesser extent, control participants (minimum, 79.55 dB SPL to maximum, 90.70 dB SPL). Habitual conversational intensity values also had a large range of intensity values, especially for the individuals with PD studied (PD group, 60.51–72.89 dB SPL; control participants 66.76–74.39 dB SPL). This large range of intensity values for the PD group could represent that there are distinct subgroups within hypophonic speakers. A growing literature suggests that there are specific subgroups of PD based on clinical

presentation. Given the variable clinical presentation of PD, the idea of subtypes of PD is not an implausible idea. Jankovic et al. (1990) classified PD into the following subtypes based on clinical presentation: tremor predominant, freezing predominant, akinetic-rigid, and "unclassified." Other subtype classifications of PD were suggested by Lewis, Foltynie, Blackwell, Robbins, Owen, and Barker (2005). This subtype classification included patients with a younger disease onset, tremor dominant, nontremor dominant with significant levels of cognitive impairment and mild depression, and rapid disease progression but with no cognitive impairment. The notion of subgroups was suggested in a study that investigated comprehension of prosody in PD. Lloyd (1999) found that only some individuals with PD showed impairment in the comprehension of prosody and lexical stress. Yahalom, Simon, Thorne, Peretz, and Giladi (2004) also investigated subgroups within PD. These researchers found that one of four subgroups (i.e., only the tremor-dominant subgroup) of individuals with PD had specific pacing disturbances in a study that investigated rhythmic movements of the hand. These researchers suggested that identifying specific PD subtypes may serve to illuminate the underlying physiological mechanisms of the disease as well as differential treatment responses. Lewis et al. (2005) suggested that delineating subgroups of PD may be a useful predictor for the management of PD as well as in the creation of novel experimental therapies. This is an interesting theory to consider given the differential patterns of response observed in our investigation of maximum intensity and habitual conversational speech intensity. Beyond the subtypes classified (i.e., tremor dominant, akinetic-rigid, and so on), it would be of interest to determine if there is heterogeneity within a population demonstrating a specific impairment. For example, all participants with PD in the current study were assessed to be hypophonic speakers, but there was a wide pattern of response across hypophonic speakers. That is, during the maximum speech intensity task, one subgroup could generate increased speech intensity, but the other subgroup had more difficulty or could not increase speech intensity. The latter group may have demonstrated difficulty possibly because of physiological impairments caused by the disease process (i.e., bowed vocal folds, decreased breath support) or decreased self-monitoring of appropriate loudness (i.e., disease-related sensorimotor integration

deficits). Future studies could include the investigation of whether the differential patterns of response within a specific speech impairment (i.e., hypophonia, dysfluency) could be attributed to overall disease severity or the idea of subgroups within a specific impairment. This information could provide potentially valuable information not only our understanding of the nature of PD but could also help refine and tailor treatment approaches based on one's pattern of response. For example, for individuals with PD who can increase their speech intensity based on an external cue, a voice recalibration treatment program such as the Lee Silverman Voice Treatment (LSVT) program (Ramig, Countryman, Thompson, & Horii, 1995) or use of a portable masking device such as the Edinburgh Masker (Dewar, Dewar, & Barnes, 1976) could be the most clinically beneficial treatments; however, in those individuals who do not respond well to external cueing, assistive devices such as a voice amplifier may be a more viable option for treatment success. Overall, the significant and robust findings in the current study demonstrate acoustically that the participants with hypophonia associated with PD, in general, have significantly lower maximum speech intensity and habitual conversational speech intensity levels than the control participants.

Conversational speech intensity of both participant groups was evaluated in a range of intensity levels of multi-talker background noise. Of particular interest was to determine if participants with hypophonia secondary to PD demonstrated a normal Lombard function during presentation of five intensities of multi-talker background noise. Significant differences were demonstrated between experimental groups, suggesting that the participants with PD were less intense than control participants across all levels of multi-talker background noise. Generally, participants with PD were approximately 5 dB SPL less intense than control participants across all five levels of multi-talker background noise. The mean difference between the least intense background noise condition (50 dB SPL) and the most intense background noise condition (70 dB SPL) was 4.39 dB for the control group and 3.89 dB for the group with PD. These results suggest that both groups were responding to background noise in a similar way, but the speech intensity of the participants with PD was always less intense than that of the control participants.

Group slope values also suggest a similar pattern of response to increasing intensities of multi-talker background noise. Whereas the mean slope value for control participants was 0.21, the mean slope value for the participants with PD was 0.18. In general, these positive slope values suggest a normal but slightly flatter than would be expected Lombard function across both groups. Lane and Tranel (1971) suggested that a normal Lombard sign has a slope or a noise-compensation function of approximately 0.50. This means that a fourfold increase in noise should produce a twofold increase in speech intensity (Lane & Tranel, 1971). Lane and Tranel (1971), however, described several other studies that produced flatter noise-compensation functions, typically in the order of 0.10 to 0.20. These studies, however, evaluated the Lombard function during reading tasks. It is possible that the task (i.e., reading vs. memorized sentences vs. conversation), noise condition (i.e., multi-talker vs. pink vs. white noise), mode of noise presentation (i.e., headphone vs. free field), age of the speaker, interlocutor distance, or age-related hearing loss could differentially impact one's noise-compensation function or Lombard function in response to increasing intensity levels of background noise.

Studying the robustness or the magnitude of the Lombard sign in individuals with PD has generated some important findings regarding the nature of hypophonia in PD. There is a small but growing body of research suggesting that individuals with PD can produce increased speech intensity in response to increasing background noise levels (i.e., Lombard effect) but that their speech intensity is consistently lower than that of control participants. On average, these studies have suggested that the speech intensity of individuals with hypophonia associated with PD is 2 to 4 dB lower than that of control participants (Adams et al., 2005; Adams & Dykstra, 2008; Ho et al., 2001). This suggests that individuals with PD may have a normal pattern of intensity regulation but with an "overall gain reduction" for intensity (Adams & Dykstra, 2008). Interestingly, the maximum speech intensity levels of the participants with PD (76.59 dB SPL) was almost the same as the speech intensity used by the control group (76.72 dB SPL) in the 70-dB SPL background noise condition. This result suggests that the participants with PD have the capability of achieving the same speech intensity levels as the control participants in the most intense

background noise conditions, but the participants with PD consistently underregulate their speech intensity. In general, the results of this study are congruent with this growing empirical literature. This study found that the participants with PD demonstrated a similar but attenuated pattern of response to the various intensity levels of background noise as the control participants. In other words, the participants with hypophonia associated with PD were less intense than the control participants across all background noise conditions, but they still demonstrated a normal Lombard effect when speaking in increasing levels of multi-talker background noise. The current results are consistent with those of Moon (2005), who also found that PD participants demonstrated a relatively normal Lombard function but consistently underregulated speech intensity across a wide range of background noise intensity levels. It is possible that hypophonia may be a sensoriperceptual deficit in the regulation of speech intensity relative to background noise conditions. These sensoriperceptual deficits often manifest as difficulties in sensory self-perception of effort or in the scaling of motor output and effort (Lewis & Byblow, 2002). Kaji (2001) suggested that the basal ganglia may be responsible for the gating of sensory input for motor control. Therefore, hypophonia may represent a deficit in motor output and effort scaling. Hypophonic speech potentially could be considered an analogue to the hypometric movements observed in the limbs such as the characteristic shuffling gate, reduced arm swing, or micrographic handwriting in individuals with PD. Results of the current study suggest that a potentially important and fundamental aspect of hypophonia associated with PD may be the lack of ability to accurately regulate speech intensity relative to background noise conditions.

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