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Loudness perception and speech intensity control in Parkinson's disease

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ABSTRACT

The aim of this study was to examine loudness perception in individuals with hypophonia and Parkinson's disease. The participants included 17 individuals with hypophonia related to Parkinson's disease (PD) and 25 age-equivalent controls. The three loudness perception tasks included a magnitude estimation procedure involving a sentence spoken at 60, 65, 70, 75 and 80 dB SPL, an imitation task involving a sentence spoken at 60, 65, 70, 75 and 80 dB SPL, and a magnitude production procedure involving the production of a sentence at five different loudness levels (habitual, two and four times louder and two and four times quieter). The participants with PD produced a significantly different pattern and used a more restricted range than the controls in their perception of speech loudness, imitation of speech intensity, and self-generated estimates of speech loudness. The results support a speech loudness perception deficit in PD involving an abnormal perception of externally generated and self-generated speech intensity.

Learning outcomes: Readers will recognize that individuals with hypophonia related to Parkinson's disease may demonstrate a speech loudness perception deficit involving the abnormal perception of externally generated and self-generated speech intensity.

1. Introduction

Hypophonia or low speech intensity is one of the most common speech deficits associated with Parkinson's disease (PD) (Adams & Dykstra, 2009; Duffy, 2013). It has been suggested that a deficit in the perception of speech loudness may play a causal role in the hypophonia of PD (Ho, Bradshaw, & Iansek, 2000; Kwan & Whitehill, 2011). Ho et al. (2000) proposed that in PD there is an abnormal integration of the sensation of one's own loudness during the motor output of speech intensity. Thus, the hypophonia of PD is hypothesized to be the result of a sensorimotor integration disorder. A sensorimotor integration disorder has been proposed for other motor control deficits in PD such as bradykinesia and hypokinesia

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(Abbruzzese & Berardelli, 2003; Schneider, Diamond, & Markham, 1986; Tatton, Eastman, Bedingham, Verrier, & Bruce, 1984). Sensorimotor integration relates to the use of sensory information to guide movements. This implies that a sensorimotor integration deficit is a movement-dependent sensory deficit that is most apparent during ongoing movements. Much of the supporting evidence for a sensorimotor integration deficit in PD comes from studies that examine the effect of sensory distortions or perturbations on movements. Examples of this evidence includes, impaired head movements in response to a continuously changing sensory target (Schneider et al., 1986), impaired compensation for mechanical perturbations during walking. (Jacobs & Horak, 2006), impaired reaching, grasping, and walking when vision is blocked and proprioception must be used (i.e. internal feedback) to guide movements (Konczak et al., 2009), impaired corrective responses to perturbations of reach and grasp movements (Lukos, Snider, Hernandez, Tunik, Hillyard, & Poizner, 2013), abnormal vocal responses to perturbations of vocal pitch (Liu, Wang, Metman, & Larson, 2012; Chen et al., 2013), and abnormal vowel productions in response to perturbations of vowel formants in individuals with PD (Mollaei, Shiller, & Gracco, 2013). In its simplest form, a sensorimotor integration deficit could exist in the absence of "pure" sensory or somatosensory impairments. However, PD is complicated by the potential to develop both sensory and sensorimotor integration deficits. There is extensive evidence of a wide range of sensory and somatosensory impairments in PD (Conte, Khan, Defazio, Rothwell, & Berardelli, 2013). In a recent review, Conte et al. (2013) presented evidence from over 40 studies that have demonstrated a sensory or somatosensory impairment in individuals with PD. Examples of these sensory impairments include abnormalities in two-point tactile discrimination, spatial tactile acuity, tactile stimulus location, tactile discrimination of object shapes, pain perception, olfaction, thermal sensations, perception of weight, hepatic sensitivity, sense of limb position, and the perception of passive limb velocity (Conte et al., 2013). Some of these sensory and somatosensory deficits have been hypothesized to play a causal role in the motor control deficits of PD. Thus, motor control deficits (including hypophonia) in PD have been hypothesized to be causally linked to sensory, somatosensory and sensorimotor integration deficits.

Evidence for a loudness perception deficit in PD has been limited to brief anecdotal reports by Fox and Ramig (1997) and two previous studies (Dromey & Adams, 2000; Ho et al., 2000). In the study by Ho et al. (2000), participants with PD spoke at different intensity levels (soft, normal, loud) and then listened to these productions while adjusting a volume control knob in an attempt to match the loudness level of the audio playback to their previous spoken loudness level. Ho et al. (2000) found that the participants with PD significantly over-estimated the loudness of their speech relative to controls. In contrast, a study by Dromey and Adams (2000) failed to find a significant difference between PD and control participants on three loudness perception tasks involving loudness estimates of tones and speech stimuli and a magnitude production task involving vowels at different loudness levels. The inconsistencies between these two preliminary studies indicate the need for additional investigations of loudness perception in PD.

Although loudness perception can be measured with a variety of tasks and procedures, magnitude estimation and magnitude production procedures have been used in several previous systematic studies to examine speech loudness perception related to externally generated speech stimuli (extraphonic loudness) and self-generated speech stimuli (autophonic loudness) in non-neurologically impaired individuals (Fletcher & Munson, 1933; Lane, Catania, & Stevens, 1961; Stevens, 1960; Warren, 1973). Magnitude estimation (ME) and magnitude production (MP) procedures are considered to have several advantages over other psychophysical or loudness perception procedures. Unlike other scaling procedures (i.e. equal appearing interval scaling), ME and MP are considered to involve "direct" numerical estimates of the perceived magnitude of stimulus intensities (i.e. loudness) (Stevens, 1960). In contrast to threshold testing procedures, these procedures typically involve measures across a wide range of stimulus intensities and this allows the experimenter to examine different components of the stimulus continuum and to generate overall psychophysical magnitude functions (i.e. slope or power function) (Stevens, 1960). These procedures are not bound by fixed minimum and maximum values and therefore are not susceptible to floor or ceiling effects that can occur in other testing procedures. ME and MP procedures require minimal training because they are fairly easy for participants to understand and perform. In addition, a participant's psychophysical function can be obtained fairly rapidly with ME and MP procedures. Unlike other rating scale procedures, these procedures provide ratio-based data, instead of interval or ordinal data, and this allows for the use of standard parametric statistical analysis procedures in psychophysical experiments (Gescheider, 1985). In addition, ME and MP procedures are often used in combination in order to validate each other and to examine both the perception and production mechanisms involved in a specific psychophysical process (i.e. loudness) (Gescheider, 1985). From this perspective, ME and MP procedures seem well suited to the evaluation of sensory and sensorimotor integration functions in disordered populations.

Imitation procedures have been used extensively to examine a variety of perceptual-motor processes (Adank, Rueschemeyer, & Bekkering, 2013; Brass & Heyes, 2005; Lane, Tranel, & Sisson, 1970). Unfortunately, imitation procedures have received limited attention in previous psychophysical studies of loudness perception (Adams, Moon, Dykstra, Abrams, Jenkins & Jog, 2006; Möbes, Joppich, Stiebritz, Dengler, & Schröder, 2008). An advantage of the imitation procedure is that it allows for precise and systematic control of the stimulus targets during a stimulus-dependent motor task and therefore may provide useful and fairly detailed information about sensorimotor integration processes related to loudness perception and production.

The purpose of the present study was to examine loudness perception in individuals with Parkinson's disease to determine whether sensory and sensorimotor integration deficits may play a role in their hypophonia disorder. Loudness perception was examined using a combination of magnitude estimation, magnitude production and imitation tasks.

Table 1
Description of participants with Parkinson's disease.

Participant	Age	Gender	Time since diagnosis (years)	Levodopa daily dosage (mg/day)
PD 1	71	Male	4	500
PD 2	73	Male	17	850
PD 3	67	Male	13	800
PD 4	60	Male	5	400
PD 5	62	Male	4	600
PD 6	69	Male	15	900
PD 7	72	Male	3	400
PD 8	76	Male	1	400
PD 9	56	Male	2	0
PD 10	74	Male	17	900
PD 11	75	Male	1	400
PD 12	78	Male	4	600
PD 13	67	Male	2	600
PD 14	57	Male	1	0
PD 15	58	Female	10	800
PD 16	73	Female	7	600
PD 17	73	Male	15	600

2. Methods

2.1. Participants

This study included 17 individuals with hypophonia and Parkinson's disease (aged 56–78 years; mean = 68.3; male = 15; female = 2) and 25 age-equivalent control participants (aged 58–83 years; mean = 71.8; male = 11; female = 14). The average number of years since diagnosis of PD was 7.1 (range 1–17 years). Participants with PD were tested approximately 1 h after their regularly scheduled anti-Parkinson medication. Two of the participants with PD were not on anti-Parkinson medications while all other participants were on levodopa–carbidopa medication at daily levodopa equivalent dosages ranging from 400 to 1000 mg/day (mean = 550.0 mg/day). Participants had not received prior treatment for hypophonia. The participants had no prior history of speech, language, or hearing problems (except those related to Parkinson's disease). The Mini Mental State Examination (MMSE) was used to exclude participants with dementia (cutoff score $\geq$ 26/30). All participants passed a bilateral 30 dB HL hearing screening at 500, 1000, 2000 and 4000 Hz. This study was approved by the Health Sciences Research Ethics Board at Western University, London, Ontario, Canada. See Table 1 for PD participant demographic information.

2.2. Apparatus

Participants were seated in an audiometric booth (Industrial Acoustic Company) throughout testing. A headset microphone (AKG C420) was used to record each participant's speech. The microphone was located 6 cm from the midline of the participant's mouth. Calibration of the microphone was established through the use of a sound level meter that was placed 15 cm (6 in.) from the participant's mouth while the participant produced 1–2 s of a prolonged 'ah' at 70 dBA SPL as indicated on the sound level meter. The microphone was attached to a preamplifier (M-Audio preamp USB) and a desktop computer running Praat software (Boersma & Weenink, 2011). The recording module in the Praat software was used to digitize the speech samples at 44.1 kHz and 16 bits. For the presentation of the auditory speech stimuli, a loudspeaker was positioned at 120 cm in front of the participant. The loudspeaker was connected to an audio amplifier that received the calibrated speech stimuli from the audio output of a laptop computer that played the prerecorded audio (.wav) files. The intensity of the audio stimuli was calibrated using a 70 dBA SPL continuous pink noise reference signal that was adjusted and verified using a sound level meter positioned just above the participant's head. For the measurement of speech intensity, the recorded speech audio files were measured off-line using the acoustic intensity measurement module in the Praat program (Boersma & Weenink, 2011).

2.3. Speech stimuli

The current study used a five word sentence, "The puppies chased the ball", from a standard test of intelligibility (Assessment of Intelligibility in Dysarthric Speech; AIDS) (Yorkston & Beukelman, 1981) for all experimental conditions. This sentence was selected for the neutral tone, ease of pronunciation, and diversity of vowels and consonants. Using the same sentence for all the speech tasks helped to reduce the participant's cognitive load and allowed the participant to focus on speech loudness rather than the content of the sentence. The sentence was spoken by a 57-year-old healthy male while being recorded via an AKG C420 headset microphone attached to a preamplifier (M-Audio preamp USB) and a desktop computer

running Praat software (Boersma & Weenink, 2011). The recording module in the Praat software was used to digitize the speech sample at a sampling rate of 44.1 kHz and quantization level of 16 bits. The resulting audio file was analyzed via the intensity analysis module in Praat software to determine the average speech intensity of the sentence. Once the speech intensity of the sentence audio file was determined, this intensity value served as the 70 dB reference sample and was subsequently used to create the other experimental speech stimuli (60, 65, 75, 80 dB SPL). In order to create these additional experimental stimuli, another computer program (GoldWave: http://www.goldwave.com) was used to amplify or attenuate the reference speech audio file and create the new and appropriate experimental stimulus files (i.e. 60, 65, 75, 80 dBA SPL sentence audio files).

2.4. Procedures

The three loudness perception tasks were presented in the following order: magnitude estimation task, imitation task and magnitude production task. This particular order was selected in order to reduce the potential effect of speech-related fatigue on the magnitude estimation task. There was also a concern that the speech production and imitation tasks may influence or bias the magnitude estimation of loudness task. Each task involved 20 trials and the levels were randomized within each task.

2.4.1. Magnitude estimation task

The test sentence was randomly presented at five intensity levels (60, 65, 70, 75, and 80 dBA SPL). A duplicate of the 70 dB test sentence was assigned a loudness value of 100 and served as a reference or modulus and was presented after every three trials. For each experimental trial a test sentence was presented at a given intensity and the participants were instructed to provide a number that was proportional to the perceived magnitude of loudness.

2.4.2. Imitation task

The test sentence was randomly presented at five intensity levels (60, 65, 70, 75, and 80 dB SPL) and the participant attempted to repeat the sentence at the same intensity level.

2.4.3. Magnitude production task

Participants read aloud the test sentence at a normal intensity and this was assigned a "loudness" value of 100. The participant was then asked to produce the sentence at loudness levels that were judged to be proportional to the numbers 25, 50, 100, 200, and 400. For example, the number 200 would reflect a loudness level that was judged to be two times louder than the "normal" loudness level of 100.

2.5. Statistical analyses

A Shapiro–Wilk test of normality was performed on each of the dependent variables and was found to be not significant ($p > .05$) in every case. This suggests that normality of the data is a reasonable assumption. Based on this assumption, parametric statistical procedures were used for all of the statistical analyses. The magnitude estimation rating scores and the speech intensity values obtained from the three tasks were submitted to 3 separate, two-way, repeated measures, ANOVAs ($p < .05$).

3. Results

3.1. Magnitude estimation Task

Results of the magnitude estimation task are summarized in Table 2 and Fig. 1. The main effect of group was not significant [$F(1,38) = .267$, $p = .609$]. The main effect of stimulus intensity was significant [$F(4,152) = 149.80$, $p < .001$]. In addition, the group by stimulus intensity interaction was significant [$F(4,152) = 2.43$, $p = .049$]. A closer inspection of these results (see Fig. 1) indicates that the participants with PD had higher loudness ratings than the control participants during the less intense stimuli (60 and 65 dB) and lower loudness ratings during the more intense stimuli (75 and 80 dB). This pattern suggests that the participants with PD had a flatter psychophysical loudness function and a more restricted range of loudness ratings than the controls.

Table 2
Descriptive statistics for the magnitude estimation task.

	Intensity level (mean and SD)				
	60 dB	65 dB	70 dB	75 dB	80 dB
PD	51.76 (22.11)	71.65 (17.59)	101.38 (37.07)	130.74 (23.98)	195.44 (65.10)
Control	44.35 (10.34)	59.35 (13.94)	99.13 (19.75)	155.87 (35.26)	211.09 (67.40)

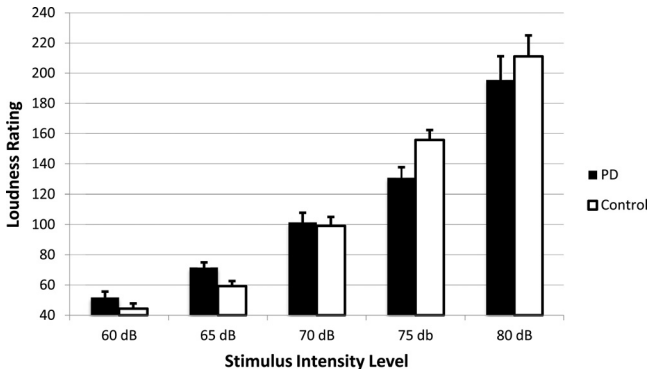

Fig. 1. Average loudness ratings provided by the PD and control participants at 5 stimulus intensity levels for the magnitude estimation task. Error bars indicate the standard error.

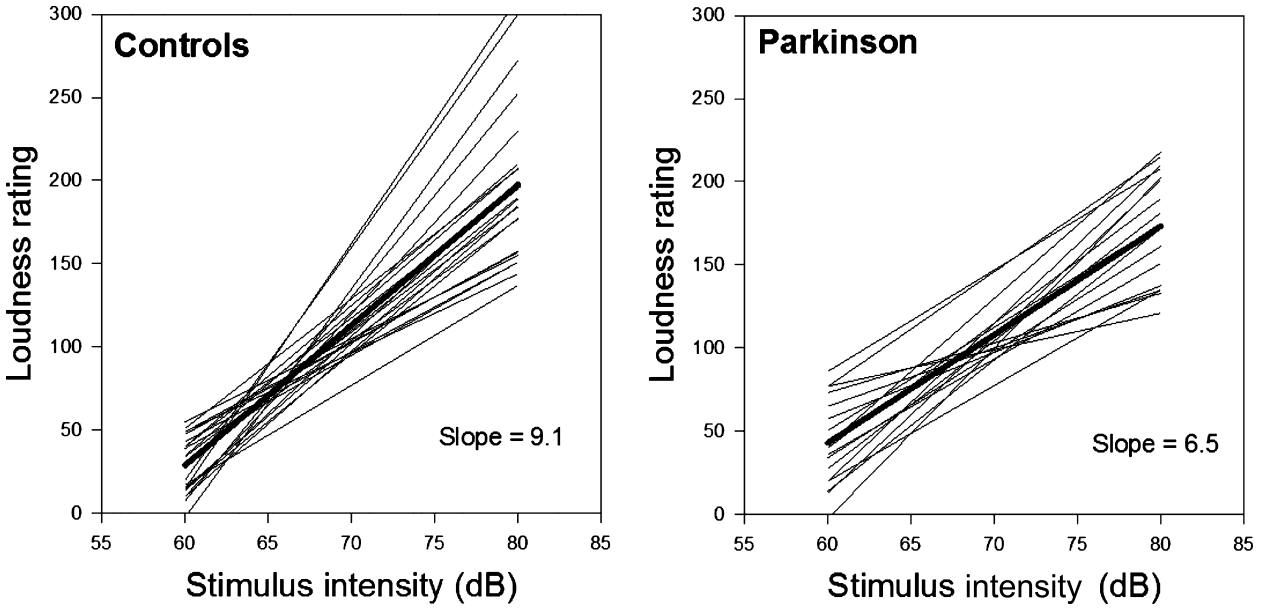

Fig. 2. Average and individual slope values related the magnitude estimation loudness ratings at 5 stimulus intensity levels for the PD and control participants. The group average is shown by the darker line.

To illustrate the psychophysical loudness function for the magnitude estimation task in greater detail, a regression analysis was performed on each participant's magnitude estimation data and the resulting slope values were submitted to an independent samples *t*-test. The PD group had a significantly ($t(40) = 5.68$, $p = .015$) lower slope value ($M = 6.5$, $SD = 2.3$) than the control group ($M = 9.1$, $SD = 3.0$). Fig. 2 shows the average and individual slope values for the PD and control groups. The results of this slope analysis, further highlights the flatter psychophysical loudness function that was obtained for the participants with PD.

3.2. Imitation task

Results for the imitation task are presented in Table 3 and Fig. 3. The main effect of group was significant [$F(1,40) = 7.05$, $p = .011$]. This result indicates that the participants with PD had significantly lower speech intensity than the control participants across all of the intensity imitation conditions. The main effect of stimulus intensity was significant [$F(4,160) = 234.06$, $p < .001$] and indicates that the participants produced a significant increase in speech intensity in response to increases in the intensity of the imitation stimuli. Interestingly, the interaction between the imitation task and group was significant [$F(4,160) = 6.47$, $p < .001$]. This significant interaction indicates that the participants with PD displayed a flatter or less steep slope in their imitation speech intensity function than the steeper slope displayed by the control participants. Two lines have been added to Fig. 3 in order to highlight the different imitation functions for the PD and control groups and the associated group by imitation task interaction.

Table 3
Descriptive statistics for the imitation task.

	Stimulus intensity level (mean and SD)				
	60 dB	65 dB	70 dB	75 dB	80 dB
PD	63.74 (3.66)	64.93 (2.95)	66.21 (2.49)	67.41 (2.76)	69.34 (2.97)
Control	65.01 (2.44)	66.24 (2.24)	68.02 (2.37)	70.27 (2.25)	72.43 (2.74)

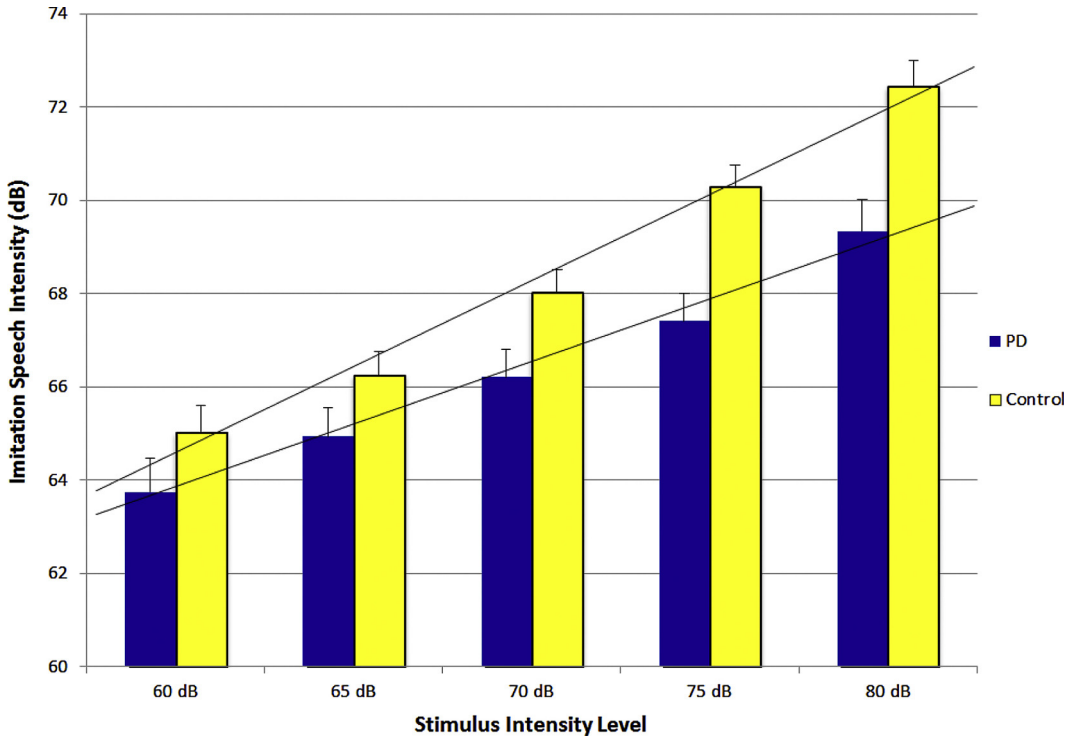

Fig. 3. Average imitation speech intensity produced by the PD and control participants at 5 stimulus intensity levels for the imitation task. Standard error was used for the error bars.

To illustrate the psychophysical imitation function in more detail, a regression analysis was performed on each participant's imitation data and the resulting slope values were submitted to an independent samples t-test. The PD group had a significantly ($t(40) = 3.96$, $p = .001$) lower slope value ($M = 0.27$, $SD = 0.08$) than the control group ($M = 0.38$, $SD = 0.10$). Fig. 4 shows the average and individual slope values for the PD and control groups. The results of this slope analysis, further highlights the flatter psychophysical imitation function that was obtained for the participants with PD.

3.3. Magnitude production task

Results for the magnitude production task are presented in Table 4 and Fig. 5. The main effect for group was significant [$F(1,40) = 14.48$, $p < .001$]. This result indicates that the participants with PD had significantly lower speech intensity than the control participants across all of the magnitude production conditions. The main effect of loudness level was significant [$F(4,160) = 385.66$, $p < .001$]. The interaction between group and production level was significant [$F(4,160) = 11.35$, $p < .001$]. This significant interaction between group and production level indicates that the participants with PD had a less steep slope for their magnitude production function than the control participants. Two lines have been added to Fig. 5 in order to highlight the different magnitude production functions for the PD and control groups and the associated group by task interaction.

To illustrate the psychophysical function for the magnitude production task in greater detail, a regression analysis was performed on each participant's magnitude production data and the resulting slope values were submitted to an independent samples *t*-test. The PD group had a significantly ($t(40) = 5.68$, $p = .001$) lower slope value ($M = 0.025$, $SD = 0.006$) than the control group ($M = 0.036$, $SD = 0.007$). Fig. 6 shows the average and individual slope values for the PD and control

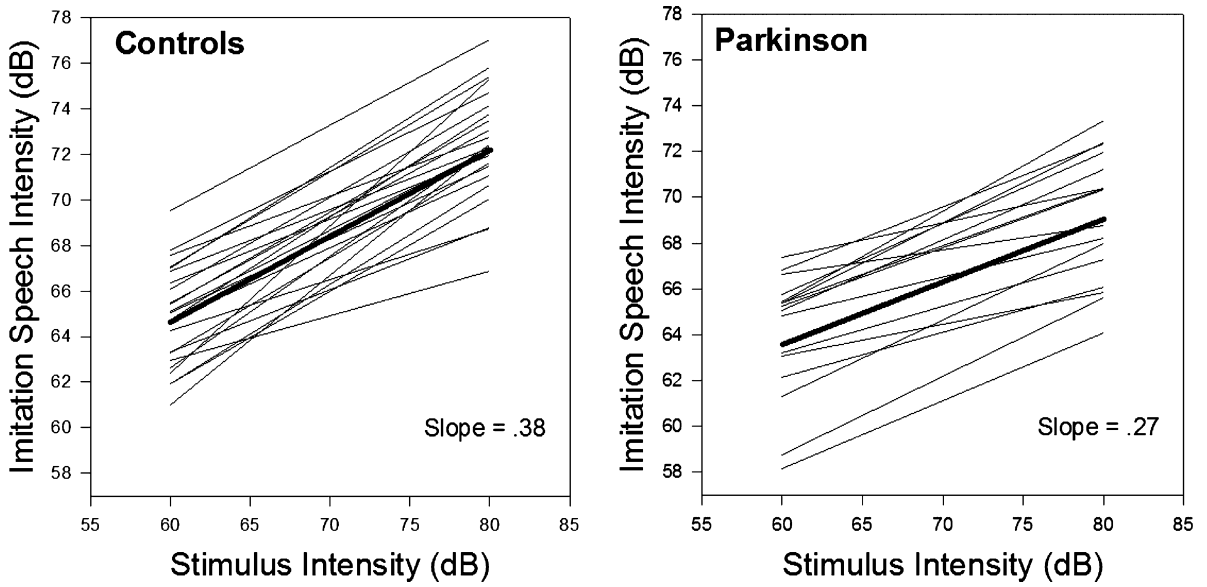

Fig. 4. Average and individual slope values related the imitation intensity for the PD and control participants. The group average is shown by the darker line.

Table 4
Descriptive statistics for the magnitude production task.

	Production level (mean and SD)				
	25	50	100	200	400
PD	60.89 (4.60)	62.96 (3.82)	65.43 (3.08)	68.33 (2.95)	71.10 (3.38)
Control	62.50 (3.32)	65.49 (3.23)	68.09 (2.57)	73.14 (2.83)	76.70 (2.45)

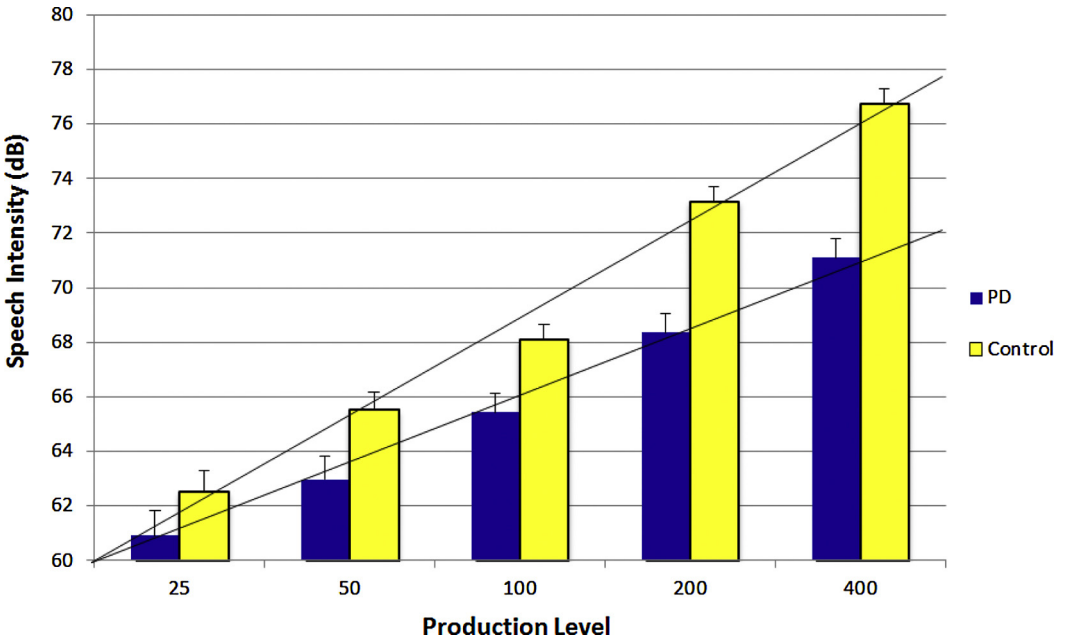

Fig. 5. Average speech intensity produced by the PD and control participants for the 5 intended loudness levels during the magnitude production task. Standard error was used for the error bars.

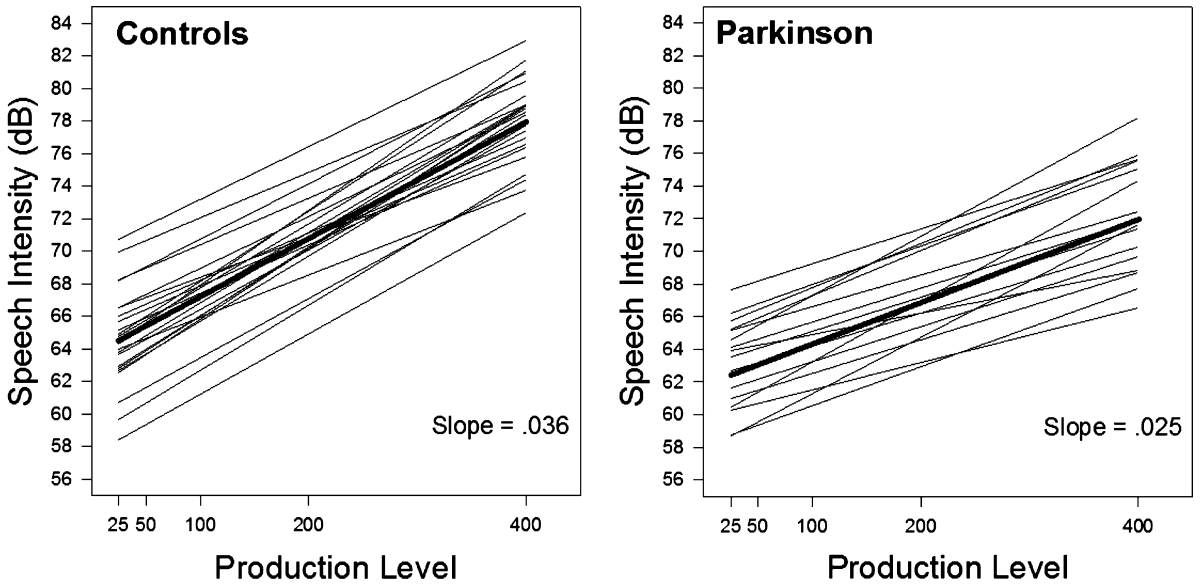

Fig. 6. Average and individual slope values related the magnitude production data for the PD and control participants. The group average is shown by the darker line.

groups. The results of this slope analysis, further highlights the flatter psychophysical loudness function that was obtained for the participants with PD.

4. Discussion

The purpose of the present study was to examine loudness perception in individuals with hypophonia and Parkinson's disease using a combination of magnitude estimation, imitation, and magnitude production tasks.

4.1. Magnitude estimation task

For the magnitude estimation task, the participants with PD and the control participants did not differ on the average level of their loudness ratings. However, the two groups did display different patterns in their loudness ratings across increases in the stimulus intensity. In particular, the participants with PD were generally found to have higher loudness ratings than the controls during the less intense stimuli (60 and 65 dB) and lower loudness ratings during the more intense stimuli (75 and 80 dB). This pattern suggests that the participants with PD were using a more restricted range of loudness ratings than the controls across the 60 to 80 dB stimuli range. This resulted in the participants with PD having a significantly flatter slope for the psychophysical loudness function than the control participants.

These results are consistent with one previous study by Ho et al. (2000) in which participants with PD overestimated the loudness level of recordings of their own speech when it was presented back to them via calibrated headphones after a brief delay. Ho et al. (2000) suggested that their results were consistent with the notion of a sensorimotor integration disorder in PD. A disorder of sensorimotor integration has been proposed as an explanation for the general hypokinetic motor control deficit in PD (Schneider et al., 1986; Tatton et al., 1984). As the term implies, sensorimotor integration is a process in which incoming, movement-related, sensory input interacts with and influences ongoing motor output. This sensory input can include both external sensory feedback and internal feedforward processes (i.e. corollary discharge). The basal ganglia are proposed to play an important role in the sensorimotor integration process (Abbruzzese & Berardelli, 2003). Ho et al. (2000) suggest that the problem of hypophonia in PD is related to a sensorimotor integration disorder that involves the abnormal integration of the sensation of one's own loudness during the motor output of speech intensity. In addition to this sensorimotor integration disorder, Ho and colleagues (2000) suggested that there also may be sensory anomalies in the perception of speech loudness in PD. This notion of a basic sensory deficit is consistent with numerous studies that have found sensory and somatosensory deficits in PD (Patel, Jankovic & Hallett, 2014). Some of these sensory abnormalities include: excessive pain, olfactory loss, visual impairment, vestibular dysfunction, proprioceptive deficits and kinesthetic dysfunction (Patel et al., 2014). Abnormal auditory sensation and perception has also been found in a few studies of PD but this area has received relatively limited attention. These previous studies have reported that PD can be associated with abnormal pure tone audiometry, auditory brainstem responses, acoustic reflexes, temporal discrimination, sound lateralization and tone discrimination (Lewald, Schirm, & Schwarz, 2004; Troche, Troche, Berkowitz, Grossman, & Reilly, 2012; Vitale et al., 2012; Yýlmaz, Karalý, Tokmak, Güçlü, Koçer, & Öztürk, 2009; Murofushi, Yamane, & Osanai, 1992). In

addition, several studies have found an abnormal perception of vocal emotion in PD (Breitenstein, Van Lancker, Daum, & Waters, 2001; Pell & Leonard, 2003; Péron, Dondaine, Le Jeune, Grandjean, & Vérin, 2012). Although these previous studies indicate abnormal auditory perception in PD, very little is known about basic loudness perception in PD. One preliminary study involving the discrimination of tone loudness was recently reported by Troche and colleagues (Troche et al., 2012). This study found that participants with PD had significantly more difficulty discriminating the loudness of 1000 Hz tones that differed by 6 dB, than control participants. This preliminary report indicates the need for additional systematic studies of loudness perception involving both speech and nonspeech stimuli.

With regard to the present study, the results of the magnitude estimation task suggest that participants with PD may have a basic sensory deficit related to the perception of externally generated speech loudness. This sensory-perceptual deficit may have a causal role in hypophonia and is likely to interact with previously proposed sensorimotor integration deficits in PD and hypophonia.

4.2. Imitation task

The participants with PD had lower speech intensity than the control participants across all of the intensity imitation conditions. It is interesting to note that, the participants with PD appeared to have the capacity to achieve speech intensity equivalent to the controls for most of the imitation conditions. For example, as shown in Fig. 3, the participants with PD produced an average intensity during the 80 dB imitation condition that was higher than the average intensity produced by the control participants during the 60 dB, 65 dB, and 70 dB imitation conditions. Thus, despite appearing to have adequate capacity, the participants with PD showed an imitation intensity that was about 2 dB lower than the control participants across all of the imitation conditions. This finding is consistent with a study by Adams, et al. (2006), that found the speech intensity of participants with PD was on average 3–4 dB lower than that of a control group on an imitation task with three imitation target levels (60, 70, 80 dB). Given that the participants with PD had reduced imitation speech intensity despite adequate capacity, it appears that these abnormal imitation results may be related to an impairment in the ability to correctly perceive the intensity of the target stimulus (sensory deficit) or an impairment in the ability to estimate the appropriate intensity level that must be generated in their response to a specific sensory or intensity imitation target (sensorimotor integration deficit). It is also possible that there are impairments in both the sensory and sensorimotor integration processes related to speech intensity regulation.

In addition to lower overall imitation intensity, the participants with PD also displayed a flatter or less steep slope in their imitation speech intensity function than the control participants. Thus, as the intensity of the imitation target increased the participants with PD showed a relatively smaller increase in their response to the target than the controls. It is possible that this flatter than normal imitation function is related to physical limits in the ability of speakers with PD to generate higher levels of speech intensity. However, the participants with PD did not show an obvious ceiling effect in their speech intensity during the imitation task. In addition, the average intensity produced by the control participants during the highest imitation condition (80 dB target) was only 72.4 dB. This value for the upper range of the imitation task seemed to be only moderately taxing and within the capacity of all of the participants with PD in the present study. While an explanation involving a physical limitation cannot be ruled out, it is also possible that the flatter imitation function is related to a sensory or sensorimotor integration deficit. This explanation would be consistent with the results from the magnitude estimation task, which did not have a motor component and also showed a flatter psychophysical function for the estimation of loudness. Thus, for both the imitation function and the loudness perception function, there was a flatter or reduced response as the stimulus intensity increased. In the only other published study that has reported intensity imitation results in PD, Adams et al. (2006) failed to observe a significant difference in the intensity imitation function of PD and control participants. It should be noted that this previous study had a smaller number of participants, fewer imitation conditions and a different speech task than the present study. Clearly, additional studies are required to examine the intensity imitation function in PD in greater detail.

Overall, the participants with PD showed an underestimation and flatter function across the imitation conditions than the controls participants. Although motor limitations may have influenced these results, it is likely that a loudness-based, sensory deficit and/or sensorimotor integration deficit played an important role in the performance of the participants with PD.

4.3. Magnitude production task

The participants with PD had lower speech intensity than the control participants across all of the magnitude production conditions. Similar to the imitation task, the participants with PD appeared to have the capacity to achieve speech intensity equivalent to the controls for most of the magnitude production conditions. For example, as shown in Fig. 5, the participants with PD produced an average intensity during the 200 magnitude production condition (i.e. 2 times louder than habitual) that was higher than the average intensity produced by the control participants during the 100, 50 and 25 conditions. Thus, despite appearing to have adequate capacity, the participants with PD showed a speech intensity during the magnitude production task that was about 2–4 dB lower than the control participants across all of the conditions. More importantly, when the participants were asked to make proportional changes in their speech intensity

(i.e. 2 times louder, 2 times softer, etc.) the participants with PD made relatively smaller adjustments in their intensity. This abnormality is reflected in observation that the participants with PD displayed a flatter or less steep slope in their magnitude production function than the control participants. This magnitude production function also has been referred to as the 'autophonic loudness function' and corresponds to the relation between a participant's perception of their intended speech loudness and their actual speech intensity (Lane et al., 1961). Thus, autophonic loudness perception relates to the perceived loudness of a one's own speech intensity. Individuals with PD have been hypothesized to have an autophonic loudness perception deficit but this has been based largely on anecdotal reports that individuals with PD often fail to identify or perceive the severity of hypophonia in their own speech (Ho et al., 2000; Kiran & Larson, 2001). Although the magnitude production task is recognized as a useful method for evaluating autophonic loudness perception in non-neurologically impaired individuals, it has rarely been reported in previous studies of PD (Dromey & Adams, 2000; Tjaden & Wilding, 2004). Dromey and Adams (2000) reported one of the few previous studies of loudness perception in PD that employed magnitude production procedures. Unlike the present study, Dromey and Adams (2000) failed to find a significant difference between PD and control participants for the slope of the magnitude production (autophonic) loudness function. Two important differences may account for the inconsistent finding of the Dromey and Adams (2000) study and the present study. First, the Dromey and Adams (2000) study used prolonged vowels (e.g. ah; 3 s) while the present study used a spoken sentence. Previous studies indicate that the speech task can have a significant effect on speech intensity and estimates of the severity of hypophonia in PD (Fox & Ramig, 1997). In particular, prolonged vowels are typically associated with relatively high speech intensity values and may be less sensitive measures of PD-related hypophonia than various types of connected speech tasks (Fox & Ramig, 1997; Rosen, Kent, & Duffy, 2005). Second, the study by Dromey and Adams (2000) did not select participants with PD on the basis of signs or symptoms of hypophonia while the present study only selected participants with PD who also had hypophonia. Given the hypothesis of a potentially important causal link between loudness perception deficits and hypophonia in PD, the inclusion of participants with hypophonia may be an explanation for the inconsistencies between the current and previous study and may be an important consideration in future loudness perception studies of PD.

Similar to the imitation task results, the participants with PD showed an underestimation of their own speech loudness and flatter autophonic loudness functions across the magnitude production conditions relative to the control participants. Although motor limitations may have influenced these results, it is likely that a loudness-based, sensory deficit and/or sensorimotor integration deficit played an important role in the performance of these participants with hypophonia and PD.

4.4. Limitations and future directions

The results of the present study were limited to the perception of a fairly short, five-word, declarative sentence with a relatively limited set of phonemes. Additional studies are required to determine if the results will generalize to other sentences with different phonemes, greater lengths and more diverse prosodic patterns. The generalization of these results to other speech tasks, such as picture description, monolog, and conversation, also needs to be examined.

The participants hearing evaluation was limited to the successful completion of a 30 dB HL screening test at 500, 1000, 2000 and 4000 Hz. Future studies of loudness perception in PD should include more extensive audiometric evaluations. As previously mentioned, audiometric abnormalities have been noted in a few previous studies. Further studies are needed to systematically investigate the causal links between hearing performance, loudness perception and hypophonia in PD.

The evaluation of the cognitive abilities of the participants with PD was limited to acceptable performance on a simple cognitive screening test, the Mini Mental Status Exam (MMSE). Since the MMSE is only a screening test, it does not provide an extensive evaluation of cognitive function or allow for the reliable detection of mild cognitive impairments. Performance on some of the loudness judgment and scaling tasks may have been influenced by cognitive performance and specific cognitive abilities (i.e. working memory). Future studies are needed to systematically examine the relationship between specific cognitive functions and performance on loudness perception tasks in PD.

Investigations of loudness perception have been the subject of numerous studies for more than 70 years. A review of these investigations indicates that loudness perception can be approached from many different perspectives and with a wide variety of different methodologies (Florentine, Fay, & Popper, 2011). Some of these diverse methodologies include the following procedures: matching procedures, discrimination procedures, scaling procedures, imitation procedures, detection procedures, and adaptive procedures (Florentine et al., 2011). In addition, previous investigations have revealed that there is a very wide range of specific context effects on loudness perception. Some of these context effects include: stimulus range and sequencing effects, cross-modal effects, noise effects, binaural effects, free field effects, and mental state effects. There also are numerous influences related to the characteristics of the stimulus (frequency, complexity, length, speech, music, noise, tones, etc.) on loudness perception (Florentine et al., 2011). Future studies of loudness perception in PD may need to consider investigating a wider range of evaluation procedures, stimulus characteristics and contexts in order to obtain a more complete understanding of the loudness perception deficit and the role of sensorimotor integration in the regulation of speech intensity in PD.

Finally, if a loudness perception deficit is confirmed to play a role in the hypophonia of PD, then new perceptually oriented approaches to treatment may need to be developed. An example of such an approach might involve the extensive use of perceptual learning procedures that focus on improving the accuracy of externally and internally generated estimates of speech loudness stimuli in a variety of speech conditions and contexts.

5. Conclusions

This study investigated the role of loudness perception in participants with hypophonia related to Parkinson's disease (PD) and controls. For each of the three loudness perception tasks (magnitude estimation, imitation and magnitude production) the participants with PD used a significantly different pattern than the control participants in their self-generated estimates of speech intensity and their judgments of speech loudness. In general, the participants with PD had a more restricted range and a flatter slope than controls for the psychophysical functions related to judgments of speech loudness. These results suggest that individuals with PD may have a speech loudness perception deficit involving the abnormal perception of externally generated and self-generated speech intensity stimuli. These speech loudness perception results appear to provide additional support for both sensory impairments and sensorimotor integration deficits in PD.

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Appendix A. Continuing education

CEU questions

1. Rating the perceived loudness of externally presented speech stimuli is associated with the following procedure:
   a. Magnitude production
   b. Imitation
   c. Masking
   d. Magnitude estimation
2. The term hypophonia is also referred to as:
   a. reduced pitch
   b. monoloudness
   c. low speech intensity
   d. slow speech rate
3. In the present study, the following was found for the slope of the psychophysical loudness function.
   a. Participants with Parkinson's disease and controls had equivalent slope values.
   b. Participants with Parkinson's disease had flatter (lower) slope values than controls.
   c. Participants with Parkinson's disease had steeper (higher) slope values than controls.
   d. Participants with Parkinson's disease had a positive slope value and controls had a negative slope value.
4. In the present study, the following result was found for the speech intensity imitation task.
   a. Participants with Parkinson's disease had significantly lower imitation intensity than controls.
   b. Participants with Parkinson's disease had significantly higher imitation intensity than controls.
   c. Participants with Parkinson's disease had an imitation intensity that was equivalent to that of the controls.
   d. Across the imitation levels, the participants with Parkinson's disease and controls showed a similar pattern of imitation intensity.

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