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Autophonic loudness perception in Parkinson's disease

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The relationship between the intensity and loudness of self-generated (autophonic) speech remains invariant despite changes in auditory feedback, indicating that non-auditory processes contribute to this form of perception. The aim of the current study was to determine if the speech perception deficit associated with Parkinson's disease may be linked to deficits in such processes. Loudness magnitude estimates were obtained from parkinsonian and non-parkinsonian subjects across four separate conditions: self-produced speech under normal, perturbed, and masked auditory feedback, as well as auditory presentation of pre-recorded speech (passive listening). Slopes and intercepts of loudness curves were compared across groups and conditions. A significant difference in slope was found between autophonic and passive-listening conditions for both groups. Unlike control subjects, parkinsonian subjects' magnitude estimates under auditory masking increased in variability and did not show as strong a shift in intercept values. These results suggest that individuals with Parkinson's disease rely on auditory feedback to compensate for underlying deficits in sensorimotor integration important in establishing and regulating autophonic loudness.
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I. INTRODUCTION

The speech disorder associated with Parkinson's disease (PD), hypokinetic dysarthria, is characterized by a reduction in the scale and variation of movement amplitude (i.e., bradykinesia) involving the speech musculature (Darley *et al.*, 1969). These physiological effects give rise to deficits in speech such as reduced loudness, monopitch, and reduced stress, that negatively impact intelligibility (Walsh and Smith, 2012; Duffy, 2014). Interestingly, dysarthric speakers with PD are often unaware of these changes, and typically over-estimate the loudness of their speech despite consistently lower vocalization volumes compared to controls (Ho *et al.*, 1999a, 2000; Fox and Ramig, 1997). A hypothesis of "reduced gain," or "reduced cortical motor set," whereby impaired movement preparation restricts subsequent movement extent, has been proposed to explain production difficulties (Ho *et al.*, 1999a; Ho *et al.*, 1999b), but this notion fails to explain the apparent inability to perceive a mismatch between heard and expected speech intensity. In fact, despite important advances in understanding parkinsonian sensory disorders in general (Tolosa *et al.*, 2009; Siderowf and Stern, 2008), a clear hypothesis regarding the underlying mechanism of speech loudness perception deficits and its link to dysarthric motor control has yet to be developed (cf. Dagenais *et al.*, 1999; Forrest *et al.*, 1998; Moore, 1987).

An important aspect of this perception deficit is that it is specific to self-generated (or "autophonic") speech. Hearing acuity is not affected by PD and, with a few exceptions (Artieda *et al.*, 1992; Lewald *et al.*, 2004), auditory function is generally not impaired. Understanding what it is that makes self-perception "special" may therefore help elucidate what exactly gives rise to this apparent deficit, in persons with a hypokinetic dysarthria, in perceiving and controlling the loudness of their voice.

Early psychoacoustic studies of loudness demonstrated that the relationship between acoustic intensity and estimates of perceived loudness follow different psychophysical curves depending on whether the sound is self-generated or listened to passively (Lane *et al.*, 1961). Specifically, the slope of the loudness function is steeper in autophonic perception compared with auditory-only conditions, as illustrated in the first panel of Fig. 1. This means that an increase of the physically determined intensity of a sound [e.g., dB sound pressure level (SPL)] is perceived as increasingly louder when it is self-generated than when that exact same

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acoustic signal is presented purely auditorily. This finding has prompted a distinction between the sense of loudness and that of effort,[1] and has been proposed to arise from the addition of somatosensory information when speech is self-generated (Lane and Tranel, 1971; Lane *et al.*, 1961).

The dissociation between autophonic and passive-listening loudness functions is pertinent to the speech perception deficit linked to hypokinetic dysarthria in light of findings that vocal tract somatosensory feedback is affected in PD (Hammer and Barlow, 2010; Schneider *et al.*, 1986). This possibility was addressed by Dromey and Adams (2000) in a study comparing individuals with PD and age-matched controls on loudness magnitude estimation (LME) and speech production tasks. No significant differences were found between groups, however. Since all tasks were completed under normal sensory feedback conditions, it is possible that compensation from a differential weighting of one or both of the contributing sensory systems may have obscured differences. Additional control conditions, such as auditory masking, may have revealed those differences, but no follow-up studies have yet tested this.

A related phenomenon that has been more extensively studied in individuals with PD is that of speech in noise (i.e., the Lombard Effect; Lombard, 1911). The general finding has been that speech intensity increases under various masking conditions, but to a lesser degree than among non-parkinsonian controls (NCs) (Darling and Huber, 2011; Coutinho *et al.*, 2009; Adams *et al.*, 2006; cf. Liu *et al.*, 2012, who report an *over*-compensation in an auditory feedback perturbation experiment). The mechanism responsible for the Lombard Effect thus appears to be partially affected by PD. It is important to understand, however, that this effect is conceptually and empirically separable from autophonic loudness perception. Speaking in noise shifts the autophonic loudness function along the intensity axis, but does not change the slope of that function, a natural consequence of the involuntary and unconscious shift in vocal intensity that defines the Lombard Effect (Pick *et al.*, 1989). This effect is illustrated in the second panel of Fig. 1. The observation of

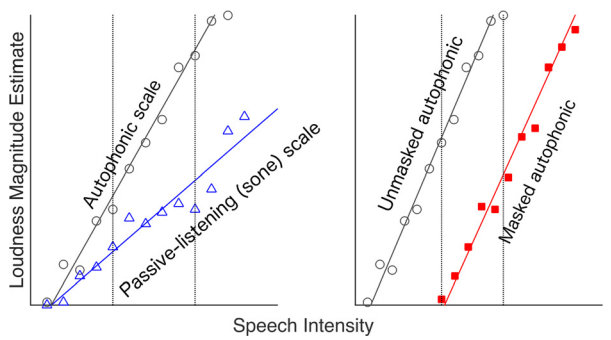

FIG. 1. (Color online) Explanatory sketch of typical loudness curve characteristics in different experimental conditions. Panel 1: Autophonic (gray circles) vs passive-listening (blue triangles) loudness estimates and fitted lines. Autophonic loudness estimates are increasingly larger than passive-listening estimates as intensity increases. Panel 2: Autophonic loudness functions in normal (gray circles) and masked (filled red squares) auditory feedback conditions. Masking shifts the intercept to the right along the intensity axis, but does not affect the relative increase in loudness as a function of intensity (slope invariance).

slope invariance, even under significant auditory masking, is what led Lane and colleagues (1961) to conclude that, "…[auditory feedback] gain has little effect on a person's assessment of the relative levels of his own voice" (p. 165), and to propose a primarily somatosensory basis for the control of speech intensity (Lane and Tranel, 1971; Allen, 1971).

Interestingly, the same reasoning argues against a somatosensory basis for loudness estimation as well. If autophonic loudness is determined uniquely from somatosensory feedback, the increase in produced intensity due to the Lombard Effect should be accompanied by a sensation of increased effort, and no shift in the loudness function intercept should be observed. This is not the case, however. Manipulating auditory feedback intensity is consistently associated with an intercept shift (Lane and Tranel, 1971; Lane, 1962).

In short, the difference in slopes between autophonic and passive-listening conditions, together with the slope invariance and intercept shifts observed under varying feedback conditions, clearly indicate that the perception of self-generated speech cannot be based on sensory feedback alone. This is not to say that the role of sensory feedback is limited to short-term, compensatory behaviors like the Lombard Effect. Speech adaptation studies that have found, following a period of practice under altered feedback conditions, a gradual de-adaptation effect (rather than a sudden reversion) after feedback perturbation is removed (e.g., Houde and Jordan, 1998; Purcell and Munhall, 2006) suggest that perceptual recalibration is possible from sensory manipulations alone. Rather, speakers may derive magnitude estimates from an internal representation of expected sensory consequences established through the integration of movement specifications and prior sensory experience (i.e., an *internal forward model*; see Shadmehr *et al.*, 2010, for a review). Robust psychophysical relationships could therefore be maintained despite variability in movement and sensory feedback (cf. Schmidt, 1975). Dysfunction of motor planning, sensory feedback, or their integration may differentially impact speakers' perception, moreover (Arnold *et al.*, 2014; Tatton *et al.*, 1984).

The implications for speech loudness perception and its deficit in PD are as follows: (1) PD speakers may be expected to maintain an "auditory vs autophonic" loudness difference, as has been demonstrated by Dromey and Adams (2000), on the assumption that these involve different forms of perception (cf. Hafke, 2006; Repp, 2000). (2) Perturbations in auditory feedback may have a detrimental impact on PD speakers if they rely on that form of feedback to compensate for motor deficits and subsequent sensorimotor integration, resulting in poorer psychophysical correlations and a change in loudness function slope. (3) A diminished Lombard Effect may be present in PD speakers, as previously demonstrated (Darling, 2011), resulting in a smaller intercept shift than control subjects.

The present study tested these assumptions in a LME task completed under four different conditions: normal auditory feedback, intensity-shifted auditory feedback, auditory-only (passive listening of recorded speech), and masked auditory feedback.

TABLE I. Participants characteristics. Only the highest (worst) hearing threshold in dB HL, and corresponding frequency in Hz, found on hearing screening are reported. The average intensity on oral reading is reported in dB SPL. Age and education are reported in years. WNL: within normal limits.

Group	#	M/F	Age	Educ.	dB HL	Hz	MoCA	Speech	dB SPL	UPDRS	H&Y
NC	1	M	63	18	30	4000	30	WNL	69.7	0	0
NC	2	F	75	14	30	250	29	WNL	71.0	0	0
NC	3	F	60	12	15	125	26	WNL	75.0	0	0
NC	4	F	63	14	20	all	29	WNL	70.7	0	0
NC	5	F	64	12	25	125	29	WNL	74.6	0	0
NC	6	F	57	14	30	500	28	WNL	70.0	0	0
NC	7	F	51	16	30	4000	29	WNL	72.7	0	0
NC	8	M	59	16	30	4000	28	WNL	72.5	0	0
NC	9	M	60	15	25	4000	29	WNL	73.7	0	0
NC	10	M	48	14	30	4000	27	WNL	75.0	0	0
PD	1	M	72	10	30	4000	27	severe	66.8	41	4
PD	2	M	70	14	30	4000	28	mild-mod	69.7	26	3
PD	3	F	62	18	25	4000	29	mild	69.1	5	2
PD	4	M	59	16	30	4000	24	mild-mod	70.3	29	3
PD	5	F	53	14	25	125	30	mild	69.7	0	2
PD	6	F	51	16	30	125	24	mod	70.5	27	3
PD	7	M	50	16	30	4000	29	mild	70.9	19	2
PD	8	M	56	15	30	500	30	mild-mod	74.5	39	3
PD	9	M	81	11	25	all	26	mod	72.8	32	3
PD	10	M	67	18	30	4000	30	mod	75.1	10	2

II. METHODS

A. Participants

Twenty-four individuals were recruited for this study: twelve with a diagnosis of PD and 12 age-matched controls. One NC subject had significant hearing loss and was excluded from the experiment. One PD subject was excluded because she did not present with dysarthria on either perceptual or acoustic measures. Data from two additional subjects (one NC, one PD) were not included because of errors in equipment calibration.

Of the 20 remaining subjects, all passed a hearing screening with pure-tone thresholds at or below 30 dB hearing level (HL) for frequencies between 125 and 4000 Hz. All subjects scored at least 24 [mean = 27.8, standard deviation (S.D.) = 2.1] on the Montreal Cognitive Assessment (Nasreddine *et al.*, 2005) and reported over 9 yrs of education (mean = 14.5, S.D. = 2.2). Fourteen subjects were English-dominant speakers and read "The Rainbow Passage" (Fairbanks, 1940), seven were French-dominant and read "The Elves and the Shoemaker" (Grimm and Grimm, 1982). Subjects' speech was later perceptually characterized based on audio recordings of their oral readings. Speech dimensions were rated by an experienced Speech-Language Pathologist (first author) on a seven-point (1 = normal to 7 = profound) scale following the Darley *et al.* (1969) classification system elaborated in Duffy (2014). All subjects with PD displayed some degree of monoloudness (mean = 2.4), loudness decay (mean = 2.8), and monopitch (mean = 2.2). Other relevant dimensions included breathiness (9/10 subjects, mean = 2.3), imprecise consonants (8/10 subjects, mean = 1.9), weak pressure consonants (6/10 subjects, mean = 1.4), slow rate (8/10 subjects, mean = 1.6), and reduced stress (8/10 subjects, mean = 2.5). No deviant speech characteristics were identified in the control group's recordings. Calculations of signal intensity showed that PD subjects read aloud at lower volumes than controls, with a mean of 69.9 (S.D. = 2.2) vs 72.0 (S.D. = 2.0) dB SPL for English, and 72.8 (S.D. = 2.1) vs 74.3 (S.D. = 1.0) dB SPL for French.

PD group scores on portions II and III of the Movement Disorder Society (MDS)-sponsored Unified Parkinson's Disease Rating Scale (UPDRS; Goetz *et al.*, 2008) averaged 16.4 ($\sigma = 8.3$) and 21.6 ($\sigma = 12.7$), respectively. Mean Hoehn and Yahr staging was 2.3 ($\sigma = 1.1$). Subject characteristics are summarized in Table I. All individuals in the PD group were off medication at least 2 h before beginning the experiment. Recruitment and experimental procedures were reviewed and approved by the McGill University Faculty of Medicine Institutional Review Board.

B. Procedure

Subjects were asked to either produce or listen to the vowel /ʌ/ at different intensities, then rate their impression of the loudness of each sound using any positive-numbered scale of their liking (Zwislocki and Goodman, 1980). Instructions were to "say the sound 'uh' as a short syllable, not a sustained vowel"; duration was not otherwise regulated. Ratings were made under four feedback conditions, each consisting of 30 repetitions, presented in the following fixed order: (1) speech with normal auditory feedback (*SPEECH-NAF*), (2) speech with intensity-altered auditory feedback (*SPEECH-AAF*), (3) passive listening of played-back speech (*LISTEN*), and (4) speech with masked auditory feedback (*SPEECH-MASKED*). In the three speech production conditions, each prompt to vocalize was preceded by the instruction "Say 'uh' normally," "Say 'uh' more loudly," or "Say 'uh' more softly," allowing for a sampling of a wide range of intensities. Instructions within conditions were

TABLE II. Summary of experimental conditions. Speech intensity was measured from the signal produced at the microphone and what was presented to the earphones, except in the *LISTEN* and *SPEECH-MASKED* conditions. Speech tokens presented in the *LISTEN* condition were recordings from the *SPEECH-AAF* presented in a randomly permuted order.

	Signal		
Condition	At the microphone	At the earphone	Feedback alteration
SPEECH-NAF	speech	speech	none
SPEECH-AAF	speech	speech	0, 3, 6 dB SPL
LISTEN	none	speech	N.A.
SPEECH-MASKED	speech	noise	noise

completely randomized. The experimenter deliberately avoided any mention of "effort" or how a production "felt."

Subjects' speech intensity in the *SPEECH-AAF* condition was altered by ±6, ±3, or 0 dB. Settings were controlled electronically in combination with the presentation of visual stimuli. Signal intensity was altered immediately following the onset of a visual cue to vocalize. After each vocalization, the processor returned to a 0 dB (no shift) setting while the subjects provided their magnitude estimation. A random direction and magnitude of feedback alteration was then applied during the inter-stimulus interval and up to the subsequent shift at the following vocalization cue to deter anticipation of feedback perturbation—if perceived—at the vocalization cue.

Tokens played back in the *LISTEN* condition were recordings of the subject's own productions made during the *SPEECH-AAF* condition, presented at their original production levels (dB recorded at the mouth) and in a randomized order. Auditory feedback in the *SPEECH-MASKED* condition was masked by 90 dB speech-weighted noise, presented when cued to vocalize and ending when asked to make a LME.

Sound intensity was measured at the mouth (microphone) and the ear (earphones) for each of the three speech production conditions. The experimental data were then examined across the four feedback conditions, with separate measures for the signal measured at the microphone and at the ear for the first two conditions, as summarized in Table II.

C. Instrumentation

Participants spoke into a head-mounted microphone (DPA 4066, DPA Microphones Inc., Longmont, CO) positioned 4–5 cm from the mouth. The signal was pre-amplified and passively split into two channels. One channel was recorded directly onto a computer. The second channel was routed to an acoustic signal processor (Yamaha SPX, Yamaha Commercial Audio Systems, Buena Park, CA) capable of altering signal intensity. The processed acoustic output signal was again split, with one channel digitally recorded and the other channel amplified and presented binaurally through insert earphones (Etymotic ER2, Etymotic Research, Elk Grove Village, IL). The delay between the signal at the microphone and the signal feedback at the ear was less than 10 msec in all conditions. The digital recording and adjustment of the signal processor was controlled using custom software written in MATLAB (v. 2010b, Mathworks, Natick, MA). All signals were digitized at 44.1 kHz sampling rate with 16 bit quantization. The recording and playback equipment was calibrated to dB SPL by sampling different intensities of a speech-weighted noise (with cutoffs at 700 and 5000 Hz) and applying a fifth-order polynomial fit to the measured dB as a function of signal root-mean-square; sound level meter settings were A weighting, wide averaging window, with a range from 50 to 90 dB SPL and an error of ±1 dB.

III. DATA ANALYSIS

A. Intra-subject correlations

Recorded signals were segmented using custom software that identified sound onset and offset from the average absolute difference level (McLoughlin, 2009), then converted the segmented signal's root-mean-square to decibels per calibrated values. Null entries (tokens where subjects failed to vocalize) and those outside the 50–90 dB SPL calibration range were removed from the data set. Productions averaged 273 msec (S.D. 0.1, range 112–643 msec) for control subjects and 366 msec (S.D. 0.14, range 171–826 msec) for persons with PD. Overall, high intensity-to-loudness correlations were observed for all data sets, with notable exceptions in the altered feedback condition (dB at the ear) across groups and in the masked feedback condition for the Parkinson group. A Friedman's analysis of variance (ANOVA) (Friedman, 1937) of those correlations reached significance ($\chi^2(5) = 31.07$, $p < 0.001$); Wilcoxon rank-sum tests comparing groups in each condition, corrected using the Holm-Bonferroni method (Holm, 1979), revealed a significant difference for the masking condition alone ($W_s = 142$, $z = 2.76$, $p = 0.006$, corrected $p = 0.04$). Table III provides correlation coefficient means and S.D.

B. Curve fitting

Linear, logarithmic, and power fits to the data were then calculated for each condition for every subject. An assessment of goodness-of-fit using coefficients of determination (R^2) failed to show a significant difference between fit types. Linear regression was therefore considered an appropriate method for loudness function estimation.

Responses were normalized to a 0–10 scale by multiplying each subject's magnitude estimates by the largest estimate at 90 dB SPL across all conditions. In order to avoid extrapolating values beyond the calibrated range, intercepts were normalized relative to the intercept in the first (normal feedback) condition. Intercepts for conditions 2–4 therefore represent magnitude estimation relative to the normal feedback conditions.

Loudness functions were subsequently analyzed using a by-participant linear regression (Lorch and Myers, 1990). This is a two-phase process that involves first computing separate linear regression equations for each subject, followed by a test of those regression coefficients. Regression models were accepted if the calculated coefficient of determination was greater than 0.1. This threshold was equivalent to rejecting models with coefficient p-values greater than 0.05.

TABLE III. Mean and S.D. of correlations coefficients across conditions.

Group	Condition					
	SPEECH-NAF(mic)	SPEECH-NAF(ear)	SPEECH-AAF(mic)	SPEECH-AAF(ear)	LISTEN	SPEECH-MASKED[a]
NC	0.83 (0.095)	0.83 (0.095)	0.87 (0.061)	0.66 (0.156)	0.78 (0.096)	0.85 (0.114)
PD	0.86 (0.060)	0.87 (0.060)	0.84 (0.090)	0.66 (0.156)	0.78 (0.077)	0.62 (0.226)

[a]An asterisk marks within-condition significant difference ($p < 0.05$, corrected for multiple comparisons).

IV. RESULTS

Mixed design ANOVAs with group (NC, PD) as between-subjects factor and condition (feedback type) as within-subjects factor were run on the regression coefficients. With respect to slope, no interaction ($F(5,90) = 1.965$; $p = 0.092$) or main effect ($F(1,18) = 0.144$; $p = 0.709$) of group were found, but there was a significant main effect of condition ($F(5,90) = 7.342$; $p < 0.001$). Collapsing across groups, *post hoc* comparisons between conditions were carried out using *t*-tests corrected for multiple comparisons with the Holm-Bonferroni method. Significant differences were identified between the following conditions: SPEECH-NAF vs SPEECH-AAF at the ear ($t(19) = 4.4$, corrected-$p = 0.002$), SPEECH-NAF vs LISTEN ($t(19) = 3.6$, $p' = 0.01$), SPEECH-AAF at the mouth vs at the ear ($t(19) = 5.3$, $p < 0.001$), and SPEECH-AAF at the mouth vs LISTEN ($t(19) = 3$, $p' = 0.035$). Distributions of calculated slopes for each condition and in each group are displayed in Fig. 2.

With respect to intercept, a significant interaction ($F(5,90) = 4.0$; $p = 0.002$) and main effect for condition ($F(5,90) = 2.467$; $p = 0.038$) were found, but no main effect for group ($F(1,18) = 1.570$; $p = 0.226$). *Post hoc* t-tests with Holm-Bonferroni correction comparing groups in each condition indicated the effect arose from differences between groups in the masking condition: SPEECH-NAF $t(18) = -2.2$, corrected-$p = 0.18$, SPEECH-AAF $t(18) = 0.25$, corrected-$p = 1.2$, LISTEN $t(18) = 0.5$, corrected-$p = 1.8$, SPEECH-MASKED $t(18) = -2.9$, corrected-$p < 0.05$. Distributions of loudness function intercepts relative to the intercept of the normal feedback condition are plotted in Fig. 3.

Group-averaged loudness functions for SPEECH-NAF, SPEECH-MASKED and LISTEN conditions are plotted in Fig. 4.

Post hoc analyses were conducted to assess the contribution of side-tone (SPEECH-AAF) and Lombard (SPEECH-MASKED) effects on subjects' productions. The intensity-shifted feedback analysis was limited to tokens produced on "normal" instructions since these did not require subjects to base themselves on preceding production volumes and were presumably drawn from the center of their loudness distributions. A mixed ANOVA comparing shift direction (down, 0, up) and group (NC, PD) failed to reach significance, $F(2,36) = 0.13$, $p = 0.88$. With respect to the SPEECH-MASKED condition, a Wilcoxon rank-sum test comparing subject groups on feedback intensity differences (masked minus normal feedback) was significant ($W_s = 137$, $z = 2.38$, $p = 0.017$). The increase in vocal intensity for control subjects approached 10 dB SPL, equivalent to the intercept shift observed in that same condition. The median increase in intensity for PD subjects approached 4 dB SPL, indicating that the overall Lombard Effect was present but not as strong in this group (cf. Darling, 2011; Adams *et al.*, 2006).

Much of the group difference observed in loudness estimate intercepts appears to be driven by a subset of individuals in the PD group who had difficulty gauging loudness under auditory masking and gave seemingly arbitrary responses. Two examples are provided in Fig. 5. Panel 1 shows a limited range of produced intensities and no shift

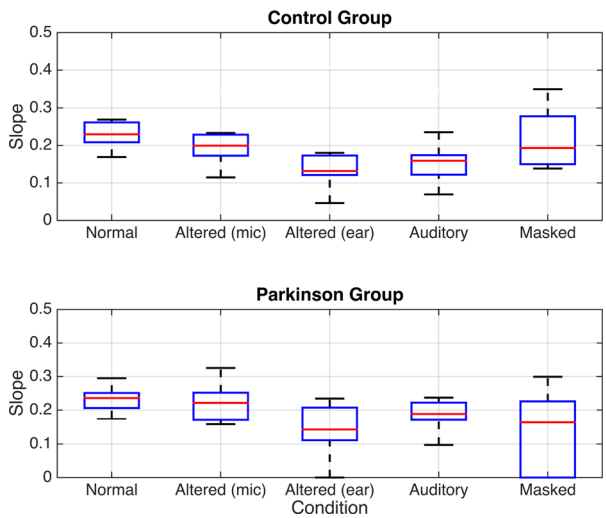

FIG. 2. (Color online) Per-group distributions of slopes in each of the experimental conditions: normal feedback, altered feedback (intensity-shifted), auditory-only, and masked feedback. Slopes calculated relative to the intensity measured at the mic and that measured at the earphone are included for the altered feedback condition. Each box represents 25th and 75th percentiles, the center line the median, and whiskers extend to 2.7 S.D.

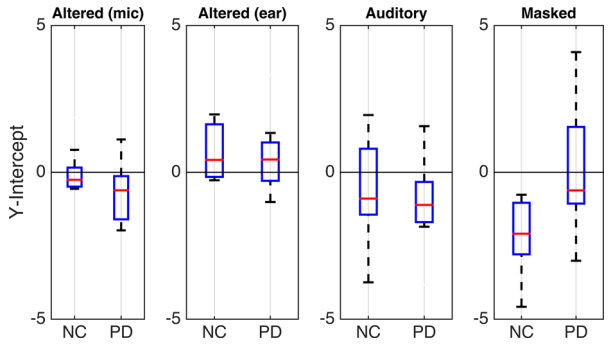

FIG. 3. (Color online) Group (NC, PD) comparisons of loudness function intercepts relative to intercepts in the normal feedback condition. Each box represents 25th and 75th percentiles, the center line the median, and whiskers extend to 2.7 S.D.

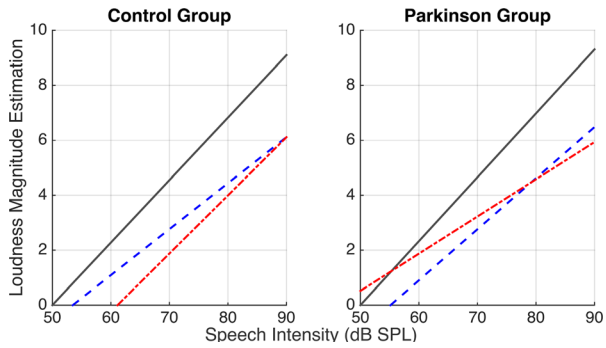

FIG. 4. (Color online) Group-averaged loudness magnitude estimates for the normal (solid gray line), masked (dotted red line), and auditory-only (dashed blue line) conditions.

with masking. Panel 4 shows a more extended range of responses and a clearer shift in produced intensities, but still restricted with respect to other conditions. No clear relationship could be identified between apparent subgroups and disease severity or other demographic variables. Given demonstrated variability in response to treatment (Kompoliti *et al.*, 2000) and suggestions of possible subtypes of hypokinetic dysarthria in PD (Logemann *et al.*, 1978), however, it is possible that the participants with PD presented with qualitatively different self-monitoring deficits.

V. DISCUSSION

The goal of the present study was to determine whether the self-perception of speech loudness in persons with PD differed from age-matched controls when auditory feedback was masked or shifted in intensity. In line with previous findings (Dromey and Adams, 2000; Lane *et al.*, 1961), LME slopes were steeper in autophonic as compared to passive-listening conditions, for both Parkinson and control groups. A given increase in speech intensity was perceived approximately 1.3 times louder when self-generated than when listened to passively. This difference, along with the fact that slopes remained stable across autophonic conditions despite changes in auditory feedback, supports the notion that it is the act of vocalizing that informs subjects' loudness perception of their own speech, and not merely changes in auditory signal intensity (Lane, 1962; Eriksson and Traunmüller, 1999).

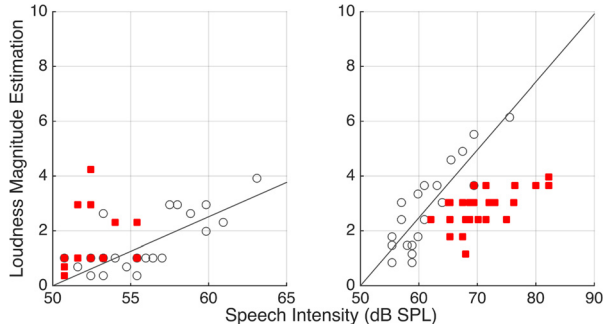

FIG. 5. (Color online) Two example subjects in the Parkinson group with poor correlations in the masking condition. Autophonic loudness functions for normal (gray circles) and masked (filled red squares) feedback conditions. Note that the *x* axis of the left panel has been shortened to allow clearer visualization of responses.

Loudness magnitude estimates in the auditory-masking condition provide a particularly strong argument for this interpretation. At 90 dB SPL, the masking noise effectively removes all auditory feedback of subjects' speech, except possibly at the highest vocalization intensities. Speakers are forced, in other words, to make loudness judgments based on non-auditory, physiological dimensions, such as their sense of effort. Control subjects maintained an intensity-to-loudness relationship (slope) equivalent to other speaking conditions, and did so with little dispersion around the fitted loudness curve (high correlations). Subjects with PD, on the other hand, showed a significant increase in dispersion in the masking condition, suggesting that they were in fact relying on auditory feedback to compensate for deficits in other mechanisms that are otherwise contributing factors in establishing magnitude estimates.

In the altered feedback (intensity-shifted) condition, magnitude estimates correlated more closely with produced than heard intensities. This is again consistent with the notion that the estimates are not based on auditory feedback. No group differences were found, however; PD subjects did not over-compensate to intensity-shifted feedback, as has been found elsewhere (Liu *et al.*, 2012). The lack of an effect is likely due to the nature of the task and consequent restriction on the calculation of response magnitude. Since the perturbation was applied before vocalization onset and maintained throughout the vocalization, we could not normalize to each production's "baseline" as is commonly done in auditory perturbation paradigms (e.g., Liu *et al.*, 2012), but instead normalized across tokens with similar instruction and feedback parameters. The size of any compensatory response, in other words, was based on an overall average intensity, obscuring compensation at the level of individual tokens.

In light of the current findings, we propose that, outside its role in eliciting the Lombard Effect, sensory feedback in general provides fine-grained control over a loudness scale that is coarsely specified by planned actions and their predicted consequences. A degradation or absence of auditory information requires greater reliance on intact feedback systems to retain equivalent levels of accuracy. In individuals with deficient somatosensation, the relationship between measured intensity and magnitude estimates may begin to show some dispersion, as was the case with our PD speakers that had poorer yet relatively intact loudness estimation slopes. An additional deficit at the level of motor planning (or programming) would then result in increased dispersion and dissolution of the autophonic scale, as was observed among PD speakers who failed to show loudness functions different from a simple mean-response model. The shape of the autophonic scale would therefore primarily reflect planned movement goals, yet require additional information from sensory systems to correlate with physical attributes of the speech signal. This explanation is supported by pitch and intensity perturbation studies that have shown greater signal gain on smaller perturbations than larger ones (Bauer *et al.*, 2006), as corrections based on sensory feedback act to refine existing sensorimotor coordinations, not to generate them. It also agrees with the finding that individuals with PD

continue to display a Lombard Effect (Liu *et al.*, 2012; Darling and Huber, 2011), despite poor loudness estimate-to-intensity correlations.

In conclusion, the consistency of autophonic loudness functions across varying auditory feedback conditions and their difference relative to passive-listening of the same speech tokens supports the hypothesis that the judgment of loudness of self-generated speech relies principally on non-auditory information, possibly in the form of internal predictions based on the integration of central (efferent) signals and prior sensory feedback (Niziolek *et al.*, 2013; Schmidt, 1975). The finding that auditory masking leads to deficient autophonic loudness scaling among individuals with mild-to-moderate hypokinetic dysarthria associated with PD suggests, however, that sensory feedback helps to fine-tune a scale that is only generally specified from planned motor consequences. The patterns of breakdown observed among PD subjects under auditory masking further indicate that loudness perception deficits associated with hypokinetic dysarthria result from deficits in both somatosensory and motor systems. Methodologies that are able to tease apart independent contributions from these systems to compensatory vocal responses like the Lombard Effect, as well as speakers' self-perception, will help ascertain the validity of these claims.

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[1]The notion of effort as a distinct psychophysical percept, differentiable from loudness, was debated and tested in a number of studies, with the general consensus that both are multidimensional and inter-related phenomena (Allen, 1971; Brandt *et al.*, 1969; Ladefoged and McKinney, 1963). This debate was entirely focused on acoustics; however, all magnitude estimates were completed under passive-listening conditions. These studies did not directly address the role of non-auditory, physiological dimensions implicated in the distinction between autophonic and "passive-listening" scales such as the sone scale (Stevens, 1955).

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