Sensorimotor Adaptation of Speech in Parkinson's Disease

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ABSTRACT: The basal ganglia are involved in establishing motor plans for a wide range of behaviors. Parkinson's disease (PD) is a manifestation of basal ganglia dysfunction associated with a deficit in sensorimotor integration and difficulty in acquiring new motor sequences, thereby affecting motor learning. Previous studies of sensorimotor integration and sensorimotor adaptation in PD have focused on limb movements using visual and force-field alterations. Here, we report the results from a sensorimotor adaptation experiment investigating the ability of PD patients to make speech motor adjustments to a constant and predictable auditory feedback manipulation. Participants produced speech while their auditory feedback was altered and maintained in a manner consistent with a change in tongue position. The degree of adaptation was associated with the severity of motor symptoms. The patients with PD exhibited adaptation to the induced sensory error; however, the degree of adaptation was reduced compared with healthy, age-matched control participants. The reduced capacity to adapt to a change in auditory feedback is consistent with reduced gain in the sensorimotor system for speech and with previous studies demonstrating limitations in the adaptation of limb movements after changes in visual feedback among patients with PD. © 2013 *Movement* Disorder Society

Key Words: sensorimotor adaptation; speech production; Parkinson's disease; speech motor learning; auditory feedback

Although the cardinal symptoms associated with Parkinson's disease (PD) are predominantly associated with motor system impairment, sensory deficits are well known.[1–4] Two of the major speech characteristics of PD are reduced vocal loudness and reduced respiratory control,[5] which appear to have a sensory contribution. For example, when asked to increase speech loudness, patients with PD often complain of a perception of being too loud.[6] The misperception of self-generated loudness may be due to the apparent reduced sensitivity of laryngeal mucosal mechanoreceptors[7] generating a false sense of increased effort while speaking. In the auditory domain, when presented with a sudden change in auditory feedback of the pitch of their voice, individuals with PD respond with a larger adjustment than non-affected controls.[8] In addition to problems associated with speech loudness or pitch control, these speech symptoms are consistent with a more general problem of sensorimotor integration.[9–11]

One consequence of the sensory and motor impairments in PD patients may be reflected in the difficulty they exhibit in learning new motor skills and modifying existing motor behaviors.[12–14] In speech, this deficit is observed in the extensive behavioral training needed to ameliorate their speech disorder.[6,15,16] Until recently, behavioral treatment for PD resulted in minimal and short-term gains. In contrast, intensive, high-effort training and a focus on improving sensorimotor processing have provided some of the most robust

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treatment effects with this population.[17] It appears that the speech symptoms in PD patients are not localized to the inability to adjust speech parameters, such as loudness or articulation, but reflect a more general problem with sensory recalibration, which, by extension, may reflect impaired sensorimotor learning.

We addressed the question of sensorimotor integration and learning for speech through an experimental manipulation of auditory feedback in real time during speech production. This manipulation introduces a predictable and constant feedback error associated with discrete acoustic properties of the speech signal that have predictable acoustic/motor mappings (eg, vowel formant frequencies or consonant fricative energy). Over the course of a single experimental session, these manipulations result in speech motor changes that adapt to the induced error.[18–24] For example, altering the first formant (F1) frequency of a vowel (an acoustic cue related to vertical tongue position) yields a change in speech motor output consistent with a compensatory change in tongue height.[20,21,25,26] As a result, by evaluating the ability of individuals to engage a process of sensorimotor adaptation (SA), it is possible to evaluate whether and the extent to which the direct sensorimotor (acoustic-articulatory) mapping is intact in patients with PD and to evaluate their ability to learn new sensorimotor mappings independent of vocal (pitch and loudness) or respiratory control.

We used an SA paradigm to assess the capacity of PD patients to alter their speech motor control in response to a perceived auditory-sensory error. We hypothesized that deficits in sensorimotor integration would limit the capacity for speech motor learning in PD patients—a result that, if demonstrated, would have important implications for understanding the speech disorder of patients with PD as well as their ability to change their speech through behavioral intervention.

Participants and Methods

The study was approved by the Institutional Review Board at the Faculty of Medicine, McGill University. All participants gave written informed consent.

Participants

Participants included nine individuals with idiopathic PD (7 men, 2 women; mean age, 63.8 years) and nine healthy, age-matched controls (4 men, 5 women, mean age, 60.3 years). The severity of PD motor symptoms, which we evaluated using the Unified Parkinson's Disease Rating Scale (UPDRS), ranged from mild (a score of 15) to moderate (a score of 88; mean $\pm$ standard deviation [SD] score, 33.2 ± 13.5). Cognitive functioning evaluated with the Mini-Mental State Examination was in the normal range for all PD patients (scores > 24). All patients were taking L-dopa and other medication, including dopaminergic and/or anticholinergics. Participants were tested during their *off* state (6 hours off medication); one patient with PD was tested only two hours off medication due to disease severity. Only two participants had a history of speech therapy; one could not remember the specifics of the treatment, and one recalled that treatment consisted of exercises focusing on increasing intelligibility and loudness.

For each PD participant, the Rainbow Passage was recorded for perceptual analysis. A licensed Speech-Language Pathologist rated participants' speech on 43 perceptual dimensions that evaluated all the speech subsystems.[33] Overall, the severity of the PD participants was rated as moderate (one patient), mild (six patients), and within normal limits (two patients). We used the perceptual and UPDRS scores to assess any relationship between severity of speech and motor symptoms with the magnitude of adaptation.

Participants in the control group had no history of neurological impairment. All participants had binaural pure tone hearing thresholds of 40 dB HL or less at 250, 500, 1000, 2000, and 4000 Hz. None of the participants wore hearing-aids.

Audio Recording

Participants sat in front of a computer monitor and spoke into a microphone located 20 cm from their mouth. The microphone signal was digitized at 44.1 kHz (16 bit). The microphone signal was presented back to the individual (see below for details) through headphones (880 Pro; Beyerdynamic, Inc. USA, Farmingdale, NY).

Speech was cued by presentation of the target word on the computer display. Each stimulus was presented for 2 seconds followed by a 1-second blank display. Participants produced the target word at a comfortable rate and volume immediately after the visual presentation. Speech volume was maintained by providing the participant with a volume unit meter on the computer display.

Manipulation of Auditory Feedback

Participants produced individual words under conditions of altered auditory feedback (for details, see Rochet-Capellan and Ostry[21] and Shum et al[25]). Briefly, the first formant frequency of the vowel /ɛ/ in the target word "head" (/hɛd/) was manipulated with a digital signal processor (DSP) specialized for speech acoustic signals (VoiceOne; TC Helicon Vocal Technologies, Victoria, BC, Canada) in near-real time (11-ms delay). During maximal acoustic perturbation, the first formant frequency was increased by 30%, resulting in an acoustic signal that was closer to the vowel /æ/ (as in the word "had"). The altered speech signal was amplified and presented at a volume of approximately 70 dB. To reduce the participant's perception of their unmodified,

air-conducted and bone-conducted feedback signal, the altered speech signal was mixed with pink noise (presented at approximately 60 dB).

Testing Sequence

Participants produced 200 repetitions of the word /hɛd/ ("head") in the following sequence: 30 trials produced under an unaltered feedback condition (baseline phase), followed by the sudden introduction of the acoustic perturbation, and 100 trials under conditions of maximal acoustic perturbation (hold phase). Finally, auditory feedback was gradually returned to normal over 40 trials (ramp-offset phase) and then maintained for 30 trials to facilitate de-adaptation (after-effect phase). See Figure 1 for schematic.

Data Analysis

For each participant, the values of the first two formant frequencies (F1 and F2) were computed over a 30-ms window centered at the mid-point of the vowel /ɛ/. In addition, we calculated the difference between F2 and F1 (F2-F1). Several factors motivated our use of this parameter. In typical productions of the vowels /ɛ/ and /æ/, the contrast is associated with a change not only in F1 but in the difference between F2 and F1—/æ/ being associated with a smaller F2-F1 separation than /ɛ/ (eg, see Peterson and Barney[28] and Kent and Read[29]). We were further motivated to include F2-F1 as a dependent measure because, in prior studies of speech adaptation (eg, Rochet-Capellan and Ostry[21]), compensatory changes in speech output have been observed in both F2 and F1 in response to a manipulation of auditory feedback involving an increase in F1 alone.

To evaluate the changes in speech production across the 200 tokens in the speech adaptation task, individual F1, F2, and F2-F1 values were normalized and then averaged over successive blocks of 10 trials. The raw formant values (in Hz) were converted to reflect the proportional change in frequency relative to the baseline phase under normal (unaltered) feedback conditions. This eliminated the influence of baseline differences before averaging across participants, in addition to representing the change in speech output in the same units as the feedback manipulation itself (a proportional change in F1).

For each dependent variable, a mixed-factorial, two-way analysis of variance was carried out to examine differences between the PD group and the control group (GROUP: the between-subjects effect) at four time points of interest during the adaptation protocol (PHASE: the within-subjects effect): (1) the beginning of the *hold* phase, characterizing the degree of adaptation immediately after the sudden onset of the feedback perturbation; (2) the end of the *hold* phase, characterizing the degree of adaptation at the end of 100 trials of practice under conditions of maximum feedback alteration; (3) the beginning of the *after-effect* phase, reflecting vowel production after the gradual restoration of unaltered feedback; and (4) at

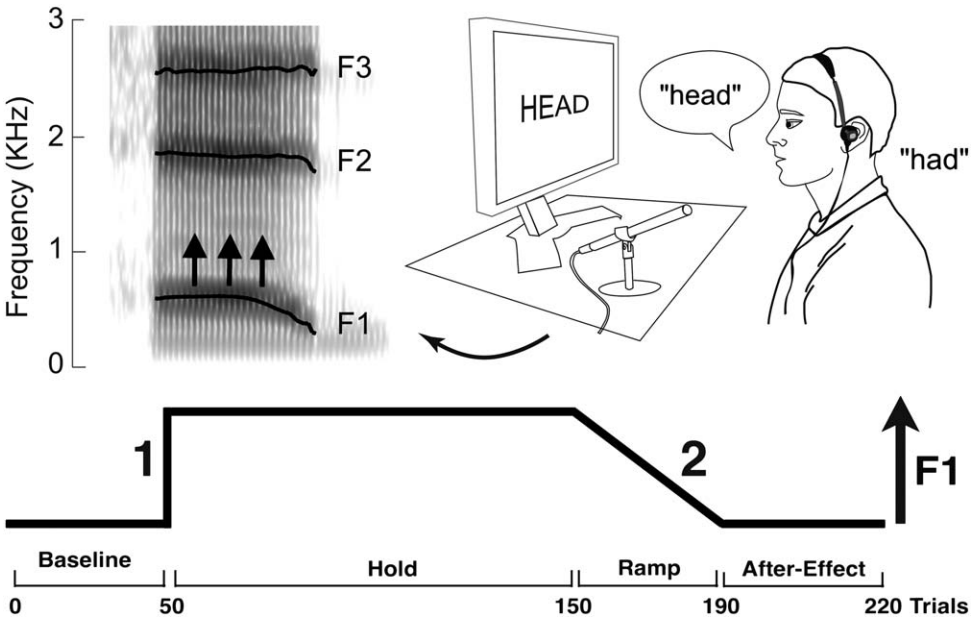

FIG. 1. Top: The testing sequence illustrating the scheme for introducing the auditory feedback manipulation. **Bottom:** During the baseline trials, participants read words off a computer screen in the presence of unaltered auditory feedback (50 trials). During the Hold phase, the feedback alteration was introduced suddenly and was maintained for 100 trials; this was followed by a period of gradual restoration of auditory feedback (40 trials) and another 30 trials with normal auditory feedback (after-effect). The feedback manipulation used was an increase in the first formant (F1) frequency of the participants' auditory feedback, which is associated inversely with the height of the tongue in the oral cavity.

the end of the *after-effect* phase (at the end of 30 trials under normal auditory feedback conditions).

Results

Baseline productions of the vowel /ɛ/, taken over trials 16 through 30 (under conditions of unaltered auditory feedback), were comparable between the PD group and the control group. Mean and variability measures (SD) values of F1 and F2 frequency (in Hz) are shown in Figure 2. No statistically reliable differences were observed between the groups for any of the measures (F1 mean: $t[16] = -0.12$; $P > 0.05$; F2 mean: $t[16] = 0.23$; $P > 0.05$; F1 variability: $t[16] = -0.13$; $P > 0.05$; F2 variability: $t[16] = -0.32$; $P > 0.05$). Average root mean square values were obtained from each participant's vowel recordings during the last 15 trials of the baseline and hold phases to evaluate changes in speech loudness over the course of the experiment. No statistically reliable differences were found for either group (PD group [mean ± SD]: baseline, 0.1819 ± 0.0175 Hz; hold, 0.1844 ± 0.0143 Hz; $t[8] = 2.1435$; $P = 0.07$; control group [mean ± SD]: baseline, 0.1660 ± 0.0157 Hz; hold, 0.1824 ± 0.0136 Hz; $t[8] = 0.1519$; $P = 0.88$).

Changes in F1 and F2 values throughout the speech adaptation procedure are shown in Figure 3. For the control group (Fig. 3, top), compensation to the auditory feedback manipulation (decrease in F1 and increase in F2 relative to baseline) can be observed, with the changes beginning soon after the sudden introduction of the feedback manipulation (beginning in block 6) and maintained throughout the hold phase. The gradual restoration of normal auditory feedback during the ramp-offset phase yielded a nearly complete de-adaptation in F1 and F2. By the final block of after-effect trials, vowel productions resembled those observed during the null phase. The PD group (Fig. 3, bottom) also exhibited compensatory change in vowel formants beginning immediately after the sudden introduction of the auditory feedback manipulation and continuing throughout the hold phase. However, the maximum change in F1 and F2 for the PD group at the end of the hold phase was notably reduced compared with that of the control group. The proportional change in F1 and F2 for the control group averaged −0.90 (−9.0%) and 0.044 (4.4%), respectively, whereas the proportional change in F1 and F2 for the PD group averaged −0.058 (−5.8%) and 0.014 (1.4%), respectively. Both groups exhibited a de-adaptation effect in F1 and F2 during the ramp-offset phase.

The effects of GROUP and PHASE on F1, F2, and F2-F1 were evaluated using a two-way analysis of variance. Mean formant values at four epochs are shown in Figure 4. For F1, a reliable main effect of PHASE ($F[2.17,34.81] = 14.19$; $P < 0.01$) was found with no reliable effect of GROUP ($F[1,16] = 0.659$; $P > 0.05$) or two-way interaction ($F[2.17,34.81] = 1.20$; $P > 0.05$). For F2, a significant main effect of PHASE ($F[3,48] = 7.96$; $P < 0.01$) was found; however, in this case, the main effect of GROUP also was significant ($F[1,16] = 6.96$; $P < 0.01$). The two-way interaction in F2 was not significant ($F[3,48] = 0.485$; $P > 0.05$).

For the combined F2-F1, measure a significant main effect of PHASE ($F[2.25,36.12] = 16.90$; $P < 0.01$) and a significant main effect of GROUP ($F[1,16] = 9.12$; $P < 0.01$) was noted with no reliable interaction ($F[2.25,36.12] = 0.932$; $P > 0.05$). Hence, although both groups exhibited a change in speech output in response to the different phases of the auditory feedback manipulation (owing to the reliable effect of PHASE), the overall magnitude of the compensatory change in vowel acoustics was found to be smaller in the PD group compared with the control group.

An additional analysis was carried out to confirm whether the overall adaptation effect (F2-F1) at the end of the *hold* phase reliably changed from baseline for both groups. A one-sample *t*-test comparing the results from each group against a hypothesized mean of zero confirmed that, for each group, the adaptation effect was reliably different from zero (control group: $t[8] = 5.34$; $P < 0.01$; PD group: $t[8] = 4.39$; $P < 0.01$).

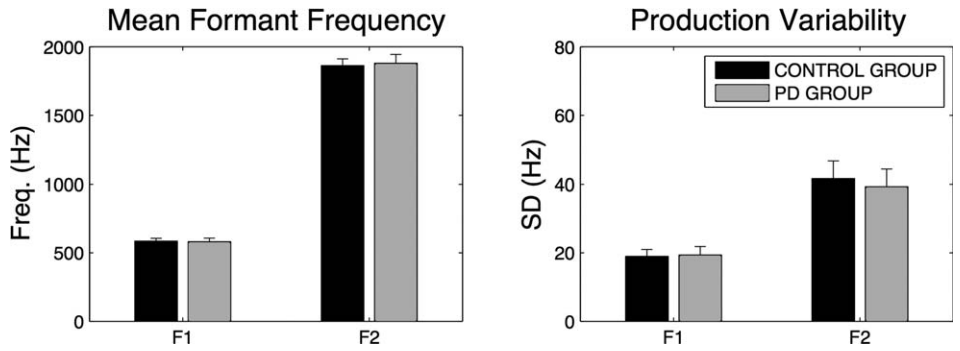

FIG. 2. The mean first formant (F1) and second formant (F2) frequencies (left) and standard deviation of the mean of F1 and F2 (right) are shown in Hertz for the baseline productions of the target vowel /ɛ/ for the Parkinson's disease (PD) group and the control group. SD, standard deviation.

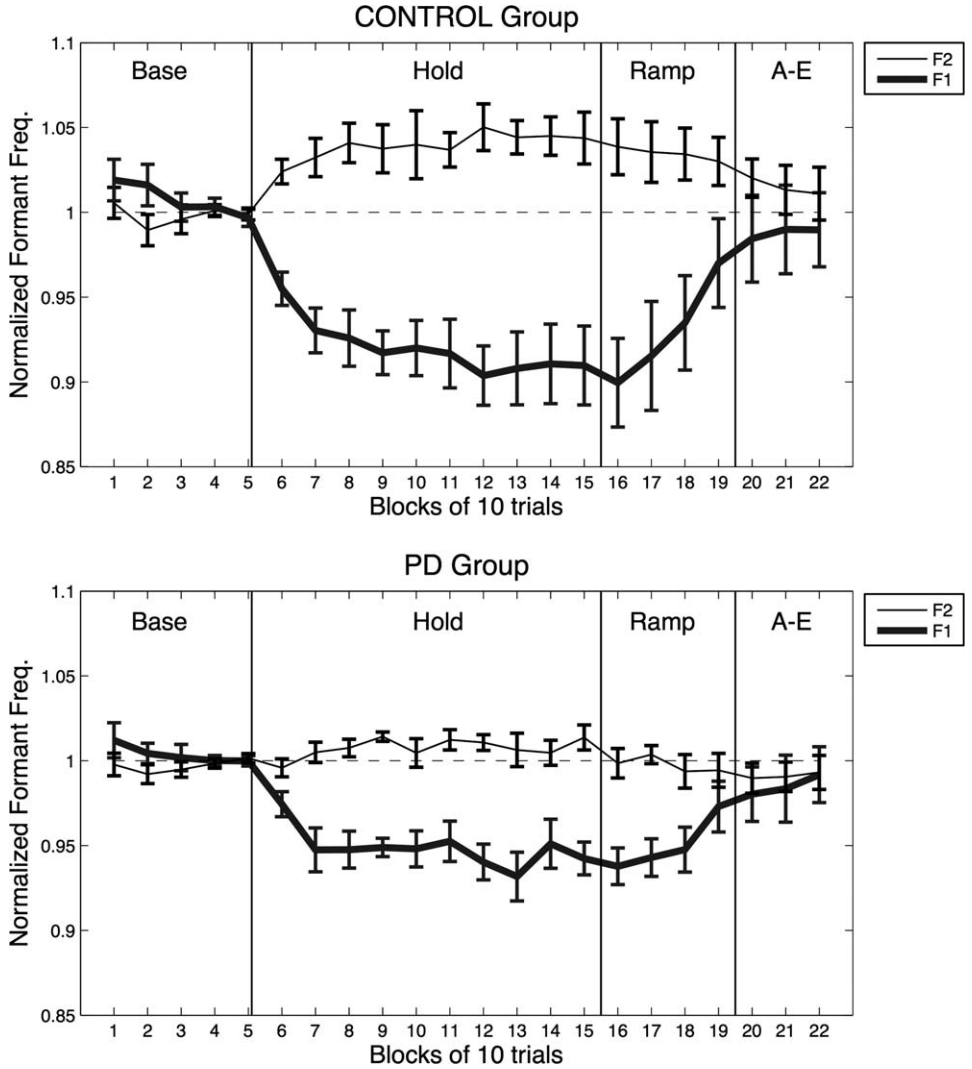

FIG. 3. Change in the acoustic vowel characteristics (first formant [F1] and second formant [F2]) for the target word throughout the speech adaptation testing sequence. Shown are blocks of 10 trials with onset of the feedback alteration beginning at trial block 6, followed by 100 trials (10 blocks) under maximum feedback alteration (hold phase), and then gradual (RAMP OFF) and subsequent complete restoration of unaltered feedback (after effect [A-E]). Freq., frequency.

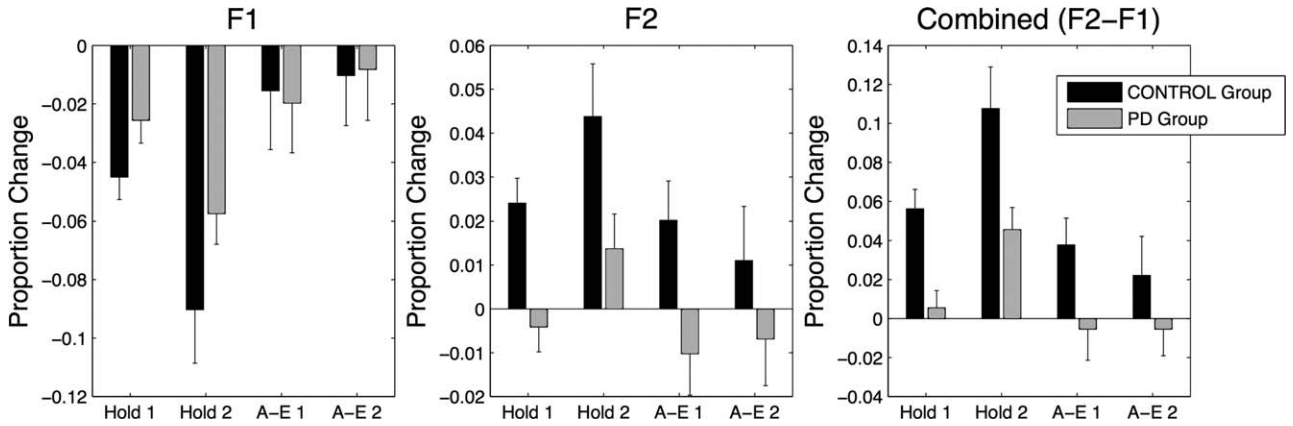

FIG. 4. Mean normalized formant values at the four time-points of interest during the speech adaptation procedure. See text for details. F1, formant 1; F2, formant 2; A-E, after effect; PD, Parkinson's disease.

A final analysis focused on the relationship between the degree of adaptation in F1 or F2-F1 and the degree of severity of either the perceptual or UPDRS clinical rating scores. No significant correlations were found for any of the perceptual speech scores (by subsystem or overall), but a significant negative correlation was found between the degree of F1 adaptation and the severity of motor symptoms as indexed by UPDRS scores ($r = -0.74$; $P = 0.011$).

Discussion

During speech production, sensory feedback is used both for real-time control and for sensorimotor learning. Here, we examined the manner in which PD, secondary to basal ganglia dysfunction, affected the process of sensorimotor integration and adaptation and, by extension, speech motor learning. The results of the present study illustrate that patients with PD are capable of adapting their speech articulation in response to a change in auditory feedback related to vowel formants; however, the result also demonstrated a clear reduction in the magnitude of sensorimotor adaptation relative to the control group. The current findings highlight a number of issues related to the speech production capacity of individuals with PD, the role of sensory information in speech, and the functions of the basal ganglia for speech.

Sensorimotor Interactions for Speech in PD

Overall, individuals in our PD group were able to adjust their motor output in a consistent and predictable way to offset the specific auditory manipulation. The manipulation in F1 frequency was accompanied in both groups by a reciprocal adjustment in F1 and F2 to partially offset the induced error. Thus, for both the control group and the PD group, the acoustic manipulation was mapped onto the appropriate articulatory/motor adjustment. Similar to results previously reported for both loudness and pitch perturbations[8] and delayed and frequency-altered auditory feedback,[30] individuals with PD maintain the functional ability to integrate sensory feedback with speech motor output. The change resulting from the disease, rather, appears to be related to the degree to which sensory feedback modulates or adjusts speech motor output.

Sensorimotor Adaptation in PD

The sensorimotor adaptation paradigm involves the introduction of a constant and predictable change in reafferent feedback to assess short-term sensorimotor learning. The current study is the first to evaluate motor learning in the speech domain in individuals with PD. The reduction in adaptation for the PD group is consistent with the role of the basal ganglia in sequential motor learning[31] and previous observations of dysfunction in learning new motor sequences in PD.[32,33] The present result is also consistent with prior findings of impaired sensorimotor learning in visually guided reaching movements in patients with PD relative to healthy controls.[34–36] Interestingly, the ability of the PD participants to adjust to the feedback alteration was related to their overall motor system involvement as measured by the UPDRS.

The reduced response to changes in auditory feedback associated with control of the oral articulators stands in apparent contrast to a recent study in which an enhanced laryngeal response was demonstrated after a perturbation in feedback related to loudness and fundamental frequency.[8] In that study, loudness and pitch feedback was altered suddenly and unexpectedly for brief periods during the production of sustained vowels in patients with PD and age-matched controls. After the manipulation, it was observed that the patients with PD exhibited larger compensatory changes in loudness and fundamental frequency than the healthy controls.

There are two possible explanations for the different findings. One is that the auditory feedback mechanisms contributing to loudness, vocal pitch, and articulatory control may be differentially impaired in PD. However, it is well known that increasing loudness is accompanied by changes in articulation, and this relationship is a hallmark of one of the more effective behavioral treatments for PD (the Lee Silverman Voice Treatment or LSVT).[37,38] What is more likely is that all of these parameters (loudness, pitch, articulation) are all affected in some complex way rather than all being independently controlled.

A perhaps more parsimonious explanation is that the tasks used by Liu et al and our adaptation task tap into different sensorimotor processes. Liu et al studied the immediate response to an unexpected and intermittent auditory feedback change during the production of a sustained vowel. Such a manipulation can be used to probe the control system (the operation of the participant's "internal model") without examining changes to the model (ie, no learning). In contrast, in the current study, we employed an approach well established in the literature for studying speech motor learning.[18,19,26,39–44] In this approach, a change in sensory feedback related to speech production is introduced and then maintained for a period of time, allowing participants, with repeated practice, to gradually alter their speech motor plans in order to reduce the perceived sensory error. This approach, therefore, focuses more on the role of auditory feedback in speech motor learning (updating and maintaining the accuracy of the internal model) rather than on the use of sensory input for the immediate correction of motor output.

A recent study provided some empirical evidence and arguments in support of separate cortical regions used for learning but not for control.[25] These two related but independent processes, sensorimotor control and sensorimotor learning, may be affected differentially in PD. For normal speech production, basal ganglia damage may amplify reafferent feedback, which, in patients with PD, creates the false perception of excessive loudness or effort. Under conditions in which speech motor patterns are modified (eg, during therapy), basal ganglia damage may increase the threshold for reafferent feedback, reducing the overall system gain and increasing the need for intensive and extensive training.

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