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Haptic feedback from manual contact improves balance control in people with Parkinson's disease

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Abstract

Parkinson's disease (PD) degrades balance control. Haptic (touch and proprioception) feedback from light contact with a stationary surface inadequate to mechanically stabilize balance improves balance control in healthy people. In this study we tested whether PD impairs use of haptic cues independent of mechanical support to control balance. We measured postural sway in thirteen individuals with PD (H&Y 1–3, median=2, Q₁=2, Q₃=2) and thirteen age-matched controls balancing in a widened, sharpened Romberg stance in four conditions: eyes-closed, no manual contact; eyes-closed light-touch contact (<1N), eyes-closed, unrestricted contact; and eyes-open, no contact. To determine whether PD-severity affects any of these balance strategies, PD participants were tested on- and off-medication, and using the more- and less-affected body side in the stance and manual contact.

Individuals with PD simultaneously maintained non-supportive fingertip contact and balance in this task without practice. PD participants swayed more than control participants (ML CP $p=0.010$; shoulder $p<0.001$), but manual contact reduced sway. Non-supportive manual contact stabilized balance more than vision ($p<0.05$). PD-severity factors had no significant effect ($p>0.05$).

We conclude the effect of PD on balance is not specific to vision or haptic feedback. Nevertheless, haptic cues from manual contact, independent of mechanical support, improve balance control in individuals with PD. We discuss the implication that PD or associated dopaminergic pathways do

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There is no conflict of interest for this paper:

PD impairs balance even in the presence of visual or haptic feedback
 Balance of people with PD is enhanced by haptic feedback
 Participants with PD simultaneously maintained non-supportive touch and balance
 PD severity factors (medication and body side) did not significantly affect balance
 PD does not affect the use of haptic cues to control balance per se

not directly affect haptic feedback balance control mechanisms, including arm/posture coordination and proprioceptive integration.

Keywords

Parkinson's disease; balance; touch; postural control; proprioception

INTRODUCTION

Leaning on canes, walkers, and banisters increases stability by increasing the base of support. However, touching rather than leaning on external objects can stabilize posture by conferring sensory feedback about body orientation through haptic (touch and proprioception) modalities^[1–3]. This is demonstrated by touching a stable surface with one fingertip at forces too small to add significant mechanical stability ($<1\text{N}$; “light-touch”)^[1]. It has been shown that light touch improves postural stability by increasing postural tone and enabling a local frame of spatial reference^[4].

Cane usage improves fall recovery in people with PD^[5], who are often at increased risk of falling^[6, 7]. However, since canes provide mechanical support, it is unclear whether people with PD can use haptic feedback to control balance. Several earlier findings suggest it might. Haptic feedback improves voluntary movement accuracy^[8,9]. Fine grasp control similar to fingertip control in light-touch is intact in PD^[10]. Instructing individuals with PD to adopt an external sensory focus (concentrating on the surface on which they stand, as opposed to their feet) improves balance^[11]. However, two important issues suggest this balance strategy is problematic for people with PD. Proprioception, critical to correctly interpret tactile cues in this task^[12], is degraded in PD^[9,13–15], and controlling the fingertip might distract from and degrade simultaneous balance control in individuals with PD^[16, 17].

To determine whether individuals with PD can utilize and benefit from haptic feedback to improve balance, we tested whether their balance improves while using haptic feedback from non-supportive fingertip. To determine whether PD-severity affects mechanisms underlying postural control with haptic feedback we compared, within-participants, performance with versus without medication, and balance with manual contact using the more- versus the less-symptomatic hand.

METHODS

Participants

Thirteen participants with PD (9 male; 9 fell in last 6 months; ages 60–84, mean=72.2 $\pm$ 7.1; H&Y 1–3, median=2, $Q_1=2$, $Q_3=2$; Table 1), and thirteen controls (9 male; ages 78–59, mean=72.1 $\pm$ 5.9; 2-tailed $t(24)=-.682$, std error difference=2.71, $p=0.39$, partial eta squared=0.19) participated. Participants with other chronic neurological diseases, arthritis, dementia, tactile perception threshold $>2\text{g}$, two-point discrimination threshold $>3\text{ mm}$, Columbia modified Mini-Mental State Examination score $<40/57$, upper extremity weakness, and orthopedic problems were excluded. Participants with PD were tested 12h off-medication and 1–2h on-medication.^[18] Effects of medication were demonstrated by significantly higher UPDRS, motor scores when off-meds (30.38) compared to on-medication (26.46, paired sample $t(12)=-3.71$, std error=1.06, $p=0.003$, partial eta squared=0.534, Table 1). The more PD-affected side was determined using items 23–26 of the UPDRS motor scale. All procedures were approved by the institutional review board of NYCOM.

Experimental task

Participants stood as still as possible for 25s in a modified sharpened Rhomberg stance (a tandem stance widened by a 10 cm medial-lateral gap). This posture was destabilizing medial-laterally but could be performed by all participants without loss of balance.

Experimental task conditions

The four experimental task conditions included three conditions with the eyes closed: (1) eyes-closed, no manual contact; (2) eyes-closed, unrestricted manual contact; (3) eyes-closed, restricted “light-touch” fingertip contact (during which participants were reminded not to lean on the touch plate by a buzzer which sounded when contact forces exceeded 1N); The final condition (4) was with eyes open, no manual contact, to determine relative effectiveness of vision and touch. Non-touching arms were relaxed by the side.

Effect of disease severity

Since PD typically presents asymmetrically, we compared performance during conditions with manual contact of the more- vs. the less-affected hand. The front and back feet co-varied with the touching hand since the greater portion of body weight—and therefore the greater burden of control—resides on the rear foot. Thus, no-manual contact condition trials were repeated with each foot in back, making 8 conditions per experimental session.

Effect of dopaminergic therapy

To address how dopaminergic medication affects balance control with haptic feedback, participants with PD were tested during and prior to medication. To control for potential order effects, six participants with PD were assigned randomly to testing initially in the OFF-medication state (12h following medication); seven initially ON-medication (1–2hrs following medication).

Apparatus and measurements

Center of pressure (CP): Participants stood with each foot on separate floor-mounted force plates (120Hz; AMTI, Watertown, MA) which measured CP movement. The overall CP was calculated from data from both plates. Manual contact forces: Participants touched a 6”×6” force plate (120Hz; AMTI) adjusted to waist height measured contact forces and center of pressure of the fingertip during manual contact trials. Real time force plate signals triggered computer beeps to signal when contact force exceeded non-supportive levels (>1N).

Postural sway kinematics

A seven-camera motion analysis system (60Hz; Vicon, Centennial, CO) recorded postural sway using reflective markers at the touching shoulder lateral acromion. Calibrated 3D data was exported from Vicon, low-pass filtered (60Hz) and analyzed with Matlab.

Protocol

Prior to balance tests, we administered the UPDRS motor subscale, the Columbia modified Mini-Mental State Examination^[19], and determined sensory thresholds and two-point discrimination of the fingertips. To determine the benefits of haptic feedback from manual contact independent of mechanical support, participants were asked to maintain fingertip contact at non-supportive levels. Prior to experimental testing participants probed the touch plate with the alarm activated to appreciate the required non-supportive manual contact levels without task-specific practice (with eyes-closed or modified stance).

One trial of each condition was tested to observe effects of conditions independent of practice effects and to minimize fatigue. Balance was tested without shoes. Foot placement was marked on the force plate for consistency. Participants stepped onto the floor-mounted force platform, were informed of the condition, and assumed the appropriate stance and manual contact mode. To control for possible fatigue participants rested sitting as long as desired between trials, and condition order was counter-balanced.

Analysis and interpretation

The manual control of individuals with PD and healthy controls during trials with manual contact was characterized by contact force magnitude (the mean resultant force magnitude) and the fingertip contact location spatial stability (the mean absolute deviation from mean position).

Postural sway and CP movements during quiet stance reflect unintentional drift and intentional postural corrections^[20]. As smaller postural sway and CP amplitude is associated with more efficient postural control^[1,12], we calculated the mean amplitude (the mean unsigned deviation from the mean position) of medial-lateral (ML) and anterior-posterior (AP) CP and body sway at the shoulder. If haptic feedback benefits postural control, then sway amplitudes with light-touch would be smaller than without touch.

Statistical analysis

A 4×2×2 repeated-measures ANOVA tested the significance of effects of *feedback modality* (eyes-closed without manual contact; eyes-closed unrestricted contact; eyes-closed light-touch; vision without manual contact), body side involved (less-, more-affected foot back), and medication (on-,off-) on postural stability measures (CP, shoulder) of individuals with PD. As the effects of body side and medication were not significant (see Results), results of participants with PD within the four experimental task conditions were collapsed across side and medication and compared to healthy controls in a split-factor ANOVA, with participant group (PD vs. control) as the between-group factor to determine the significance of effects of *feedback modality* (experimental conditions).

Tukey post-hoc pair-wise comparisons evaluated individual condition differences. A split-factor ANOVA with participant group (PD vs. control) as the between-group factor evaluated effects of touch-condition (unrestricted touch, light-touch) on fingertip forces and position control.

RESULTS

Individuals with PD stabilized touch contact levels <1N while balancing

The average fingertip contact location amplitudes of individuals with PD were 0.34 cm (+/-0.45) during unrestricted manual contact and 0.7 cm (+/-0.61) while touching lightly. This was not statistically different from control participants (0.07 cm; +/-0.08 and 0.58 cm +/-0.45 respectively, $p=0.153$). The mean fingertip contact force level of the PD participant group was 0.36N (+/-0.17) when asked to use light-touch. The mean unrestricted manual contact force of individuals with PD (4.4N +/-2.2) was greater than the restricted force ($p<0.001$), but less than that of control participants (9.4N +/-6.0, $p=0.042$).

Individuals with PD sway more than control participants; both groups benefit from light-touch

Participants with PD swayed more than control participants (ML: CP $p=0.010$, shoulder $p<0.001$; AP: CP $p=0.109$, shoulder $p<0.001$). Main effects of feedback condition were

significant on all sway measures (ML and AP CP, ML shoulder $p < 0.001$) except AP shoulder ($p = 0.277$). There were no significant group $\times$ condition interactions.

Pair-wise comparisons in ML CP and shoulder sway were significant ($p < 0.05$) between all feedback conditions except between ML shoulder during light-touch and leaning ($p = 0.491$). ML mean sway amplitude of individuals with PD with eyes closed was 0.58 cm (± 0.27) (CP); 0.90 cm (± 0.65) (shoulder). Visual feedback (eyes open-no touch) reduced ML sway to 0.40 cm (± 0.18) (CP) and 0.65 cm (± 0.16) (shoulder). Leaning (eyes closed-unrestricted touch) reduced ML sway to 0.16 cm (± 0.07) (CP) and 0.40 cm (± 0.18) (shoulder). ML mean sway amplitude with light-touch (eyes closed-restricted touch) was 0.25 cm (± 0.06) (CP) and 0.44 cm (± 0.39) (shoulder).

Pair-wise comparisons of AP CP were significant ($p < 0.05$) between all conditions except vision and light-touch. AP CP mean sway amplitude of individuals with PD with eyes closed was 6.9 cm (± 7.2). Non-supportive manual contact significantly reduced AP CM sway to 1.8 cm; ± 1.0), as did leaning (1.0 cm; ± 0.9) and vision (3.2 cm, ± 0.8).

Although dopaminergic medication significantly impacts motor function in the individuals with PD (Table 1), there was no significant effects of dopaminergic therapy on CP amplitude (ML: $F = 0.982$, $p = 0.360$; AP: $F = 1.105$, $p = 0.341$), shoulder amplitude (ML: $F = 0.965$, $p = 0.352$; AP: $F = 1.853$, $p = 0.232$), fingertip contact force (ML: $F = 0.597$, $p = 0.465$) and fingertip contact location amplitude (ML: $F = 2.092$, $p = 0.176$). Likewise, there were no significant effects in individuals with PD of more- versus less-affected side bearing more weight and touching: CP amplitude (ML: $F = 0.020$, $p = 0.893$; AP: $F = 0.325$, $p = 0.593$); shoulder amplitude (ML: $F = 0.263$, $p = 0.621$; AP: $F = 0.890$; $p = 0.389$); fingertip contact force ($F = 4.373$, $p = 0.075$); fingertip contact location amplitude ($F = 0.032$, $p = 0.861$).

DISCUSSION

Our findings demonstrate that individuals with PD benefit from haptic sensory feedback to control balance and reduce postural sway. ML and AP CP sway and ML shoulder sway in individuals with PD was less with non-supportive fingertip contact than without. We interpret the null effect of condition on AP shoulder sway as a ceiling effect since the AP base of support and potential ankle/knee/hip leverage was very large in our test stance. Likewise, AP CP sway amplitudes, an order of magnitude larger than ML, may not represent a greater “instability” per se. Although postural sway of the PD participant group was consistently larger than control levels, the benefits conferred by non-supportive manual contact show that individuals with PD can utilize and benefit from sensory information from manual contact independent of mechanical support to control balance. Postural sway attenuation with haptic feedback was present immediately; there was no practice prior to running trials. Likely, participants spontaneously adopted non-supportive manual contact since the cutaneous receptor peak sensitivity to force changes is $\sim 0.5\text{N}$ ^[21].

Null medication, affected side and group $\times$ condition effects suggest PD does not affect balance control with haptic feedback

Our results suggest that PD does not affect haptic-driven balance control mechanisms per se in two ways. First, the relative balance control quality among all conditions was the same for PD and control groups. Thus, PD balance control deficits may not be due to deficits in one particular feedback mode in the balance control context. Second are the null-effects of within-subject PD severity (medication and more- or less-symptomatic body side used for touching) on the outcome of using haptic feedback. That PD participants' balance improved with haptic feedback suggests that optimal function of dopaminergic systems affected by PD is not required to improve balance with haptic feedback.

Haptic feedback was equally beneficial on- and off-medication, and when using either the more- or less-symptomatic body side to touch while in the modified Rhomberg stance. Although the clinical significance of these null-effects is clear, the mechanistic significance is less obvious. The lack of difference of medication on balance control with haptic feedback may be related to the established phenomenon that postural instability is often unchanged by dopaminergic therapy^[22,23]. Although responses to discreet balance perturbations are more efficient during dopaminergic medication, overall stability is not improved^[22,23]. This might be because there are non-dopaminergic postural control pathways^[22] or because levodopa may degrade balance by reducing muscle tone^[23].

The present null-effects of medication and body-side suggest that the integration of proprioception and tactile feedback in this task is independent of dopaminergic pathways affected by levodopa. Previous reports of dopaminergic medication effects on proprioception in PD differ widely. Dopaminergic therapy has been reported to affect proprioception in PD positively^[14], negatively^[24,25], and indifferently^[15]. Thus dopaminergic therapy could appear to improve proprioception by acting on dopaminergic pathways affected by PD. Alternatively, further impairment of proprioception by dopaminergic therapy suggests a side-effect along non-Parkinsonian dopaminergic pathways. Our results suggest that the integration of proprioception with tactile cues in the context of balance is independent of either pathway underlying proprioceptive deficits related to PD, or proprioceptive pathways impaired by medication.

The null-effect of body side suggests that balance with light-touch may involve bilaterally coordinated mechanisms, or circuitry not directly involved with the substantia nigra. Similarly, axial hypertonic asymmetry, which contributes to postural impairments in PD, correlates with the UPDRS but does not correlate with limb asymmetry and is not affected by levodopa^[25]. These differences might be explained by different neural circuits underlying axial and limb control, differentially affected by PD^[25].

PD and haptic balance control mechanisms: proprioceptive integration and coordination of balance and precision manual contact

Although haptic or visual feedback improved balance of individuals with PD, individuals with PD still swayed more overall. This may be related to several factors associated with PD for which haptic feedback cannot compensate, such as reduced postural muscle strength^[23], altered sensory integration, and impaired organization and scaling of postural reflexes during postural tasks^[6,26]. PD may degrade reflex scaling by saturating the underlying neural pathway^[26]. Although it is unlikely that haptic feedback directly affects reflex scaling or reduced muscle strength related to poor postural control, haptic feedback may at least improve control efficiency to function within a range below reflex saturation.

The relative improvement in balance of individuals with PD with haptic feedback suggests PD may not specifically affect haptic feedback balance control mechanisms. This is noteworthy in light of prior evidence of PD impairing proprioception. Proprioception is critical in “light-touch” balance control to distinguish fingertip skin deformation as due to either postural drift or arm adjustments^[12]. Proprioceptive movement control is impaired by PD^[9,13–15]. In particular, proprioceptive balance control^[27], and small amplitude movements such as maintaining constant arm positions^[28] are degraded in PD.

Also noteworthy is that any cost of simultaneous balance and precision touch control did not degrade balance in people with PD. It was unclear prior to our study whether maintaining ‘precise’ manual contact required to test haptic feedback independent of mechanical support would be so challenging as to degrade balance in individuals with PD, for whom divided attention correlates with falls^[16,17]. Secondary motor tasks such as thumb/finger opposition

degrades balance control in individuals with PD, particularly those with a history of falling, although not as much as secondary cognitive tasks^[17]. PD also diminishes manual/postural coordination: anticipatory postural reflex amplitudes are diminished^[29], and disrupts the temporal coordination of arm movement associated postural adjustments^[30].

It remains unclear whether simultaneous minimization of fingertip contact force and postural sway is a dual task in which arm and body control compete for attentional resources. Verbal reaction times suggest this is true for gait while using a walker^[31,32]. A dual task conception of balance with light touch is supported by the independent degrees of freedom of the body and the fingertip as demonstrated by different fingertip and shoulder amplitudes, and that the control of each can be independently disrupted^[12]. In the absence of observable performance costs, balance with light touch could be considered a low-cost dual task^[33]. Our results indicate that for individuals with PD, like healthy subjects, benefits from increased feedback from touch outweighed any possible costs of simultaneously controlling balance and the fingertip/sensor.

Alternatively, light-touch balance control has been conceptualized as a generalization of precision grip^[1,12], a reflex mechanism used to adjust grip force during hand movement or load adjustments. In this conception of postural control, haptic feedback is a whole-body motor-equivalence of manual precision grip control, and the body and arm are controlled together to stabilize the fingertip as one task^[34]. Insofar as people with PD perform well in precision grip tests^[10] and dual-task control is degraded in individuals with PD, the success of haptic feedback with individuals with PD support a generalized precision grip conception of light-touch balance control, rather than a dual-task conception.

Limitations of the present study—It is possible that less sway in individuals with PD is more a sign of rigidity than improved postural control^[35]. This has been observed and argued in the context of recovery from a perturbation, which we have not tested.

Our interpretation of intact “light touch” balance control in individuals with PD as successful execution of a low-cost dual task is limited. Our paradigm does not determine whether simultaneous balance and precision fingertip control is either a low-cost dual task which individuals with PD can perform without practice, or *not* a dual task.

Conclusions—We observed that although individuals with PD consistently have larger postural sway than healthy individuals, individuals with PD can improve balance with manual haptic feedback cues independent of mechanical support. PD severity factors such as medication and body side had no effect on this phenomenon. We interpret from this that balance mechanisms affected by PD are not specific to mechanisms related to individual sensory modalities.

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Figure 1. Representative trial data

XY plots of center of pressure and shoulder motion. 10-second excerpts of trials of 1 participant with PD off-medication using their more-affected side (top), on-medication (middle), and 1 control participant (bottom).

■ PD
□ control

Figure 2. Manual contact control participant group mean data

Mean fingertip contact outcomes. **(a)** mean resultant magnitude of fingertip contact forces (N). **(b)** mean fingertip contact location amplitude (cm). Results of the PD participant group have been collapsed across (non-significant) conditions of medication and affected side. For all graphs, filled bars represent individuals tested with PD. Unfilled bars represent healthy control subjects. Error bars are standard deviation ($n=13$). * indicates significant main effects ($p < 0.05$) on manual contact force **(b)** between light touch and unrestricted contact, and also between participant group within the light touch condition.

Figure 3. Amplitude of postural sway participant group mean data

Mean postural sway outcomes. **(a)** mean medial-lateral sway amplitude of the center of pressure (cm). **(b)** mean medial-lateral sway amplitude of the shoulder of the more involved arm (PD) (cm). **(c)** mean anterior-posterior sway amplitude of the center of pressure (cm). **(d)** mean anterior-posterior sway amplitude of the shoulder of the more involved arm (PD) (cm). Results of the PD participant group have been collapsed across (non-significant) conditions of medication and affected side. For all graphs, filled bars represent individuals tested with PD. Unfilled bars represent healthy control subjects. Error bars are standard deviation ($n=13$). * indicates significant main effects ($p < 0.05$) for participant group (both

CP, **a**, and shoulder, **b**), and between all conditions (CP, **a**), and all conditions except between light touch and manual contact unrestricted contact (shoulder, **b**).

Table 1

Description of subjects with Parkinson's disease.

age	sex	PD duration	H&Y	UPDRS motor section ^a on medication	UPDRS motor section ^a off medication	MM ^b	falls in last 6 mos.	side more involved	sensory threshold (g) ^c	two-point discrimination threshold (mm) ^c	medication ^d
84	m	15	2	37	39	29	yes	l	0.07	3.0	carbidopa (25 mg)/levodopa (100 mg), selegiline (5 mg)
78	f	5	2	20	22	29	yes	r	0.4	2.5	pramipexole ^e
78	m	7	2	24	31	28	yes	r	0.07	2.5	ropinirole Hcl (12 mg), carbidopa (25 mg)/levodopa (100 mg)
76	m	25	3	47	43	30	yes	r	0.4	2.0	pramipexole (25 mg), carbidopa (25 mg)/levodopa (100 mg), eldepryl (5 mg)
76	f	20	2	27	30	29	yes	r	0.4	1.0	carbidopa (25 mg)/levodopa (100 mg), rasagiline (25 mg)
75	m	8	3	31	34	29	yes	r	0.4	1.5	carbidopa (25 mg)/levodopa (100 mg), rasagiline (1 mg), amantadine (100 mg)
75	f	12	2	32	40	26	no	r	0.4	1.0	rasagiline (1 mg)
73	m	3	2	29	40	30	yes	l	0.4	1.0	carbidopa (25 mg)-levodopa (100 mg)
69	m	8	2	24	28	29	no	l	0.4	2.0	ropinirole Hcl (12 mg), carbidopa (37.5 mg)/levodopa (150 mg)/entacapone (200 mg)
68	m	5	1	12	13	30	no	l	0.4	1.0	rasagiline (1 mg), ropinirole Hcl (12 mg), carbidopa (25 mg)/levodopa (100 mg)
64	f	2	1	16	24	27	no	r	0.07	2.0	ropinirole Hcl (0.5 mg)
62	m	20	1	19	22	30	yes	r	0.4	3.0	carbidopa (37.5 mg)/levodopa (150 mg)/entacapone (200 mg), pramipexole (1 mg), selegiline (5 mg), rasagiline (0.5 mg), amantadine (100 mg)
60	m	7	2	26	29	30	yes	r	0.4	2.0	carbidopa/levodopa/entacapone ^e , pramipexole ^e , selegiline ^e

^aItems 18–31 on the UPDRS, representing the motor portion, with a possible maximum score of 108

^bColumbia modified Mini-Mental State Examination, with a possible maximum score of 57

^cmore involved hand

^dtaken 1 hour prior to testing ON-medication

^edosage not available