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Parkinson's disease shows perceptuomotor asymmetry unrelated to motor symptoms

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Abstract

Evidence of neglect symptoms in Parkinson's disease (PD) have been reported during visuoperceptual tasks and linked to side of disease onset. The goal of this study was to determine if perceptual asymmetry is also evident in perceptuomotor tasks without visual input. The task was to point to a remembered straight-ahead (SA) target in peripersonal space. During baseline pointing a bias left of SA was evident in PD patients and right of SA in healthy controls. To evaluate whether this was linked to a proprioceptive bias in PD, pointing during axial twisting of the trunk was tested. Axial rotation ($\pm 15^\circ$, $1^\circ/\text{sec}$) of the lower-body about shoulders fixed against rotation induced a non-veridical perception of upper-body rotation and lower-body stationarity. Pointing endpoints were shifted right of the actual SA during clockwise (CW) lower-body rotation and left of SA during counter-clockwise (CC) rotation, despite the fact that the shoulders and head were not rotated. In PD patients, endpoints relative to SA were shifted less during CW than CC rotation of the lower-body, whereas controls showed symmetrical pointing. Levodopa did not significantly change this bias. Both hands were tested in each subject and bias appeared regardless of hand used. Neither disease progression nor side of disease onset was linked to the direction or size of pointing bias. These findings suggest that PD manifests a contraction of left external hemispace relative to right hemispace, which affects generation and execution of motor commands throughout disease progression.

Keywords

hemi-Parkinsonism; hemineglect; sensorimotor integration; muscle tone

Introduction

Space is three-dimensional, homogeneous, continuous and boundless, however perceptual space does not always retain these properties. Examples of anisotropy of perceptual space are well documented in vision, where lengths presented in depth typically appear shorter than equal lengths presented in the frontoparallel plane [2,19]. Anisotropy along the left/right axis also has been given much attention. Pathological cases involving damage to right temporoparietal area can give rise to hemineglect. In classic hemineglect left visual space and sometimes the left body-half is ignored [3,6,11]. Furthermore, it's notable that hemineglect can be compensated by natural or artificial asymmetrical proprioceptive stimulation of the neck [16, 17,26], which reveals proprioceptive inflow can bias external spatial perception. In Parkinson's disease, proprioceptive asymmetry characterizes the earliest stages of the disease. Indeed, this asymmetry may underlie neglect-like symptoms found in PD [12,18].

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An extensive body of literature reports on visuospatial asymmetries in PD [9,12,14,18,27]. Some studies have also shown a correlation between direction of impairment and side of disease onset. These studies mostly employ visual and visuospatial tasks. Ebersbach and colleagues [10] found that left-affected PD patients (LPD) tend to visually explore right hemispace first. Harris and colleagues [12] reported that identical rectangles are perceived narrower in left hemispace for LPD, and during a line bisection task rightward bias is found [27]. Together this suggests at least a small, but reliable left-neglect for LPD.

Evidence of lateralized impairment in PD may also be found in everyday behaviors. A study which surveyed over eighty PD patients using a self-report questionnaire showed LPD report bumping into the left side of doorways more than right-affected PD [9]. Lee and colleagues [18] quantified this accident-prone behavior and concluded that LPD compress the visual representation of a doorway asymmetrically when judging body-scaled doorway aperture width. Though these findings provide convincing evidence for left-neglect in LPD, the presence of an asymmetry in visuoperceptual space may not translate directly to perceptuomotor space.

Evidence linking side of disease onset to perceptuomotor impairment is less clear. When visuoperceptual and perceptuomotor ability were both evaluated in the same hemiparkinson subjects, no relation between observed perceptuomotor impairment and side of disease onset was reported [14]. Conversely, visuospatial motor *imagery* and side of disease onset is linked [1]. Because the relation of these perceptual and motor asymmetries remain unclear, our goal was to further examine integration of motor and non-visual perceptual processing in PD by using a motor task that excluded visual feedback, since dopamine receptors in the retina show depletion and may underlie visual bias [5,12]. If perceptuomotor asymmetries exists, we can examine their link to side of disease onset, disease progression, and dopaminergic medication use.

Perceptuomotor skills can be evaluated by employing a standard straight-ahead (SA) pointing task. In order to perform this task, peripheral sensory inputs are centrally transformed into the egocentric coordinates from which actions originate. The equilibrium position of egocentric reference depends on symmetrical (sub-)cortical activity. Any disruption to this balance can be measured by a subject's indication of the mid-sagittal plane, which divides personal and extra-personal space into right and left [30]. Although pointing may be a good indicator of balanced neural activity, compensations, either central or peripheral, may obscure this during normal functional tasks. However, by shifting the relation of proprioceptive, motor, and remembered visual reference frames, asymmetries might be more readily revealed. Evidence from the classical hemi-neglect research of right brain-damage patients show one-sided stimulations such as right-ear vestibular [25] or rightward optokinetic [23] inputs worsen contralateral deficits. A directional bias in the relation between egocentric and exocentric reference frames can also be induced by rotating the lower-body relative to the upper-body without actually changing upper-body and head orientation. Fixing the shoulders relative to external space and rotating the lower-body about the subject's yaw axis has been shown to induce the perception of upper-body rotation relative to an immobile lower-body [15,21]. Because actual rotation of these body parts has been shown to influence the direction of pointing and saccades [17,26], we maintain the orientation of the shoulders, arms, and head fixed relative to external space. Pointing during a perceived shift in trunk orientation can be used to determine whether this illusory shift in the subjective SA is also used to generate motor outputs. We evaluate perceptual lateralization of peripersonal space in PD, by quantifying pointing behavior during both left and right rotations. Pointing bias can be compared to the side of disease onset, to disease progression, and to levodopa usage.

Materials and Methods

Subjects

Eight PDs and eight healthy aged-matched controls (CTL) were tested (age PD: 62.2 ± 11.0 years, duration of disease 8.8 ± 4.2 years; age CTL: 63.8 ± 10.3 years). PDs were all responsive to levodopa and were tested in two states: practical OFF-state (levodopa wash-out of more than 12 hours) and ON-state, about 1–1.5 hours after resuming levodopa at the usual dosage. The Unified Parkinson's Disease Rating Scale Motor Part (UPDRS) was 32.6 ± 14.6 (SD) when OFF-meds, which decreased significantly ($p < 0.001$) to 19.4 ± 13.7 when ON ($p < 0.001$). Hoehn and Yahr Stages ranged from 1.5 to 4 (OFF-meds) and 1 to 3.5 (ON-meds), which was significantly lower ($p < 0.05$). Patients were divided equally for side of disease onset. All subjects gave informed consent and experiments were conducted in accordance with the OHSU Internal Review Board regulations for human subjects' studies and the Declaration of Helsinki.

Protocol and procedures

Subjects sat on a flat, stable seat with no back support. Feet were supported by a footrest and placed symmetric to mid-line. A snug-fitting shoulder harness was donned and used as a firm fixation point between the subject and external frame. The external fixation point was a double-hinge which allowed shoulders to translate within normal sway boundaries, without allowing any shoulder rotation. Thus, the moveable fixation could not serve as a spatial reference and required subjects to maintain seated equilibrium, avoiding unnaturalness in the postural task. The seat was placed on a rotating platform, which rotated the seat and subject's lower-body, while upper-body fixation held the shoulders in a straight-ahead unrotated position. The platform was rotated at constant low velocity ($1.0^\circ/\text{s}$, $\pm 15^\circ$) about the yaw axis. Feet and hips rotated together with the seat, and the torso was subjected to torsion about fixed shoulders. Starts and stops of rotation followed an acceleration profile meant to reduce sensation of dynamic phases ($< 12^\circ/\text{s}^2$), which together with the low constant velocity created a tonic input, virtually eliminating phasic sensory inputs. Subjects sat in front of an earth-fixed table upon which a visible straight line (60cm long $\times$ 4mm thick) projected from a point orthogonal to the subject's fronto-parallel plane approximately 10cm above the naval. The task required subjects to point to this remembered earth-fixed SA line while blindfolded. The initial position of the hand was held comfortably next to the chest without touching the body or table. The arm returned to this position after each pointing motion without touching the body or table, minimizing extraneous tactile spatial cues. The motion was practiced several times with visual and experimenter feedback before testing. The initial position of the pointing arm was closely monitored and verbally corrected if it deviated. The unused arm was placed on the ipsilateral leg.

Subjects pointed to the remembered SA every 10 seconds as cued by an automatically timed auditory signal during each phase of the trial. The time of pointing was not limited, but subjects typically made a single, fluid pointing motion and placed their mark with ink on the table top. There was no feedback about pointing during the trial. Subjects pointed during five phases of lower-body rotation: 1) body unrotated for 90s, nine points (baseline), 2) body rotating, one point during rotation (CW ramp-up to 15° at $1.0^\circ/\text{s}$), 3) body held in the 15° rotated position for 30s, four points (plateau), 4) body returning to unrotated position, one point, 5) body unrotated for 50s, five points (post-rotation). Phases 2–5 are then repeated for CC 15° rotation. The order of CW and CC rotations was balanced across subjects. Both left and right hands were tested for all subjects in counter-balanced order.

Analysis of results was accomplished on a case-by-case basis, and by using group statistics. Repeated-measures or between-group ANOVA was performed where applicable with statistical significance set at $p < 0.05$. Angular displacement relative to the center of platform

rotation was used to determine pointing deviations, expressed in degrees of rotation relative to baseline pointing. Baseline (Phase 1) was established for straight-ahead pointing by taking the signed average of the pre-rotation points relative earth-fixed SA with at least three consecutive points with less than 2° of shift being required before Phase 2 was started. The average of all four points during the rotated plateau phase (Phase 3) determined the change in subjective SA. The difference between baseline and plateau averages constituted the perceived rotational shift in subjective SA. Positive values are CW-rightward and negative are CC-leftward relative to SA.

Results

One typical patient (LPD) OFF-meds is depicted in Figure 1 to represent mean results for all patients. A leftward bias relative to the remembered SA during baseline pointing is observed in Phase 1. During platform rotation, this patient (as with all subjects) reported the perception of a stable, immobile seat and a rotating upper-body relative to the remembered SA, despite the fact, that the seat and lower-body rotated with the platform and the upper-body was unrotated. When the lower-body was rotated CW (Fig. 1, upper row), pointing movements to remembered SA were shifted in the direction of lower-body rotation by an amount equal to or greater than lower-body rotation. The opposite occurred during CC lower-body rotation (Fig. 1, lower row). However, as the raw data from the patient in Figure 1 shows, the rightward shift in points after CW lower-body rotation (Phase 3) was significantly smaller than the leftward shift in points after CC lower-body rotation. This bias appears in all PDs, regardless of affected side, whereas pointing during CW and CC rotation is virtually equal in CTL. The bias in PD is unchanged by medication, hand used, or disease progression. These results are detailed below.

Asymmetry during baseline pointing

A systematic shift in straight-ahead pointing was found during the pre-rotated baseline condition (Phase 1) when comparing PDs to CTL. Initial bias was significantly shifted leftward in PDs OFF-meds ($F_{1,14}=9.28$, $p=0.009$) and ON-meds ($F_{1,14}=10.9$, $p=0.005$) relative to CTL. Average pointing deviations greater than 2SD (i.e. $>4^\circ$) were used as threshold for a non-zero baseline to evaluate each individual. CTLs tended to show rightward bias with 7/16 trials shifted more than 2SD right of SA, and only 2/16 shifted left. In total 12/16 trials (8 CTL, left and right hand = 16 trials) showed an average rightward shift during baseline pointing. Conversely, PDs showed a leftward bias in initial pointing direction. The combined results of eight PDs, left and right hand, ON and OFF meds ($8 \times 2 \times 2 = 32$ trials) showed 25/32 trials with an average leftward shift, with 12/32 being significantly shifted ($>2SD$) left and only 1/32 trials significantly shifted right. Hand used did not affect initial baseline bias ($F_{1,7}=0.06$, $p>0.10$, n.s.). Medication did not significantly affect initial biases ($F_{1,7}=2.38$, $p>0.10$, n.s.); an average leftward shift occurred in 11/16 trials when OFF-meds and 14/16 trials when ON-meds.

Asymmetry in pointing during rotation

A significant leftward pointing bias was found in PDs when rotated ($F_{1,7}=41.4$, $p<0.0005$), which was not evident in CTL (Table 1). Exposure to 15° of platform/lower-body rotation (Phase 3), induced subjects to perceive trunk rotation and make pointing movements that deviated in the same direction as platform rotation by an amount greater than 15°. The ANOVA shows average pointing bias in PDs during CC lower-body rotation was greater than during CW rotation. Only 7/32 trials showed the opposite bias. All subjects showed three out of four trials biased to the left, except one LPD who showed half. There was no difference between LPD and RPD, as determined by the main effect of side of disease onset ($F_{1,6}=0.06$, $p>0.10$) and its interaction with directional bias ($F_{1,6}=0.21$, $p>0.10$). Furthermore, three RPD patients accounted for three of the seven trials with a rightward bias mentioned above. No main effect

of medication ($F_{1,7}=0.61$, $p>0.10$) or interaction of medication and rotation direction ($F_{1,7}=0.42$, $p>0.10$) was found. Hand used did not affect bias ($F_{1,7}=0.32$, $p>0.10$).

Directional pointing dynamically updates

Deviation of endpoints relative to SA linearly updated as platform rotation amplitude increased. The gains of pointing ($\theta_{\text{Pointing}}/\theta_{\text{Platform}}$) made halfway through rotation (Phase 2) were not significantly different from gains at the static peak of rotation (Phase 3), both being approximately 1.3 ($p>0.10$). Additionally, the direction and size of initial baseline pointing bias and pointing bias during rotation were not significantly correlated ($p>0.10$) [OFF-meds: $r^2_{\text{LH}}=0.03$, $r^2_{\text{RH}}=0.15$; ON-meds: $r^2_{\text{LH}}=0.11$, $r^2_{\text{RH}}=0.39$], which suggests that rotational bias asymmetrically integrates sensory input dynamically and is not simply due to an initial static asymmetry. Furthermore, orientation updates relative to SA during rotation in a manner that scales with the amplitude of axial twisting. Pointing deviations are not simply generated during static conditions (Phase 1 and 3) as a binary “all-or-none” shift in SA, as Bayesian decision-making might suggest.

Disease progression does not predict pointing behavior

The relation of pointing bias to UPDRS score was tested. Correlations for each hand, both ON and OFF-meds and during baseline and rotational pointing were evaluated. Pointing bias did not correlate significantly ($p>0.10$) with UPDRS OFF-meds [Baseline: $r^2_{\text{LH}}=0.22$, $r^2_{\text{RH}}=0.08$; Rotational: $r^2_{\text{LH}}=0.02$, $r^2_{\text{RH}}=0.22$] or ON-meds [Baseline: $r^2_{\text{LH}}=0.09$, $r^2_{\text{RH}}=0.08$; Rotational: $r^2_{\text{LH}}=0.34$, $r^2_{\text{RH}}<0.01$]. Thus, no strong link is suggested between size or direction of pointing bias and disease progression.

Discussion

The current study examined the relation of motor asymmetries in PD to perceptual asymmetries during a perceptuomotor task. A left bias was found in baseline pointing and pointing during trunk rotation, which we interpret as a perceptuomotor asymmetry that appears when generating movements to interact with perceived external hemispace and results from left hemispace compression. This perceptual asymmetry was not linked to the side of disease onset, rather all PD patients showed the same directional bias. This difference from previous findings, which show correspondence between side of disease onset and direction of perceptual asymmetry [12,18] may be because those asymmetries were visuospatial, rather than perceptuomotor. Furthermore, pointing bias could not be predicted by level of disease progression, nor was it affected by medication.

One interpretation of the leftward pointing bias is that during CW rotation of the lower-body, subjects perceive a smaller rotation of the upper-body into left hemispace resulting in a smaller rightward pointing deviation. An asymmetry in perception of external space has been shown before in PD [12,18], however, it appeared during visual or visuospatial tasks. Our method excludes active retinal input, relying only on a remembered visual target, therefore our asymmetries cannot simply be due to visual receptor impairment. Rather, the impairment likely occurs during specialized cortical processing necessary for transforming proprioceptive coordinates into motor coordinates. In order to explain this, external and internal spatial reference frames should be distinguished from one another. In a recent study, internally and externally referenced transformations appeared to rely differentially on left motor and right parietal areas, respectively. Amick and colleagues concluded that parietal areas and frontostriatal motor pathways necessary for integrating visuospatial memory and motor imagery are impaired in PD [1].

Relating those findings to our study, both internally (upper relative to lower body) and externally (SA line relative to body) referenced transformations are invoked simultaneously. Because proprioception can be represented by an internal reference frame, a 15° rotation of axial musculature can be represented in internal coordinates relative to other body parts and/or externally with reference to the fixed world with which the body is in contact. Larger leftward pointing deviations suggest perceived CC-left upper-body rotation is smaller than perceived CW-right upper-body rotation. Thus, larger leftward pointing deviations represent a response to more largely perceived rightward upper-body rotation. However, during baseline pointing leftward bias is also observed in PD patients, even though no change in proprioceptive inputs has been evoked.

Although studies of left hemineglect using baseline straight-ahead pointing have proven difficult to explain since both leftward [7] and rightward biases have been found [13,24], we explain our baseline and rotational pointing biases together by assuming distortion in the representation of external space. An asymmetry may occur either during the transformation of symmetric (internal) proprioceptive signals into external spatial coordinates or after this transformation during generation of motor commands for interaction with external space. Our data do not distinguish between these two, but the end results are that movements through left external hemispace are smaller than movements through right external hemispace. More simply put, the straight-ahead is biased to the left due to compression of left hemispace. This explanation accounts for both baseline pointing biases and the pointing asymmetry that CW versus CC platform rotation induces.

To give a concrete example (see Fig. 1), when the platform is rotated 15° CW-right, the trunk is perceived to rotate left of SA. This perceived movement of the trunk results in approximately 17° pointing deviation right of baseline. Conversely, when the platform rotates CC-left and the trunk is perceived to rotate right, pointing deviates over 20° left of baseline. This suggests that larger pointing deviations to the left are because movement in right perceived external hemispace is larger than in left perceived external hemispace. During baseline pointing when actual and perceived external hemispaces are aligned, the left hand must travel through left hemispace and undershoots the SA, falling left of it. The right hand must travel through right hemispace and overshoots SA, also falling left of SA. Together this suggests that right hemispace is expanded into an under-represented/compressed left hemispace, so movements generated to move through right hemispace are larger than for left hemispace. Independent of whether PD patients misperceive the amplitude of proprioceptive inputs from axial rotation or generate motor responses disproportionately, a clear lateralization in scaling of movements exists.

It has been suggested that perceptual impairments in PD are largely due to motor impairments such as rigidity, bradykinesia, abnormal agonist/antagonist activation [20,28]. However, our findings provide more evidence that perceptuomotor impairments are not simply due to motor impairments. This is supported by the fact that neither initial side of disease onset, nor hand tested affects the direction of perceptuomotor bias. Although evidence from the literature exists supporting a correlation between the side of disease onset and perceptual impairments, it comes predominantly from paradigms employing visual and visuospatial tasks [10,12,18,27]. The literature provides convincing evidence for at least a degree of left-neglect in LPD patients, but asymmetry in visuoperceptual space does not appear to translate directly to asymmetry in perceptuomotor space. Inclusion of a motor task and exclusion of online visual input during trials may underlie this difference. One additional finding that suggests asymmetry is not due to motor impairment is that as the disease progresses, motor symptoms advance, however worsening motor symptoms did not increase the perceptuomotor asymmetry. However, the strength of these conclusions would benefit from running more patients, especially LPD.

In summary, we found that the *current representation* of the trunk, rather than the actual trunk-midline, is used to determine the relation between ego- and exocentric reference frames and to generate motor outputs in all subjects regardless of condition. In our sample of PD patients the subjective straight-ahead is biased, but the bias cannot be explained by the side of disease onset, disease progression, or the use of dopaminergic medication. We speculate that PD manifests a perceptuomotor asymmetry predisposed to one direction, much as classic hemineglect typically affects left hemisphere. Bilateral neural processes balance afferent sensory input from both sides of space and body to form representations referenced to the body midline. Both halves of the body are represented in each brain hemisphere, but the neural representation of the ipsilateral side is weaker than the contralateral representation [29,30]. We further speculate that PD may expose this hemispheric asymmetry independent of the motor-affected side of the disease by disrupting this equilibrium. The disruption makes the PD brain more dependent on the left brain's weakly represented ipsilateral hemisphere. Evidence from the classic hemi-neglect literature shows a double-dissociation between visuospatial neglect and somatosensory deficits [4,29], and between visuospatial and remembered spatial neglect [22], both of which may help explain why the currently observed hemiparkinson symptoms differ from those found in hemiparkinson visuoperceptual neglect.

Finally, evidence from patients with striatal lesions show that not only right basal-ganglia (BG) lesions but also left BG lesions can induce left-neglect [8]. This suggests that ipsi- and contralateral pathways exist which may predispose leftward asymmetry independent of the BG side affected. Combining this evidence with that of hemispheric specialization for spatial transformations, which overlap with striatocortical pathways affected by PD [1], it's not unreasonable to suppose behavioral asymmetry may be manifested differently depending on the BG-cortical loops activated by the visual and motor demands of the task. This may explain why others have found left-neglect in LPD more than RPD during visuoperceptual tasks [12, 18], while we find both groups affected equally during perceptuomotor tasks. In addition to running a larger sample of subjects to increase power, future studies may require brain imaging to correlate the side of nigrostriatal cell degeneration with asymmetrical cortical activity in order to explain perceptuomotor and visuoperceptual hemiparkinson sequelae.

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Figure 1.

The subject is instructed to point to the remembered earth-fixed straight-ahead line (thick gray line). Left column: Lower-body rotation (black arrow) is depicted as CW (top) or CC (bottom), while trunk orientation (dashed line) is fixed relative to SA. Middle column: The subject's perception of trunk rotation (dotted line) relative to SA, with perceived endpoints landing directly on SA line. Right column: Raw data from one representative LPD patient OFF-meds when tested with the left (triangles) and right (diamonds) hands. The five phases are described in the protocol section.

Table 1

Average pointing angle in degrees relative to the earth-fixed straight-ahead or baseline

Subjects	Baseline			Seat rotation direction					
	Unrotated			CC			CW		
	LH	RH	Mean	LH	RH	Mean	LH	RH	Mean
PD-off	-2.6 (1.2)	-2.4 (1.5)	-2.5 (1.3)	-19.1 (0.7)	-22.3 (1.9)	-20.7 (0.9)	18.4 (0.9)	15.7 (2.1)	17.0 (1.1)
PD-on	-4.1 (2.0)	-4.9 (2.0)	-4.5 (2.0)	-19.1 (3.2)	-16.9 (1.9)	-18.0 (2.3)	16.3 (2.2)	15.0 (3.4)	15.6 (2.9)
CTL	0.5 (1.6)	4.3 (1.3)	2.4 (1.5)	-25.5 (2.5)	-20.2 (2.4)	-22.9 (1.7)	25.6 (2.6)	19.2 (1.7)	22.4 (1.6)

Standard errors in parentheses.

CW is positive and CC is negative. LH – left hand, RH – right hand.