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Veering in hemi-Parkinson's disease: Primacy of visual over motor contributions

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ABSTRACT

Veering while walking is often reported in individuals with Parkinson's disease (PD), with potential mechanisms being vision-based (asymmetrical perception of the visual environment) or motoric (asymmetry in stride length between relatively affected and non-affected body side). We examined these competing hypotheses by assessing veering in 13 normal control participants (NC) and 20 non-demented individuals with PD: 9 with left-side onset of motor symptoms (LPD) and 11 with right-side onset (RPD). Participants walked in a corridor under three conditions: eyes-open, egocentric reference point (ECRP; walk toward a subjectively perceived center of a target at the end of the corridor), and vision-occluded. The visual hypothesis predicted that LPD participants would veer *rightward*, in line with their rightward visual-field bias, whereas those with RPD would veer *leftward*. The motor hypothesis predicted the opposite pattern of results, with veering toward the side with shorter stride length. Results supported the visual hypothesis. Under visual guidance, RPD participants significantly differed from NC, veering leftward despite a shorter right- than left-stride length, whereas LPD veered rightward (not significantly different from NC), despite shorter left- than right-stride length. LPD participants showed significantly reduced rightward veering and stride asymmetry when they walked in the presence of a visual landmark (ECRP) than in the eyes-open condition without a target. There were no group differences in veering in the vision-occluded condition. The findings suggest that interventions to correct walking abnormalities such as veering in PD should incorporate vision-based strategies rather than solely addressing motor asymmetries, and should be tailored to the distinctive navigational profiles of LPD and RPD.

1. Introduction

Parkinson's disease (PD) is a neurodegenerative disorder, the typical motor symptoms of which include resting tremor, bradykinesia, postural instability, freezing of gait, shuffling gait pattern, rigidity in the trunk and limbs, reduced pelvis rotation, and lack of arm swing, all of which put people with PD at a high risk of falling (Bloem, Boers, Cramer, Westendorp, & Gerschlager, 2001; Schaafsma, Balash, et al., 2003; Schaafsma, Giladi, et al., 2003; Wood, Bilclough, Bowron, & Walker, 2002). Non-motor features of the disease have also been identified. In the visual domain, these include changes in basic visual functions such as contrast sensitivity, motion and optic flow perception, color discrimination and visuospatial perception (Archibald, Clarke, Mosimann, & Burn, 2011; Bodis-Wollner, 1990; Bodis-Wollner et al., 1987; Brandies & Yehuda, 2008; Davidsdottir, Cronin-Golomb, & Lee, 2005, Davidsdottir, Wagenaar, Young, & Cronin-Golomb, 2008; Harris, Calvert, Leendertz, & Phillipson, 1990; Uc et al., 2005).

A current view is that the role of vision in spatial navigation includes not only perceiving the layout of the world, but also, importantly, controlling one's movement. Absence of proper visual inputs has long been acknowledged as a critical risk factor for falls especially for people with visual impairment due to neurological disorders or normal aging (e.g. Hafström, Fransson, Karlberg, Ledin and Magnusson, (2002), Lee and Scudds (2003), Perrin, Jeandel, Perrin and Béné (1997)). This proposition has not typically been applied to PD, because the disease was traditionally characterized as a motor disorder rather exclusively, with the focus of rehabilitation research directed at interventions targeting the motor symptoms. Davidsdottir and colleagues reported that visual and visuospatial impairments were prevalent in a sample of 81 individuals with PD, with visual hallucinations, double vision and

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contrast sensitivity deficits being associated with freezing of gait (Davidsdottir et al., 2005). Although visual processing is impaired, there is increased dependence on vision in PD for postural control (Azulay, Mesure, Amblard, & Pouget, 2002) and for gait regulation while walking (Morris, Iansek, McGinley, Matyas, & Huxham, 2005). Therefore, advancing our understanding of the non-motor symptoms of PD such as deficits in visuospatial processing, as well as their potential contribution to locomotive disability, is a pressing need in the field.

PD almost always has unilateral onset due to the underlying hemispheric neuropathology, and this laterality is reflected in the difficulties that people with PD commonly endorse in regard to navigating in space (Davidsdottir et al., 2005). During spatial navigation tasks, veering (lateral deviation from a straight or intended path) in PD has been measured quantitatively; persons with LPD veered rightward in the presence of visual input, whereas persons with RPD veered leftward (Davidsdottir et al., 2008; Young et al., 2010). This finding echoes the different profiles that LPD and RPD display on visual perception tasks, including horizontal line bisection (Davidsdottir et al., 2008; Laudate, Neargarder, & Cronin-Golomb, 2013; Lee, Harris, Atkinson, & Fowler, 2001a, 2001b), copying and drawing tasks (Shelton, Bowers, & Heilman, 1990; Vallar, 1998), self-report of daily visual function (Davidsdottir et al., 2005), reaching and grasping tasks (Rossit et al., 2012), body-scaled aperture estimation (Lee et al., 2001a, 2001b), and size perception comparison in two hemi-spaces (Harris, Atkinson, Lee, & Nithi, 2003; Milner & Harvey, 1995). Overall, individuals with LPD exhibit a rightward spatial bias, perceiving stimuli as shorter or smaller on the left than the right. By contrast individuals with RPD perceive visual stimuli more like healthy control adults, who have been reported to bisect lines slightly to the left ("pseudoneglect") (Jewell & McCourt, 2000). It appears that the consequences of right hemisphere damage (LPD) contribute to more severe visuospatial impairments than damage to the left hemisphere (RPD), as the right hemisphere mediates more visuospatial processing than the left in the general population and also in PD (Cronin-Golomb, 2010).

Asymmetry of symptoms in PD also influences the dynamics of sensorimotor coordination (Boonstra, van der Kooij, Munneke, & Bloem, 2008; Frazzitta, Pezzoli, Bertotti, & Maestri, 2013; Lin et al., 2014; Nanhoe-Mahabier et al., 2011; Plotnik, Giladi, Balash, Peretz, & Hausdorff, 2005; Yogev, Plotnik, Peretz, Giladi, & Hausdorff, 2007). Individuals with PD typically have less stable and more asymmetric gait patterns during locomotion, with shorter stride length on the initially affected body side than on the secondarily affected body side (Lin et al., 2014; Plotnik et al., 2005; Young et al., 2010). Although no conclusive association has been drawn between motor asymmetry and veering, the difference in stride length between body sides has been offered as an explanation (Guth & Laduke, 1994). Previous veering studies indicated that those with LPD veered rightward, whereas those with RPD veered leftward during normal walking, corresponding to the hemisphere with presumed lower dopamine levels and greater neuropathology (Davidsdottir et al., 2008; Young et al., 2010).

Whether the source of veering in PD is more attributed to errors in visuospatial perception or to asymmetry of motor features has not been addressed directly. These two potential mechanisms provide contradictory predictions for veering direction. If veering is primarily driven by asymmetrical walking patterns expected in PD between the relatively affected and relatively non-affected body side, a tendency to veer towards the side of body that has relatively shorter step length would be observed regardless of whether they walked with eyes open or vision occluded, i.e., LPD would veer leftward, whereas RPD would veer rightward. On the other hand, if veering is driven by visuospatial bias (as seen in mild hemineglect), veering should be shifted in the opposite direction, with LPD veering rightward and RPD veering leftward, as reported in the studies of Davidsdottir et al. (2008) and Young et al. (2010)—but these studies did not example stride asymmetry. The visuospatial bias might be observed especially when participants were asked to walk towards the self-perceived center of a horizontal line placed at the end of the corridor. The resulting visuospatial shift of the egocentric midline in PD would come into play: LPD would generate rightward error on perceiving the center of the bar, resulting in a rightward veering trajectory, and a similar (but leftward) effect would be expected in RPD, although the size of the bias would be expected to be smaller because the influence of right hemisphere dysfunction on visuospatial perception is greater than that of the left. Our goal was to assess directly whether visuospatial bias or motor bias accounts better for lateral drift in individuals with LPD and RPD.

2. Method

2.1. Participants

The study included 20 non-demented individuals who had been diagnosed with idiopathic PD (11 men, 9 women) and 13 normal control adults (NC; 4 men, 9 women). The distribution of men and women did not differ between the PD and NC groups ($\chi^2 = 1.87$, $p = 0.17$). The PD participants were recruited from the Parkinson's Disease Clinic at the Boston Medical Center and from the Fox Foundation Trial Finder. The NC group was recruited from the Fox Trial Finder and the local community. Participants underwent health history screening prior to taking part in the study. Exclusion criteria included the inability to ambulate independently or history of musculoskeletal impairments or pain condition; lower extremity impairments that prevented free movement; use of walking assistive devices; coexistence of serious chronic medical illness; history of traumatic brain injury or stroke; psychiatric or neurological diagnoses (besides PD, in the PD group); surgery affecting the thalamus, basal ganglia, or other brain regions; history of alcoholism or other drug abuse; use of psychoactive medication except antidepressants or anxiolytics in the PD group; use of any psychoactive medication in the control group; presence of clinically significant eye disease, or corrected binocular acuity poorer than 20/40. Participants were screened for acuity binocularly at a distance of 10 ft using a Snellen chart; Snellen scores were converted to logMAR scores for the analysis. Mean acuity was −0.01 (20/16 Snellen) (SD = 0.07) for the PD group, and −0.09 (20/16 Snellen) (SD = 0.03) for the NC group. There was a significant group difference with NC showing better acuity ($t[26.1] = 4.21$, $p = 0.001$, $\eta^2 = 0.29$) but this is probably not of clinical significance, as both groups' acuity was very good. Initial analysis showed no effect of acuity on veering, and accordingly it was not considered in further analyses.

All participants were right handed except three of the PD group and one of the NC group, all of whom were left handed. We conducted separate veering analyses with and without individuals who were left handed and found that the results were not affected; therefore handedness was not considered further in the analyses. All participants were native English speakers. All were non-demented as indexed by their scores on the modified Mini-Mental State Exam (mMMSE; Stern, Sano, Paulson, & Mayeux, 1987), each obtaining 26.45 or better on conversion to standard MMSE scoring.

The PD group reflected mild to moderate stages of the disorder (stages 1–3 on the Hoehn and Yahr scale) (Hoehn & Yahr, 1967) (Table 1). The average disease duration was 4.7 years (SD = 4.0). Disease severity was determined with the use of the Unified Parkinson's Disease Rating Scale (UPDRS, 4 sections; Fahn & Elton, 1987; Levy, Louis, Cote, Perez, et al., 2005). The PD group had a mean UPDRS total of 35.5 (SD = 14.5) denoting mild-moderate disease severity, with a mean motor score of 21.2