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WALKING IN CIRCLES: NAVIGATION DEFICITS FROM PARKINSON'S DISEASE BUT NOT FROM CEREBELLAR ATAXIA

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Abstract—Little is known on the role of neuronal structures for spatial navigation. Our goal was to examine how Parkinson's disease (PD) and cerebellar ataxia, as human lesion models of the basal ganglia and cerebellum, affect spatial navigation round a circular walking path, blindfolded. Twelve subjects with idiopathic PD (ON and OFF medication), eight subjects with cerebellar ataxia and a control group of 20 age-matched healthy subjects participated. All groups performed well when walking around the circle with eyes open. In the eyes-closed condition, control subjects overshot the outlined trajectory but returned to their initial position, thus walking a further distance with eyes closed than with eyes open. When OFF medication, PD subjects navigated a larger radius than controls with eyes closed. When ON levodopa, PD subjects walked a similar distance as controls but with even larger errors in endpoint. Surprisingly, cerebellar patients navigated the circular walking task in the eyes closed condition with even more accuracy (i.e. following the outlined circle) than control and PD subjects. We conclude that blindfolded navigation around a previously seen circle requires intact basal ganglia, but not cerebellar input. **© 2011 IBRO. Published by Elsevier Ltd. All rights reserved.**

Key words: levodopa, kinematics, task performance and analysis, gait, navigation.

Navigation is a complex process requiring integration of both environmental (external) and self-movement (internal) cues. In blind navigation, environmental cues (e.g., visual, auditory, olfactory) are generally limited to a remembered target and/or path (Loomis et al., 1993; Wallace et al., 2010). In that case, self-motion cues (e.g., proprioception and vestibular) are the basis for navigating through the environment and are used to update an online representation of direction and distance travelled (Berthoz et al., 1995). Path integration (or dead reckoning) is a parallel process that operates on self-movement cues and results in an estimate of the direction and distance from the position where movement was initiated (Wallace et al., 2010). A number of studies (Loomis et al., 1993; Takei et al., 1996, 1997) have looked at the ability of humans to walk blindfolded around different path shapes (straight line, circular, triangular, etc.; (Pham and Hicheur, 2009). When healthy subjects walk blindfolded around a circular path, they consistently overshoot the ideal radius, undershoot the total angle and overshoot the total path length, independent of the size of the circle (Takei et al., 1997).

Currently, little is known about the role of neuronal structures for navigation. Systematic biases in processing of incoming somatosensory/sensory information may contribute to potential abnormalities in spatial navigation in subjects with Parkinson's disease (PD) and cerebellar ataxia (Bowen et al., 1972; Rondi-Reig and Burguière, 2005; Crenna et al., 2007). However, the contribution of basal ganglia and cerebellum in non-visual locomotor navigation is currently unknown. PD is a movement disorder in which visuospatial and kinesthetic awareness is affected in addition to the classic motor deficits of bradykinesia, rigidity, tremor and balance disorders (Amick et al., 2006). Difficulty with somatosensory kinesthesia has been proposed to be responsible for undershooting of reaching targets in patients with PD (Demirci et al., 1997; Konczak et al., 2007; Wright et al., 2007a). It has been hypothesized that these kinesthesic deficits may also be responsible for undershooting walking distance and particular difficulties with making turns while walking (Crenna et al., 2007; Wright et al., 2010). Damage to the cerebellum not only results in ataxia (hypermetric stepping and lateral postural sway while walking), it may also affect the structural network involved in spatial navigation such as the spatial representation of the environment and adapting locomotion to a specific context (Petrosini et al., 1998; Rondi-Reig and Burguière, 2005).

Thus, the aim of this study was to compare distance and rotational error when walking around a remembered circular path without visual feedback in PD, cerebellar and

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Abbreviations: ANOVA, analysis of variance; ASIS, anterior superior iliac spines; CA, cerebellar ataxia; CCW, counter-clockwise; ctrl, control; CW, clockwise; EC, eyes closed; EO, eyes open; IQR, interquartile range; OFF, off levodopa medication; ON, on levodopa medication; PD, Parkinson's disease; SARA, scale for the assessment and rating of ataxia; SCA, spinocerebellar ataxia; SD, standard deviation; UPDRS, unified Parkinson's disease rating scale.

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Table 1. Characteristics of the Parkinson's disease, cerebellar and control subjects

	Parkinson's disease		Cerebellar	
	Patients (n=12)	Controls (n=12)	Patients (n=8)	Controls (n=8)
Age (y)	64±9 (46–81)	64±9 (43–81)	58±7 (48–68)	57±6 (47–64)
Gender	12 M	12 M	2 M, 6 F	2 M, 6 F
Height (cm)	176±6 (165–185)	174±6 (165–183)	169±11 (154–185)	170±11 (150–183)
Weight (kg)	82±8 (68–94)	80±11 (62–99)	75±13 (64–100)	75±13 (64–99)
Duration (y)	6±4 (2–12)	—	5±3 (2–9)*	—
Motor UPDRS-ON	23±8 (14–41)	—	—	—
Motor UPDRS-OFF	32±10 (18–47)	—	—	—
Hoehn and Yahr ON	2.0±0.1 (2.0–2.5)	—	—	—
Hoehn and Yahr OFF	2.3±0.4 (2.0–3.0)	—	—	—
SARA	—	—	14±3 (9–18)	—

Values are mean±SD (range).

UPDRS, Unified Parkinson Disease Rating Scale; SARA, Scale for the Assessment and Rating of Ataxia.

* Excluding one subject with SCA-15 suspected, not confirmed genetically, affected for >20 y.

control subjects. The results from this study will enable us to better understand the contribution of basal ganglia and cerebellum for path integration.

EXPERIMENTAL PROCEDURES

Subjects

Twelve subjects with a clinical diagnosis of "idiopathic" PD, treated with levodopa, eight subjects with cerebellar ataxia and two respective control groups participated in the study. The subjects in the control groups had no prior history of neurological diseases. All subjects were screened with a health history evaluation to ensure that they were free of musculoskeletal and any other neurological impairments that could contribute to postural instability or movement dysfunction. The control subjects were matched for age, weight and height (see Table 1 for subject characteristics). All subjects were ambulatory and able to stand without an assisting device for the experiment. The PD subjects had no history suggesting "atypical" PD symptoms, as defined by Hughes et al. (1992) or other existing neuromuscular disorders, including severely flexed posture. PD subjects included in the study had Hoehn and Yahr scores of 2 or 3. Severity of cerebellar ataxia was assessed with the Scale for the Assessment and Rating of Ataxia (SARA) and scores are presented in Table 1. Three of the cerebellar subjects were diagnosed with idiopathic cerebellar ataxia, three subjects as spinocerebellar ataxia type 6 (SCA-6), one subject as SCA-15 and one subject as olivopontocerebellar atrophy. All subjects provided informed consent in accordance to the Oregon Health and Science University Internal Review Board regulations for human subjects' studies and the Helsinki Declaration.

Protocol

The subjects walked one revolution around a 1.2-m-radius circle marked on the floor. Walking direction was alternated between each trial to avoid vestibular decay that might affect gait. The subjects were asked to maintain their head erect and not fixate the floor in order to standardize the body position across subjects and avoid leaning over to stare at the circle. To standardize upper body position and avoid arm movements that would hide body markers from the cameras, subjects walked with their arms crossed. The subjects started by executing 10 revolutions around the circle (five in each direction) with eyes opened, immediately followed by 10 additional revolutions around the circle with eyes closed (and with a blindfold). The instructions to the subjects were to walk one full turn around the circle as they had performed in the eyes opened condition and to stop once they thought they were back to their starting position. Only after the subjects had stopped were they allowed to open their eyes and lift up the blindfold to look at their current position and return to the initial position to start another trial in the opposite direction. Hence, subjects received feedback about their final position but not on how far they deviated from the circle. The only instance subjects received feedback concerning deviation from the circle was when they were stopped because they were about to hit a wall or an obstacle (chair or desk). When stopped, the subjects were asked to return to their start position and start the next trial. Table 2 describes the number of subjects who deviated away from the circle enough to be stopped during the trial. The subjects wore a safety harness equipped with a handle that could be quickly held by an assistant who was ready to catch the subjects in case of a fall. No such incident occurred for any of the subjects.

Protocol for subjects with PD and cerebellar ataxia

PD subjects were tested off medication (OFF) the morning after abstaining from levodopa overnight (washout period ≥12 h). After completing the full protocol in the OFF condition, the PD subjects were given their usual morning dose of medication, followed by a rest period of 1 h. After the rest period, once the subjects reported that they felt "ON," the protocol was repeated with PD subjects on medication (ON). The motor part III of the UPDRS (Fahn et al., 1987) was used to characterize the state of disease OFF (before starting the protocol) and ON (after the rest period) medication (Table 1).

The PD in OFF and ON condition and cerebellar subjects walked at their comfortable/preferred speed around the circle 10 times in each direction with eyes open, followed by walking around the circle 10 times in each direction with eyes closed.

Table 2. Number of subjects who were stopped during eyes closed walking around the circle

Group	No. of trials stopped	No. of different subjects
Controls	6*	4**
PD OFF meds	12	6
PD ON meds	3	3
Ataxia	3	2

* One subject was stopped when matching speed.

** One subject was stopped in both comfortable and matched speeds.

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