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Visual deprivation elicits subclinical postural inflexibilities in early Parkinson's disease☆

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ABSTRACT

Background: Postural instability is often experienced in the late stages of PD and is a marker of disease progression. Little information is available on the role of visual inputs as an adaptive strategy to compensate for postural instability in PD. The purpose of this study was to determine visual dependency for postural control in early PD.
Methods: Thirty early PD subjects without postural complaints and 30 matched controls were evaluated for subtle postural instability using static posturography under eyes opened and eyes closed conditions.
Results: No significant differences between groups were observed under eyes opened condition. In eyes closed condition, there was significantly greater mean sway in the mediolateral direction ($p = 0.01$), mean sway velocity ($p = 0.03$), lateral sway velocity ($p = 0.04$), and sway area ($p = 0.04$) in PD than in the control subjects. 95% confidence ellipse of mean sway was largest in PD patients with eyes closed. A strong and significant correlation was observed between disease duration and mean mediolateral sway, sway area, mean sway and lateral sway velocity, and a moderate correlation was shown between Hoehn & Yahr stage and mean mediolateral sway, and sway area.
Conclusion: Our findings suggest that visual dependency exists in early PD and visual deprivation task can help identify subclinical postural instability.

1. Introduction

Postural instability (PI) is considered to be a clinical hallmark of Parkinson's disease (PD) [1]. Although PI can be the presenting symptom in some PD patients, the generally accepted understanding is that the presence of PI indicates at least the moderate stage of the disease as determined by a minimum of stage 3 on the Hoehn and Yahr (HY) scale [2]. Moreover, when PI is prominent early in the course of the disease, it suggests the possibility of atypical Parkinsonian disorders, such as progressive supranuclear palsy [3]. In light of the evidence that PI is a marker for disease progression, is associated with rapid disease progression, and is a major source of disability and reduced quality of life, early recognition of this problem, even at the subclinical stage, may provide those at-risk an opportunity for early interventions to improve physical stability and reduce the risk of future falls [4–6]. The current clinical examination method of the postural instability (the pull test), which has been incorporated into the standard Unified Parkinson's Disease Rating Scale (UPDRS), can detect PI only when the symptoms are prominent and the interpretation can be quite subjective and variable among different examiners [7].

Recent evidence has demonstrated that gait and postural disturbances are present but can be subtle in the early PD stages, and will eventually affect all PD patients as the disease progresses [8–10]. In particular, early PD patients have been found to have infraclinical postural instability as manifested by dynamic postural asymmetry, a larger center foot pressure sway area with both eyes closed and eyes opened under the static condition, and a greater sway area under the dynamic condition when compared to control subjects [10–13]. Although sway parameters increase with disease severity, no direct correlation has been made with PI [14]. This impaired postural adaptation to changes in postural tasks has been referred to as 'postural inflexibility' [15]. From the pathological viewpoint, the occurrence of subclinical PI seems plausible since nondopaminergic cell loss in the locus ceruleus, which has been linked to PI, already occurs in the early, or even in the preclinical stages of PD [8,16]. However, it is difficult to determine if subclinical PI represents the disease process or the compensatory mechanisms of postural dysfunction.

The control of posture and balance requires the interaction between postural alignment and stabilization. Current evidence supports that both systems are impaired in PD [17,18]. When clinically advanced and obvious, PD patients may present with several types of postural alignment problems, including stooped posture, flexion of the knees

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and trunk with elbows bent and arms adducted. However, these features are not normally observable in early stage patients. One recent study also suggested that the orientation problems probably occur before the disequilibrium [17]. Postural control is not an isolated motor function but requires sensory information from the vestibular, proprioceptive, and visual systems. Since the basal ganglia has a central role in the integration of the state of equilibrium to regulate motor commands appropriate to sensory experience, it is likely that this function cannot operate properly in patients with PD [19]. While vestibular dysfunction may play no or a minor role in PI in PD, many studies have supported the link between PI and the deficits in the proprioceptive function [17, 20–22]. On the other hand, the contribution of visual inputs is viewed as an adaptive strategy to compensate for the proprioceptive deficits [17]. There is ample evidence to suggest that PD patients have significant increased dependence upon visual information both perceptually and motorically, particularly on the visual orientation of posture and equilibrium [23–27]. Moreover, the visual perception of verticality has been found to be abnormal in PD and correlates with PI [27,28]. Therefore, we postulate that in the early stage of PD, there is redundancy of sensory information that is conveyed to the basal ganglia, which select and suppress incongruent sensory information to compensate for situations of sensory conflict and/or sensory deprivation. As the disease progresses and the proprioceptive deficits increase, patients adopt the strategy to re-weight their sensory inputs in favor of the visual mode.

Therefore, the aim of this study was to determine the role of visual dependence on the postural control in early PD patients and if the findings could lead to a more effective method to clinically evaluate the subclinical PI in PD patients who have not as yet had gait or postural complaints or findings.

2. Methods

2.1. Subjects

30 patients with early stage of PD (HY: 1–2.5) were recruited from the outpatient clinic of the Chulalongkorn Center of Excellence on Parkinson's Disease & Related Disorders between June 2012 and October 2013. The inclusion criteria were: 1) the patient must not have had symptoms and signs of PI, which were defined as a score of "0" on item 13 of UPDRS part II (Falling which was unrelated to freezing), and a negative result of the pull test and score "0–1" on item 30 of UPDRS part III (Postural stability); 2) the patients must not have a history of falling in the past 12 months; 3) there was no history of motor complications as determined by "0" scores on the sections A (Dyskinesia) and B (Clinical fluctuations) of UPDRS part IV; 4) there was no tremor in the lower extremities that may interfere with the evaluation with posturography, and 5) there were no other comorbidities that may affect posture and balance, including neuropathy, impaired proprioception, vestibular disorders, and visual disturbances. All subjects must have had a negative Romberg's test. Patients were classified into tremor predominant, akinetic-rigid or mixed subtypes using the approach proposed by Jankovic et al. [5]. All PD patients were assessed in the morning at least 12 h after the last dose of antiparkinsonian medications in order to reduce the effect of dopaminergic medications on posturographic findings. None of the patients were taking sedatives. Subjects were excluded if they scored less than 25 on the validated Thai version of the Mini-Mental State Examination (Thai MMSE) or if they had major depression as defined by DSM-IV criteria.

The patients were compared to 30 age- (±5 years), sex-, weight- (±2 kg), height- (±5 cm), BMI- (±2) matched healthy controls. Control subjects fulfilled the same inclusion and exclusion criteria as the patients with PD and neurological examinations did not detect any abnormalities. The study was approved by the Human Subjects Ethics Committee of the Faculty of Medicine, Chulalongkorn University. All subjects gave their written informed consent before entering the study in accordance with the Declaration of Helsinki.

2.2. Static posturography

Postural sway was analyzed using a 50 * 50 cm force platform (Cosmogamma, Emildue, Cento, Italy) with three dynamometric load cells measuring the forces exerted by the subjects on the support surface. The signals were amplified and acquired by a computer with a sampling frequency of 20 Hz. Changes in position of the resultant vector on the platform, e.g., the CoP, were calculated by a software program (BalancePlatform v. 8.0.1), and the corresponding representation of COP trajectories was displayed. Platform calibration was performed each day before testing.

2.3. Experimental protocol

All subjects were asked to maintain an upright standing position on the force platform, with arms at their sides, and bare feet externally rotated at an angle of 30° and heel-to-heel distance was standardized at 2 cm. All test conditions were conducted in a comfortable quiet environment. In the eyes opened (EO) condition, subjects were asked to look straight at a fixed point on the wall at a 1-meter distance for 30 s [29]. In order to remove visual input, all subjects were asked to repeat the same procedure with eyes closed (EC). Each trial was repeated three times and mean values for these three trials were reported for all subjects. Rest periods of 2 min were permitted between each trial. Each subject was tested in a 1-day session.

The center of pressure (CoP) was computed from the vertical forces. The CoP is the point location of the ground reaction force vector and reflects the sway of the body and forces used to maintain the center of gravity within the support base. Five dependent variables were calculated from the raw data: 1) mean sway (mm): simple mean of the distance from the CoP to all recorded positions; 2) sway velocity (mm/s): average travel velocity of the CoP calculated by dividing the total length of the CoP trajectory (mm) by the recording period length (s); 3) sway path length which is the total distance covering the successive positions of the moving CoP (mm); 4) sway area (cm^2); and 5) the Romberg quotient. All variables (except the Romberg quotient) were computed for the bidimensional CoP trajectory (both the anteroposterior (AP), and mediolateral (ML) directions of the total CoP displacement). The Romberg quotient was calculated for each patient (total displacement (mm) with EC/total displacement (mm) with EO). The higher the Romberg quotient, the more a subject relies on visual information to maintain postural control [24]. In addition, a 95% confidence ellipse for each trial was estimated, which encloses approximately 95% of the points on the CoP trajectory. The area of the confidence ellipse (ellipse area) and the direction of maximal sway were quantified.

2.4. Statistical analysis

Baseline characteristics and postural parameters were summarized using either means and standard deviations (SD), or frequencies and percentages as appropriate. For each posturographic parameter and for both conditions, the mean of the three trials was used in the analysis. The paired *t* test was used to compare the results between PD patients and normal controls in both EO and EC conditions. Correlations across the sway data, and between the sway data and the patients' demographics, were evaluated with the non-parametric Spearman's rank test. Subgroup analysis of posturographic variables between different subject groups was performed by one-way ANOVA. A $p < 0.05$ (2-tailed) was considered statistically significant. Statistical analysis was performed using SPSS version 17.0 software (SPSS Inc., Chicago IL).

3. Results

Demographic and baseline characteristics are summarized in Table 1. There were 30 subjects in each group: 15 males and 15 females. The patient group had a mean age of 66.07 (SD = 8.54) and a mean HY