


# Impact of Cognitive Loading on Postural Control in Parkinson's Disease With Freezing of Gait

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## Abstract

**Objective:** To assess standing balance in Parkinson's disease (PD) patients with and without freezing of gait (FOG) during cognitive loading. **Method:** A balance assessment with cognitive loading, reading (RE) and counting backward (CB), was performed by the Nintendo Wii Fit in 60 PD patients (Hoehn and Yahr stages 1-3) at Thammasat University Hospital, Thailand. The participants were grouped into FOG and non-FOG according to the Freezing of Gait–Questionnaire (FOG-Q) scores. The center of pressure (CoP) in terms of path length (PL), sway area (SA), root mean square (RMS), medio-lateral (ML), and antero-posterior (AP) were analyzed. **Results:** Significant increases of PL were observed in both groups of PD patients during cognitive loading ( $p < .001$ ). Meanwhile, the increased differences of PL during cognitive loading in PD-FOG were larger than in PD-non-FOG. The ML displacement during counting backward was significantly increased in PD-FOG ( $p = .012$ ). **Conclusion:** Cognitive loading influenced standing balance and postural sway of PD patients. The effects were more prominent in PD-FOG. These findings represent the interactions between cognitive function, postural control, and FOG in PD.

## Keywords

Parkinson's disease, freezing of gait, postural instability, postural control, cognitive loading

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## Background

Cognitive impairment (CI) is an important problem for Parkinson's disease (PD) patients. It is a common non-motor symptom that could occur in the early stages, and develop progressively in the advanced stages of the disease (Dujardin, Moonen, & Behal, 2015; Mak, Su, & Williams, 2015). The degree of CI in PD ranges from mild cognitive impairment (PD-MCI) to dementia (PDD), resulting in burden to family members and caregivers (Mak et al., 2015). The onset of cognitive decline in PD is often associated with older age, lower level of education, greater disease severity, postural instability (PI) and gait difficulty subtype (PIGD), and a long duration of the disease.

Several studies reported interferences between postural control and cognitive tasks passing the visuospatial pathways. Attentional demands such as auditory cues and cognitive tasks have been applied to distract cognitive function to evaluate stabilizing posture capability (Cook, 2000; Kelly, Johnson, & McGough, 2015; Nantel, McDonald, Tan, & Bronte-Stewart, 2012). To maintain normal human balance, there are 3 systems required; sensory input (visual,

vestibular, proprioceptive), integration (cerebrum, cerebellum, basal ganglia (BG)), and motor output (vestibulo-ocular reflex, motor impulses for eye movements, motor impulses which help adjusting posture) (Peterka, 2002; Watson & Owen, 2014). In normal state, the inputs and motor outputs are in equilibrium. In PD, according to the degeneration of BG, the loss of dopaminergic neurons in PD affects several subcortical pathways, which lowers the capability of distributing motor outputs and brings about the motor symptoms (Santens, Boon, Van Roost, & Caemaert, 2003) causing PI in PD patients. PI is one of the parkinsonian motor symptoms that usually occurs at the later stage of the disease, with increased risk of falling and

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near falls in PD patients, resulting in poor quality of life (QoL; Balash et al., 2005; Lachman et al., 1998).

One physiological factor causing PI is muscle hypertonicity. To maintain normal posture, muscle tone must remain in its normal state. PD patients experience muscle hypertonicity which is the impaired ability of motor neurons in regulating descending pathways increasing excitability of muscle spindles (Double & Crocker, 1995). Abnormal muscle tone causes the inability of controlling postural muscles for maintaining normal balance. This leads to PI in PD. Due to the main motor symptoms of the disease; tremor, bradykinesia, rigidity, and postural instability (PI), the problems of freezing of gait (FOG) and balance dysfunction are commonly found in advanced stages of the disease. FOG is a parkinsonian gait characterized by small steps, shuffling gait, feeling one's feet are glued to the ground, and/or difficulty of stepping forward which represents muscle hypertonicity (Jankovic, 2008; Rinalduzzi, Trompetto, & Marinelli, 2015).

CI is well-known in PIGD patients with FOG (Heremans, Nieuwboer, & Spildooren, 2013; Maruyama & Yanagisawa, 2006; Morris, Iansek, Smithson, & Huxham, 2000). This is because prefrontal cortex and BG play important roles in both cognitive and gait functions. Deterioration of these pathways may affect each other and cause FOG, CI, and PI, as well as the impairments of the frontostriatal neural circuitry leading especially to CI (Kelly et al., 2015; Lewis, Dove, & Robbins, 2003; Mahoney, Holtzer, & Izzetoglu, 2016). The hypothesis is that FOG, PI in static standing balance and CI might be related to each other according to the pathophysiology of the disease. CI such as reading and counting backward might interrupt postural control in quiet standing in PD patients and has greater affect in the patients with FOG. The purpose of this study is to investigate the effects of cognitive loading toward postural stability in PD patients, particularly with FOG.

## Method

### Participants

Sixty PD patients (24 male and 36 female) aged  $66.48 \pm 10.32$  years, with duration of the disease  $5.31 \pm 3.42$  years, age of onset  $61.27 \pm 10.96$  years, having Unified Parkinson's Disease Rating Scale (UPDRS) motor score of  $22.87 \pm 12.182$  ( $M \pm SD$ ), participated in this study. The patients were recruited from Thammasat University Hospital, Thailand. All the participants signed an informed consent approved by the Ethical Review Board. Idiopathic PD was diagnosed according to the United Kingdom Parkinson's Disease Society Brain Bank (UKPDSBB) criteria (Hughes, Daniel, & Kilford, 1992). Clinical staging of PD was classified according to the Modified Hoehn and Yahr Scale (H&Y; Hoehn & Yahr, 1967). Patients who were able to stand independently for at least 3 min were included to this study. Participants with other neurological problems, atypical

parkinsonism, for example, vascular parkinsonism, parkinsonism plus, drug-induced parkinsonism, motor weakness such as severe sensory neuropathy and cerebellar ataxia, unable to stand still without support, severe dyskinesia, psychological problems, vestibular dysfunction, postural hypotension, and partial or complete blindness or deaf were excluded. All participants with PD were examined during the on-time medication without presenting excessive rigidity, bradykinesia, or tremor.

### Instrumentation

Standing balance was measured by the posturographic balance platform: Nintendo Wii Fit (Nintendo of America Inc., Redmond, WA; Clark et al., 2010). It consists of a novel balance board system with a specific written program by one of the authors. The program was developed from the Wiimote library, which receives the data via Bluetooth connection on PC. It has been tested by many programmers and no issue is known concerning its validity. The input device is a platform that measures the distribution of weight bearing. The Wii Fit tracks changes in the center of pressure (CoP) by detecting the shifting of participants' weight, without stepping or moving the feet while standing on the particular platform. The platform detects shifts in weight bearing in antero-posterior (AP) and medio-lateral (ML) dimensions.

### Procedures

The participants were instructed to stand naturally on the balance platform (Wii Fit) and look at a marker on the wall, which was 3 m from the platform. The study was completed by the same instrument, which was calibrated before each data collection. The medial borders of each foot were about 10 cm apart. The participants were asked to perform two tasks in cognitive loading sessions, namely reading (RE) and counting backward (CB). The test was carried out by inviting participants to stand on the platform read pre-selected material, as well as count days backward; starting from Sunday, Saturday, Friday, . . . to Monday, for a total of 170 s. CoP movements were measured by the platform and were transferred to PC via Bluetooth. Each session was continuously proceeded, and was automatically collected by the written program.

The CoP in terms of path length (PL), sway area (SA), root mean square (RMS), medio-lateral (ML), and antero-posterior (AP; Visser, Carpenter, van der Kooij, & Bloem, 2008) were analyzed using the Unified Parkinson's Disease Rating Scale (UPDRS) motor (Items 18-31) sub-score (Visser, Marinus, & Bloem, 2003), Levodopa Equivalent Dose (LED; Alexoudi, Shalash, & Knudsen, 2015), Freezing of Gait-Questionnaire (FOG-Q; Nilsson & Hagell, 2009), Thai Mental State Examination (TMSE; Muangpaisan et al., 2015) and

**Table 1.** The summarization of participants' characteristics and clinical assessments comparing between PD patients with FOG and non-FOG sub-groups.

Variables	FOG ( <i>n</i> = 39)	non-FOG ( <i>n</i> = 21)	<i>p</i> <sup>a</sup>
Age, years ( <i>SD</i> )	65.13 ± 10.32	69 ± 10.08	1.99
Age of onset, years ( <i>SD</i> )	58.99 ± 10.87	65.2 ± 10.27	.030*
Duration of disease, years ( <i>SD</i> )	6.14 ± 3.57	3.79 (2.42)	.002**
H&Y, stages ( <i>SD</i> )	2.36 ± 0.69	1.86 ± 0.62	<.001**
UPDRS motor score ( <i>SD</i> )	24.72 ± 13.13	19.43 ± 9.59	.034*
LED, mg/day ( <i>SD</i> )	722.41 ± 392.23	452.62 ± 247.85	.007**
FOG-Q, scores ( <i>SD</i> )	11.72 ± 3.51	1.95 ± 1.43	<.001**
TMSE, scores ( <i>SD</i> )	25.33 ± 3.21	26.14 ± 2.65	.110
MoCA ( <i>SD</i> )	18.87 ± 5.05	19.81 ± 5.5	.394

Note. The data was analyzed by Mann–Whitney *U* test. PD = Parkinson's disease; FOG = freezing of gait; non-FOG = non-freezing of gait; H&Y = Hoehn and Yahr; UPDRS = Unified Parkinson's Disease Rating Scale; LED = Levodopa Equivalent Dose; FOG-Q = Freezing of Gait–Questionnaire; TMSE = Thai Mental State Examination; MoCA = Montreal Cognitive Assessment.

\**p* < .05. \*\**p* < .01.

<sup>a</sup>Mann–Whitney *U* test.

**Table 2.** The average and standard deviation of posturographic data comparing between Before I and RE, Before II and CB in the 60 PD patients.

CoP	Before I	RE	<i>p</i> <sup>b</sup>	Before II	CB	<i>p</i> <sup>b</sup>
PL, mm ( <i>SD</i> )	86.86 ± 37.3	101.42 ± 61.96	<.001**	95.37 ± 48.47	117.33 ± 84.08	<.001**
SA, cm <sup>2</sup> ( <i>SD</i> )	10.07 ± 11.61	13.13 ± 21.12	.361	15.06 ± 20.94	21.9 ± 37.28	.162
RMS ( <i>SD</i> )	2.74 ± 2.75	3.77 ± 5.97	.156	3.95 ± 4.98	6.59 ± 13.57	.083
Max ML, cm ( <i>SD</i> )	0.49 ± 2	0.67 ± 2.39	.943	0.82 ± 2.46	5.74 ± 35.74	.284
Min ML, cm ( <i>SD</i> )	−2.34 ± 2.45	−2.43 ± 2.49	.462	−2.37 ± 2.1	−2.85 ± 2.86	.339
Max AP, cm ( <i>SD</i> )	5.31 ± 1.74	5.46 ± 1.98	.256	5.36 ± 1.96	5.23 ± 1.97	.477
Min AP, cm ( <i>SD</i> )	2.34 ± 1.63	2.32 ± 1.61	.415	1.9 ± 1.64	1.69 ± 1.9	.232
ΔML, cm ( <i>SD</i> )	2.83 ± 1.14	3.1 ± 2.77	.416	3.19 ± 2.45	8.59 ± 36.46	.012*
ΔAP, cm ( <i>SD</i> )	2.97 ± 1.14	3.14 ± 1.48	.286	3.46 ± 1.75	3.54 ± 2.03	.880

Note. PD = Parkinson's disease; CoP = center of pressure; RE = reading; CB = counting backward; PL = path length; SA = sway area; RMS = root mean square; ML = medio-lateral displacement; AP = antero-posterior displacement; ΔML = maximal medio-lateral displacement – minimal medio-lateral displacement; ΔAP = maximal antero-posterior displacement – minimal antero-posterior displacement.

\**p* < .05. \*\**p* < .01.

<sup>b</sup>Wilcoxon Signed-Rank test.

Montreal Cognitive Assessment (MoCA; Kandiah, Zhang, & Cenina, 2014).

### Statistical Analysis

The CoP trajectories time series of the total participants (*n* = 60) were reported in terms of PL, SA, RMS, AP, and ML displacements by the Wii program. The descriptive analysis of the posturographic parameters was evaluated in average and standard deviation (*SD*).

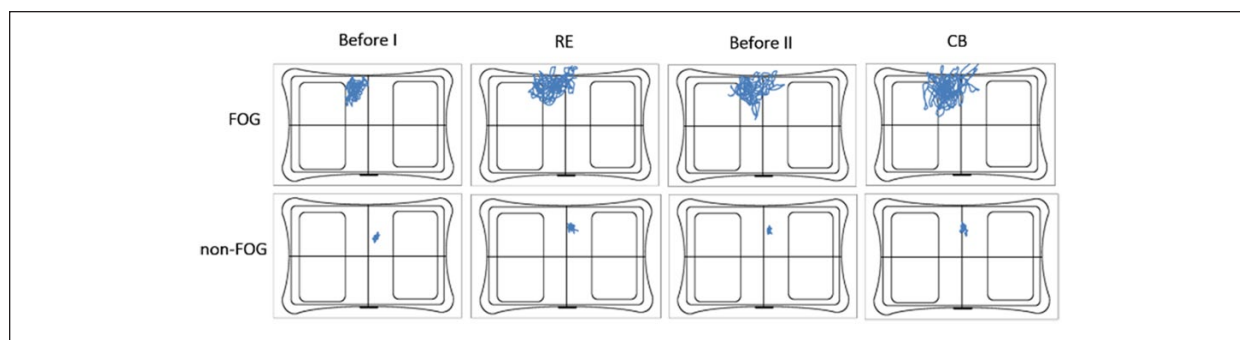
SPSS 22.0 (IBM Corp, Armonk, NY) was applied to calculate the data. All variables were tested for normality by Kolmogorov–Smirnov. Age, age of onset, duration of disease, H&Y, UPDRS (motor score), LED, FOG-Q, TMSE and MoCA were analyzed by the non-parametric Mann–Whitney *U* test. A sub-analysis was employed by categorizing the participants into two groups, FOG (*n* = 39) and non-freezing of gait (non-FOG; *n* = 21). PD patients with FOG were classified by a total score of FOG-Q ≥ 6. The comparison of mean differences of CoP between before I (before reading) and reading (RE), and before II (before counting backward) and counting

backward (CB) were calculated by using the Wilcoxon Signed-Rank test. The Spearman's rho correlation was utilized to calculate correlation between H&Y stages and posturographic parameters. The statistical significance level was set at *p* value less than .05.

## Results

### Clinical Characteristics

In total, 60 PD patients participated in this study and descriptive statistics were utilized to characterize the participants. Twenty-four (40%) were men, and 36 (60%) were women. The participants were 43 to 89 years old, and the mean age was 66.48 ± 10.32 years (*M* ± *SD*). The age of onset was 61.27 ± 10.96 years, duration of disease was 5.31 ± 3.42 years, and UPDRS motor score was 22.87 ± 12.18. The non-parametric statistics were utilized in this study and the participants' demographic and clinical assessments in PD-FOG and PD-non-FOG are summarized in Table 1. Mean ages (65.13 ± 10.32 vs. 69 ± 10.08, *p* = 1.99) and cognitive abilities test scores



**Figure 1.** CoP trajectories in the PD patients with and without FOG comparing between Before I and RE, and Before II and CB. Note. CoP = center of pressure; PD = Parkinson's disease; FOG = freezing of gait; non-FOG = non-freezing of gait; RE = reading; CB = counting backward.

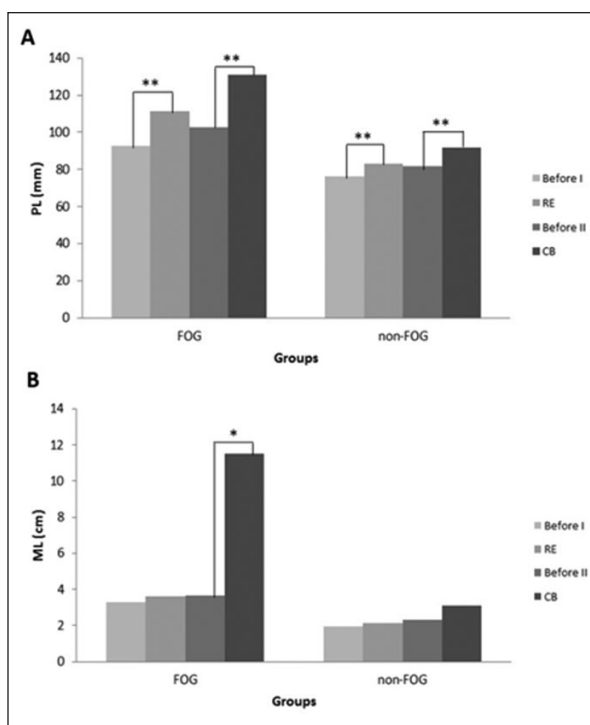
( $18.87 \pm 5.05$  vs.  $19.81 \pm 5.5$ ,  $p = .394$ ) as well as TMSE scores ( $25.33 \pm 3.21$  vs.  $26.14 \pm 2.65$ ,  $p = .110$ ) of the participants were not significantly different between groups. The significant differences were found in age of onset ( $p = .030$ ), duration of disease ( $p = .002$ ), H&Y stages ( $p < .001$ ), and UPDRS motor sub-score ( $p = .034$ ). The clinical assessments, LED, and FOG-Q score were significantly increased in the group of FOG ( $p = .007$ ,  $p < .001$ , respectively).

### Posturographic Data

In the reading sub-session, the posturographic data of 60 PD cases were compared between before I and reading (RE), and before II and counting backward (CB). Significant increases of PL were found in RE ( $p < .001$ ) and CB ( $p < .001$ ). Significant increase was found in  $\Delta$ ML in CB ( $p = .012$ ). None of the other parameters were found to be significantly different as shown in Table 2. The CoP trajectories illustrated the characteristics of CoP movements within each condition. The PD-FOG showed higher postural sway than PD-non-FOG in all scenarios. The CoP movements in RE were larger than Before I. Similarly, in CB, they revealed higher sway area and fluctuation than Before II as illustrated in Figure 1.

The sub-analysis was calculated by dividing the patients into two groups: FOG ( $n = 39$ ) and non-FOG ( $n = 21$ ). In the RE sub-session, significant increases of PL between Before I and RE were found in both PD-FOG ( $p < .001$ ) and PD-non-FOG ( $p < .001$ ). PL in PD-FOG was larger than PD-non-FOG ( $111.32 \pm 74.31$  vs.  $83.05 \pm 16.98$ ). No significant differences were observed in other posturographic parameters.

In the CB sub-session, the sub-analysis illustrated that between Before II and CB, significant increases of PL were noticed in both PD-FOG ( $p < .001$ ) and PD-non-FOG ( $p < .001$ ). The significantly increased difference in  $\Delta$ ML was found only in PD-FOG ( $p = .042$ ). PL in PD-FOG were higher than in PD-non-FOG ( $131.13 \pm 100.4$  vs.  $91.71 \pm 25.3$ ). Meanwhile, ML in FOG were larger than in non-FOG ( $11.54 \pm 43.86$  vs.  $3.11 \pm 2.6$ ) as



**Figure 2.** The bar charts showing the comparisons of CoP between before I and RE, and before II and CB in the PD patients with FOG and non-FOG: (A) PL and (B) ML. Note. CoP = center of pressure; RE = reading; CB = counting backward; PD = Parkinson's disease; FOG = freezing of gait; non-FOG = non-freezing of gait; PL = path length; ML = medio-lateral. \* $p < .05$ . \*\* $p < .01$ .

demonstrated in Figure 2. No statistically significant differences in other parameters were observed as expressed in Table 3.

### Correlation Analysis

Spearman correlation was used to perform the correlation between severity of disease according to H&Y stages and posturographic variables in RE and CB tasks. Table 4 and Figure 3 illustrate that H&Y stages correlated with PL ( $p = .014$ ), SA ( $p = .001$ ),  $\Delta$ ML ( $p = .029$ ),



**Table 3.** The average and standard deviation of posturographic data comparing the PD patients with FOG and non-FOG between Before I and RE, Before II and CB.

Parameters	FOG (n = 39)		p <sup>b</sup>	non-FOG (n = 21)		p <sup>b</sup>
	Before I	RE		Before I	RE	
PL, mm (SD)	92.62 ± 44.11	111.32 ± 74.31	<.001**	76.18 ± 14.93	83.05 ± 16.98	<.001**
SA, cm <sup>2</sup> (SD)	12.51 ± 13.27	17.4 ± 25.1	.258	5.53 ± 5.47	5.22 ± 3.81	.455
RMS (SD)	3.34 ± 3.14	4.94 ± 0.75	.081	1.62 ± 1.24	1.61 ± 1.05	.903
Max ML, cm (SD)	0.43 ± 2.27	0.75 ± 2.75	.662	0.59 ± 1.42	0.52 ± 1.59	.681
Min ML, cm (SD)	-2.87 ± 2.77	-2.96 ± 2.13	.357	-1.37 ± 1.27	-1.61 ± 1.59	.823
Max AP, cm (SD)	5.6 ± 1.85	5.82 ± 2.19	.283	4.76 ± 1.38	4.81 ± 1.36	.575
Min AP, cm (SD)	2.39 ± 1.76	2.31 (1.78)	.303	2.25 ± 1.39	2.33 ± 1.27	.862
ΔML, cm (SD)	3.3 ± 2.45	3.62 (3.2)	.422	1.96 ± 0.98	2.14 ± 1.26	.689
ΔAP, cm (SD)	3.22 ± 1.17	3.5 (1.67)	.185	2.51 ± 0.94	2.48 ± 0.71	.986
	Before II	CB		Before II	CB	
PL, mm (SD)	102.7 ± 57.58	131.13 ± 100.4	<.001**	81.77 ± 18.23	91.71 ± 25.3	<.001**
SA, cm <sup>2</sup> (SD)	19.04 ± 24.26	27.98 ± 44.36	.241	7.67 ± 9.32	10.61 ± 12.45	.414
RMS (SD)	4.5 ± 5.8	8.78 ± 16.3	.105	1.99 ± 1.77	2.51 ± 2.99	.455
Max ML, cm (SD)	0.95 ± 2.72	8.38 ± 44.3	.346	0.59 ± 1.9	0.84 ± 1.88	.627
Min ML, cm (SD)	-2.72 ± 2.19	-3.16 ± 2.93	.562	-1.71 ± 1.77	-2.26 ± 2.69	.487
Max AP, cm (SD)	5.68 ± 2.17	11.54 ± 43.86	.635	4.78 ± 1.36	4.67 ± 1.44	.578
Min AP, cm (SD)	1.82 ± 1.78	5.54 ± 2.17	.110	2.05 ± 1.38	1.96 ± 1.27	.741
ΔML, cm (SD)	3.67 ± 2.7	11.54 ± 43.86	.042*	2.3 ± 1.59	3.11 ± 2.6	.144
ΔAP, cm (SD)	3.86 ± 1.9	3.99 ± 2.27	.732	2.73 ± 1.12	2.71 ± 1.15	.768

Note. PD = Parkinson's disease; FOG = freezing of gait; non-FOG = non-freezing of gait; RE = reading; CB = counting backward; PL = path length; SA = sway area; RMS = root mean square; ML = medio-lateral displacement; AP = antero-posterior displacement; ΔML = maximal medio-lateral displacement - minimal medio-lateral displacement; ΔAP = maximal antero-posterior displacement - minimal antero-posterior displacement.

\*p < .05. \*\*p < .01.

<sup>b</sup>Wilcoxon Signed-Rank test.

and ΔAP (p < .001) in RE. No correlations were found among the posturographic variables in CB. The 95% confidence ellipse of mean ML, and AP displacements between PD-FOG and PD-non-FOG of RE and CB were demonstrated in Figure 4.

**Discussion**

The results of this study demonstrated that the cognitive loading influences postural control in patients with PD. The balance platform, Nintendo Wii Fit, utilized in this study with the written program and the cognitive loading sessions, reading (RE) and counting backward (CB), are applicable for identifying PD patients with balance disturbances particularly with FOG. The cognitive loading sessions affected the changes of CoP trajectories while the participants were asked to follow the tasks.

This suggests the ability of controlling posture of PD patients in standing while receiving the cognitive loading tasks is defective. The interferences from the tasks may disturb the brain's circuits resulting in the destabilizing of the postural muscles. The results are accordant with previous studies that PI can be found in patients with abnormal muscle tone, and the patients with the deterioration of BG present poor balance as depicted by figure 1 and the large diameter

**Table 4.** Correlation between severity of disease (H&Y stages) and posturographic variables.

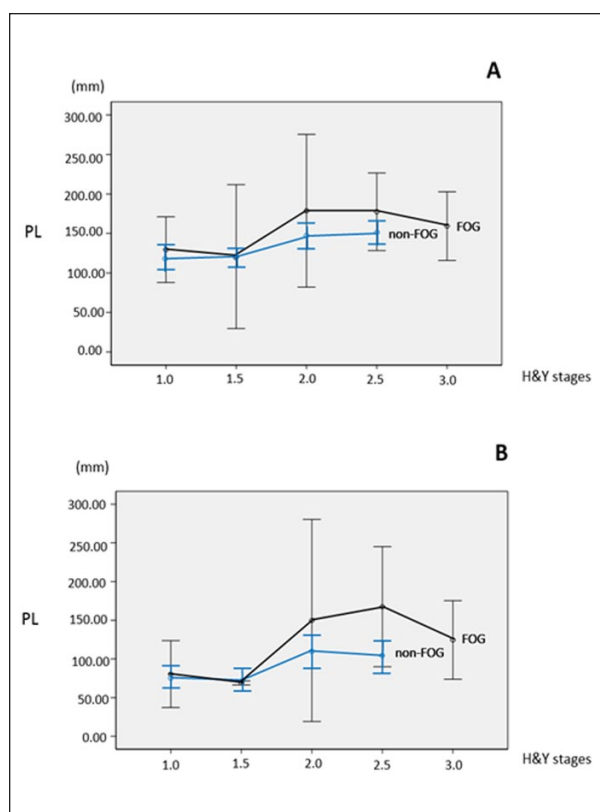
	Correlation	p <sup>c</sup>
Reading		
PL	.316	.014*
SA	.404	.001**
ΔML	.282	.029*
ΔAP	.473	<.001**
Counting backward		
PL	.229	.078
SA	.135	.304
ΔML	.149	.257
ΔAP	.220	.092

Note. H&Y = Hoehn and Yahr; PL = path length; SA = sway area; ΔML = maximal medio-lateral displacement - minimal medio-lateral displacement; ΔAP = maximal antero-posterior displacement - minimal antero-posterior displacement.

\*p < .05. \*\*p < .01.

<sup>c</sup>Spearman's rho correlation.

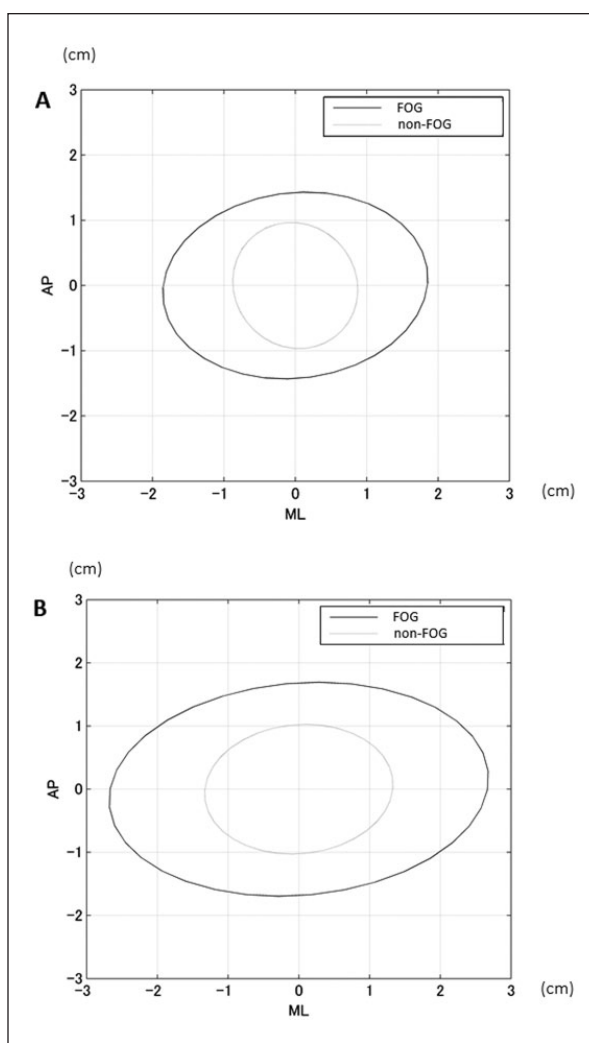
of CoP trajectories (Double & Crocker, 1995). The degeneration causes patients to lose the capability of controlling their balance (J. E. Visser & Bloem, 2005), which is similar to the results in this study. A previous study reported the effect of CIs on balance showing the reduction of ML control in PD (Shin,



**Figure 3.** Correlation analysis between severity of the disease (H&Y stages) and PL between FOG and non-FOG: (A) Reading and (B) Counting Backward. Note. H&Y = Hoehn and Yah; PL = path length; FOG = freezing of gait; non-FOG = non-freezing of gait.

Han, Jung, Kim, & Fregni, 2011). In this study, counting backward required greater postural control than reading, which might be interpreted that the counting backward was more difficult than the reading. It led to recruiting more muscles for controlling posture to maintain balance.

According to the function of BG in correcting postural responses, patients with PD gradually lose the ability of maintaining balance following the progression of the disease. The patients in advanced stages facing the problems of FOG expressed PI. MCIs have been found in the early stages (Lewis et al., 2003) where patients do not exactly manifest FOG. This statement supports our results that the non-FOG group presented the inability of controlling posture while receiving cognitive commands. ML control might be associated with the execution or cognition in PD. We found that the stabilizing in ML movements in PD-FOG was increased during counting backward. We can conclude that PI, CI, and FOG in PD have interaction. Previous studies reported CI and FOG were related. This study identified the connections of CI and FOG in terms of the CoP parameters (Heremans et al., 2013; Maruyama & Yanagisawa, 2006; Morris et al., 2000). Moreover, our study supports the studies



**Figure 4.** Ninety-five percent confidence ellipse of mean ML and AP displacements between FOG and non-FOG: (A) Reading and (B) Counting Backward. Note. ML = medio-lateral; AP = antero-posterior; FOG = freezing of gait; non-FOG = non-freezing of gait.

of Kelly et al. (2015), Mahoney et al. (2016), and Lewis et al. (2003) that perhaps the deterioration of prefrontal cortex and BG lead to the impairments of postural control in PD. Our study represents the interaction between PI, CI, and FOG, which could be explained by the decoupling of frontoparietal cortical circuits and BG (Shine et al., 2013). The deterioration of pedunculopontine nuclei (PPN) and their network could interrupt neural substrates and result in FOG (Fling et al., 2013; Shine et al., 2013; Youn et al., 2015). These influences were expressed in the postural control of PD-FOG in this study after receiving the cognitive loading tasks.

Several studies over the years (Doná et al., 2016; Frenklach, Louie, Koop, & Bronte-Stewart, 2009; Hiorth, Larsen, Lode, & Pedersen, 2014; Nantel & Bronte-Stewart, 2014) reported the severity of the disease and the stages of the disease followed by the

increase of age, age of onset, duration of disease, H&Y, UPDRS, and dopaminergic medication. These have caused changes in postural control and resulted in the increase of risk of falling and fall incidence. These factors are also presented in this study by showing significant differences between PD-FOG and PD-non-FOG. PD patients with high progression of the disease presented large dimensions of PL and SA, and an increase in  $\Delta ML$  and  $\Delta AP$  displacements. In addition, this study confirms the study by Pelykh, Klein, Bötzel, Kosutzka, and Ilmberger (2015). They documented the large dimensions of radius and sway path of the CoP in PD-FOG. The deficiency of postural control in PD-FOG during quiet standing is also concordant with the study of Schlenstedt et al. (2016). The abnormality of postural control in PD can be distributed to postural sensory impairment and was confirmed by the studies of Frenklach et al., 2009; Huh et al., 2016. PD-FOG presented worse postural control than non-FOG which can be attributed to the impairment of sensory receptors. This is supported by the study of Huh et al., 2016 that postural sensory deficits also correlated with FOG. Moreover, we found that LED was associated with PD-FOG and PD-non-FOG. These results were previously confirmed by a study by Nantel & Bronte-Stewart, 2014, which represented the contribution of dopaminergic therapy to FOG.

PL and postural sway in ML directions were significantly higher in PD-FOG than in PD-non-FOG while receiving cognitive loading. These results state the effects of cognitive declines toward PI in PD. The specific results showed in FOG that the patients have worse postural control compared with PD-non-FOG.

We acknowledge that our present study has design of experiment limitations. Our study has limited sample size. After sub-analyzing the data into two groups, the sample size of the FOG group was double the non-FOG group. This difference in study could definitely affect the results. There was no normal control group. The CoP displacements in before II might receive effects from the reading sub-session. This subsequently might lead to the results comparing the before II and counting backward sub-session. In further studies, we will enlarge the study population and adapt a study protocol to be more precise and include a resting period between each sub-session. The results encourage that specific balance programs could be considered to improve balance and cognitive function to reduce risks of falling and related problems in the future as well as to improve patients' QoL.

## Conclusion

Our study proposed that postural control in PD patients was influenced by the cognitive loading tasks: reading and counting backward. The ability of controlling balance

was required more in PD patients with FOG during cognitive demands. The changes of CoP trajectories were particularly prominent in ML displacement while performing the task of counting backwards. These findings represent the interactions between cognitive function, postural control, and FOG in PD.

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The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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