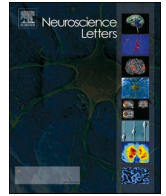


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Neuroscience Letters

journal homepage: www.elsevier.com/locate/neulet

Gait and posture are correlated domains in Parkinson's disease

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ARTICLE INFO

Keywords:

Biomechanics

Locomotion

Kinematics

Spatial-temporal parameters

Balance

ABSTRACT

Establishing a relationship between gait and posture in patients with Parkinson's disease (PD) is essential for PD treatment and rehabilitation. While previous studies have indicated that gait and posture are independent domains in PD, shared neuromechanisms related to gait and posture control and previous studies investigating the relationship between gait and posture parameters in stroke survivors and neurologically healthy older adults have shown a correlated domain. Thus, this study analyzed the relationship of gait and posture domains, primarily through gait temporal sub-phases (i.e., double support and stance phases) and step width. We analyzed the spatial-temporal gait parameters at the self-selected velocity and center of pressure (CoP) during quiet standing of 22 idiopathic PD participants under and without dopaminergic medication conditions. The association between quiet standing and gait variables was assessed through the Spearman test, controlled by age, disease duration, NFOG-Q, and levodopa dosage. In ON medication, CoP area showed a significant correlation with stance phase and total double support; and RMS ML CoP showed a significant correlation with stance phase, total double support, and step width. In OFF medication, CoP area, RMS AP CoP, RMS ML CoP, and ML CoP velocity significantly correlated with stance phase and total double support. By showing the relationship between gait and posture domains in PD, our study adds novel knowledge about the shared gait-posture control, which could collaborate with new approaches during mobility treatment and assessment.

1. Introduction

Establishing a relationship between gait and posture in patients with Parkinson's disease (PD) is essential for PD treatment and rehabilitation because a reliable correlation could mean that resources used to improve posture could also influence gait and vice-versa. However, the relationship between gait and posture in the PD context has been controversial. For example, Horak et al. [1], using factor analysis to identify independent measures of mobility extracted from the stand and walk test, found that gait specific parameters (stride velocity, stride duration) were not related to posture parameters (root mean square center of mass, sway area) in quiet standing. Based on that result, they suggested that posture and gait are different domains, so they are relatively independent. However, their analysis has some limitations: (a) the authors analyzed only general gait parameters (i.e., gait speed, step length and duration, and cadence) without addressing temporal gait sub-phases (i.

e., double support and stance phases) and step width, which may be more representative of posture during walking [2–4]; and (b) the authors did not differentiate directional posture parameters (i.e., anterior-posterior and medial–lateral sway parameters), that would best characterize the postural control [5]. In addition, shared neuromechanisms related to gait and posture control and previous studies investigating the relationship between gait and posture parameters in stroke individuals [6] and neurologically healthy older adults [2] seem to show they are correlated domains.

There are neural shreds of evidence that gait and posture share common control mechanisms [7]. For example, the common posture-gait pathway through the cerebellum. The cerebellum regulates cognitive and automatic posture and gait control processes by acting on the cerebral cortex via the thalamocortical projection and the brainstem. The feedforward information from the cerebral cortex and real-time sensory feedback to the cerebellum may play significant roles in these

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<https://doi.org/10.1016/j.neulet.2022.136537>

Received 29 October 2021; Received in revised form 15 February 2022; Accepted 15 February 2022

Available online 19 February 2022

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operations. At the same time, the cerebellum is also known for being connected to cortical motor areas, constructing motor programs, such as gait. The basal ganglia may also contribute to the modulation of each process through its projections to the cerebral cortex and brainstem [8]. The basal ganglia and cerebellum may affect both the automatic and cognitive processes of posture and gait control through reciprocal connections with the brainstem and cerebral cortex, respectively. Consequently, the common posture-gait control in the cerebral cortex, basal ganglia, and cerebellum may suggest a similar posture and gait domain.

Biomechanically, studies show that spatial-temporal gait parameters are predictive variables for postural control in early-stage PD patients during stability. For example, Yang et al. (2008) found that gait speed and stride length are correlated with dynamic posture, particularly in the forward direction during standing. Also, patients with postural impairment often have the stance phase of gait extended, probably as a tool for maintaining both feet on the ground for more extended periods, keeping posture [4,9].

Considering Horak's study limitations related to gait temporal sub-phases and directional sway parameters and the gait-posture shared neuromechanisms, there is an open question of whether gait variables are correlated with postural control domains in quiet standing in PD patients. No previous studies specifically investigate the relationship between gait variables such as double support duration, step width, and posture. To overcome this gap, this study analyzes the relationship of gait and posture domains, primarily through gait temporal sub-phases and step width and directional sway parameters. We hypothesize that gait and posture are associated on PD, especially for gait variables related to posture, such as step width and double support duration. Also, we investigate the gait and posture domains when the patients are under (ON) and without (OFF) dopaminergic medication conditions. Since levodopa may improve gait but impair postural sway during stance [10], we hypothesize that correlations will differ between medication conditions.

2. Methods

2.1. Participants

This study included 22 idiopathic PD participants (Table 1). The diagnosis was confirmed by a movement disorders specialist and FoG

Table 1
Mean (standard deviation) of the characteristics of the participants separately by medication condition.

	ON	OFF	p
Demographic and anthropometric			
Men/Women (n)	17/5	–	–
Age (years)	64.11 (11.04)	–	–
Body mass (kg)	71.36 (12.53)	–	–
Height (cm)	167.21 (7.35)	–	–
Clinical			
FoG/nFoG (n)	11 / 11	–	–
Disease duration (years)	8.32 (5.27)	–	–
L-Dopa equivalent units (mg•day ⁻¹)	772.24 (473.95)	–	–
NFoG-Q	7.42 (10.19)	–	–
MoCA (score)	22.95 (4.50)	23.37 (4.24)	0.647
H&Y stage (score)	2.21 (0.71)	2.32 (0.67)	–
UPDRS-III (score)	19.26 (9.21)	25.84 (11.35)	0.001 *
Mini-BESTest (score)	26.11 (4.94)	24.58 (5.92)	0.025 *
FES-I (score)	24.00 (7.11)	32.32 (11.07)	0.001 *

FoG: Freezing of gait. NFoG-Q: New Freezing of Gait Questionnaire; MoCA: Montreal Cognitive Assessment; UPDRS: Unified Parkinson's disease rating scale; Mini-BESTest: Mini-Test of Balance Assessment System scale; FES-I: Falls Efficacy Scale International. * indicates significant difference ($p < 0.05$).

based on question 1 of the New Freezing of Gait Questionnaire (NFoG-Q) [10]. Inclusion criteria were to walk independently at least 10 m without significant freezing episodes, the absence of neurological or physical dysfunctions other than those associated with PD, and no diagnosed vestibular, visual, or somatosensory dysfunctions as self-declared. The individuals were in a stable dose of L-DOPA for at least one month. All participants provided written informed consent to participate. The University's Ethical Committee approved this study.

2.2. Task and equipment

Participants performed two experimental tasks: quiet standing and unobstructed walking.

In quiet standing, the participants were required to stand barefoot and as still as possible for 60 s with their arms at their sides and look at a 5 cm round black target placed on the subject's eye height on a wall 3 m ahead. The participant's feet were placed with an angle of 20 degrees between them, and their heels were kept 10 cm apart by requesting the subjects to stand on lines marked on the top of the force platform (OPT400600-1000; AMTI, frequency 100 Hz).

Participants walked, without any assistance and any obstacle in the 10 m long walkway, at a comfortable self-selected speed. All gait trials were performed in barefoot conditions, and the participants wore comfortable shorts. In the middle of the walkway, at floor level, there was an electronic walkway system (Zebris FDM, frequency 100 Hz), composed of two connected electronic walkways (totaling 6 m long and 60 cm wide).

2.3. Procedures

The PD individuals participated in two experimental sessions at an interval of one week, one of which was in the ON condition (one hour after intake of the dopaminergic medication) of the medication and the other was in the OFF condition (at least 12 h without using any medication for PD). The order of the sessions was randomized among the participants. The start time of the experiment was the same as the experimental sessions.

The initial evaluations consisted of anamnesis to collect clinical data, medication, and disease diagnosis. At the beginning of each session, the following rating scales were applied: motor score of the Unified Parkinson's disease rating scale (UPDRS-III), Hoehn & Yahr (H&Y), New Freezing of Gait Questionnaire (NFoG-Q), Montreal Cognitive Assessment (MoCA), Mini-Test of Balance Assessment System scale (Mini-BESTest), and Falls Efficacy Scale International (FES-I).

After the initial clinical evaluations and a 10-minute rest period, the participants did the two experimental tasks. The order of the experimental tasks was randomized among participants. Participants performed ten trials to unobstructed walking and three trials to quiet standing tasks. Among the experimental tasks, participants had a 10-minute rest period. The assessments were repeated by the same investigator for all PD individuals.

2.4. Outcome measures and statistical analysis

Considering an expected correlation coefficient of 0.6, an error of 0.05, and the desired power of 0.80, the power analysis determined that 19 individuals were needed [11].

The measurement of gait velocity, cadence, stride length, step time, step width, stance phase, and total double support time was calculated. Preprocessing of raw data and extraction of gait variables were performed using appropriate data acquisition. The values of each parameter were the mean between the left and right sides.

Analysis of quiet standing was based on feet center of pressure (CoP) on the ground, assessing the area and root mean square (RMS) and mean velocity module in the anterior-posterior (AP) and medial-lateral (ML) directions. After visual data inspection, data extraction and processing

were made automatically through a Matlab (Mathworks) routine. CoP data were low-pass filtered through a fourth-order Butterworth filter with a cutoff frequency of 10 Hz. A description of outcome variables is in the [supplementary material](#).

As the data from the clinical scales, gait, and quiet standing were not normal (according to the Shapiro-Wilk test), we used a non-parametric analysis. The clinical scales were analyzed using a Wilcoxon signed-rank test. The association between quiet standing and gait variables was assessed through the Spearman test, controlled by age, disease duration, NFOG-Q, and levodopa dosage (only in ON condition). Given the explorative nature of this study and considering the addition of these covariates, we decided not to correct for multiple comparisons, as this might lead to false negatives [12]. The significance level was set at $p < 0.05$. Statistical procedures were performed using the software Matlab (Mathworks).

3. Results

The anthropometric and clinical characteristics of the individuals are presented in [Table 1](#). There were significant differences between the ON and OFF conditions in the UPDRS-III, miniBESTest, and FES-I scores.

The generalized Spearman correlation and rank matrix with the levels of significance (p-value) between the quiet standing and gait task variables in both medication conditions are shown in [Fig. 1](#). In ON medication, CoP area showed a significant correlation with stance phase ($\rho = 0.50, p = 0.040$), and total double support ($\rho = 0.54, p = 0.027$); and RMS ML CoP showed a significant correlation with stance phase ($\rho = 0.56, p = 0.020$), total double support ($\rho = 0.54, p = 0.026$), and step width ($\rho = 0.54, p = 0.025$). In OFF medication, CoP area showed a significant correlation with stance phase ($\rho = 0.58, p = 0.015$), and total double support ($\rho = 0.55, p = 0.022$); RMS AP CoP showed a significant correlation with stance phase ($\rho = 0.55, p = 0.022$), and total double support ($\rho = 0.53, p = 0.030$); and RMS ML CoP showed a significant correlation with stance phase ($\rho = 0.59, p = 0.013$), and total double support ($\rho = 0.56, p = 0.019$); ML CoP velocity showed a significant correlation with stance phase ($\rho = 0.50, p = 0.039$), and total double support ($\rho = 0.50, p = 0.039$). Since stance and swing phase are complementary, the ρ values are reversed. The ρ and p values for all comparisons are in the [supplementary material](#).

4. Discussion

This study analyzed the relationship between postural control and gait domains in PD patients. Our findings suggest that gait temporal sub-phases, such as stance time and double support time, and step width were related to body sway parameters (e.g., RMS, velocity, and area of CoP) during quiet standing in PD patients. Thus, it seems to support the notion that biomechanical characteristics and neural control of posture and gait are relatively dependent domains, especially when the gait sub-phases are analyzed. However, not all aspects of gait were related to posture. Here, we need to highlight the double support, stance phases, and step width. The relationship between gait and posture domains in PD adds novel knowledge about the shared gait-posture control, collaborating with new approaches during mobility treatment and assessment.

Corroborating our hypothesis, gait temporal sub-phases and step width were related to postural parameters in PD patients. Postural sway measures while quiet standing is correlated with double and stance periods and step width of gait in PD patients. Our results suggest that body sway control impairments may affect gait stability in PD. Our results share common conclusions as the study conducted by Yang et al. [13], where they stated that there are close relationships between step width and stability. In addition, they noticed that short-term voluntary adoption of wider steps helped increase lateral stability due to the increase in the base of support. On the other hand, while Horak et al. [1] indicated that posture and gait are relatively independent domains, we

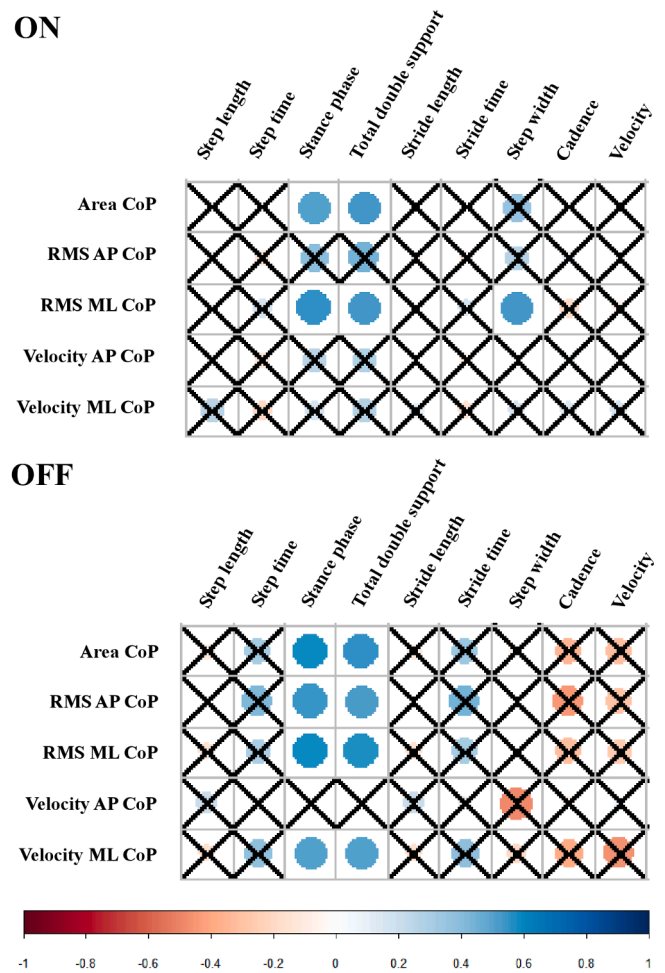


Fig. 1. Correlation matrix and generalized Spearman rank correlation with significance levels (p-value) between the quiet standing and gait variables in ON and OFF medication. The red and blue dots correspond to negative and positive correlations, respectively. Small dots with light colors represent lower intensity correlations, and larger dots with darker colors correspond to higher intensity correlations. X means there is no statistical significance. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

suggest a relatively dependence between these domains. However, we cannot consider that the study's findings are contradictory since the parameters of length and time of the step and stride were not related to posture in both studies. A particular strength of this study is the focus on specific gait stability parameters – double and stance period and step width - which were not measured in the previous study. Thus, our findings extend the results of previous studies, indicating that more “posture-centric” gait outcomes, such as DST and step width, were related to body sway parameters.

ML sway was positively correlated with gait temporal sub-phases and step width, mainly in ON-state and OFF-state (i.e., stance phase). The findings of total double support and stance phase may be associated with instability in PD patients. In a systematic review, Zanardi et al. [14] found that the broad scale of disease progression (H&Y) shows an increase in differences for stance phase between PD individuals and healthy controls. Extended total double support may reflect an inability to adequately transfer weight mediolaterally to prepare for stepping, possibly compensating for the postural instability [15]. This supports the idea that ML stability is an essential property that sub-serves gait [16]. Studies in other pathologies, like in stroke [17], found that increased ML oscillation in the standing position may indicate gait instability. In addition, the ML measures were associated with a history

of falls and poor posture performance in older people [18], and PD [19]. Thus, we can suggest that a worse posture control in the ML direction can directly affect gait in PD patients.

Partially corroborating our hypothesis, there are a few different correlations according to the medication condition. In OFF medication, we found a positive correlation between total double support and RMS AP CoP. Corroborating our results, Sutter et al. [20] analyzed the relationship between gait and compensatory stepping responses in AP direction in PD patients in OFF medication. They found correlations between postural responses and gait speed and step length. The difference in the correlations between ON and OFF medication can be explained by the effect of medication on the spatial-temporal gait parameters and postural control domains. Mondal et al. [9] showed a decrease in the total double support in the ON medication. Thus, there is evidence that levodopa treatment appears to help postural sway early in the disease but worsens later [21]. These studies combined with our findings may suggest that levodopa medication can mainly influence the ML posture control, compared to AP posture; however, further investigation should be made. Another point to consider is that levodopa is most likely to impact gait, but not bipodal posture; this may hypothesize those correlations would be less pronounced in ON medication.

The sample was relatively limited in size and heterogeneous. Future studies could expand the sample to confirm our findings. Also, multiple correlations were performed with a reasonably small cohort, especially with multiple covariates. We acknowledge that the lack of correction for multiple comparisons increased the chance of type II error. However, this exploratory study and power analysis showed that our analysis had significance. So, our conclusions should be considered cautiously. As main conclusions, our study highlighted the correlation between postural control and gait domains, especially for gait temporal sub-phases and step width. As an implication for rehabilitation, the correlations of postural control and gait parameters are essential for assessing and rehabilitating patients because a reliable correlation could mean that resources used to improve posture could also influence gait.

Full financial disclosures

This study was supported by Fundação de Amparo à Pesquisa do Estado de São Paulo from Brazil (#2014/13502-7, #2015/14810-0, and #2019/06604-1).

CRedit authorship contribution statement

Thaisy Moraes Costa: Conceptualization, Data curation, Methodology, Writing – original draft. **Lucas Simieli:** Data curation, Methodology, Writing – original draft. **Felipe Marrese Bersotti:** Writing – original draft. **Luis Mochizuki:** Visualization, Writing – original draft, Writing – review & editing. **Fabio Augusto Barbieri:** Conceptualization, Visualization, Writing – original draft, Writing – review & editing. **Daniel Boari Coelho:** Conceptualization, Data curation, Formal analysis, Funding acquisition, Investigation, Methodology, Project administration, Writing – original draft, Writing – review & editing.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.neulet.2022.136537>.

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