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Short communication



## Gait and axial postural abnormalities correlations in Parkinson's disease: A multicenter quantitative study

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## ABSTRACT

**Introduction:** Gait and axial postural abnormalities (PA) are common and disabling symptoms of Parkinson's disease (PD). The interplay between them has been poorly explored.

**Methods:** A standardized protocol encompassing videos and photos for posture and gait analysis of PD patients with a clinically defined PA (MDS-UPDRS-III item 3.13 > 0) was used in 6 movement disorder centers. A comprehensive evaluation was performed to clarify the association between gait performance and the presence and severity of PA.

**Results:** 225 PD patients were enrolled: 57 had severe PA, 149 mild PA, and 19 did not meet criteria for PA, according to a recent consensus agreement on PA definition. PD patients with severe PA were significantly older ( $p < 0.001$ ), with longer disease duration ( $p < 0.007$ ), worse MDS-UPDRS-II and -III scores and axial sub-scores ( $p < 0.0005$ ), higher LEDD ( $p < 0.002$ ) and HY stage ( $p < 0.0005$ ), and a significantly lower velocity ( $p < 0.001$ ) and cadence ( $p < 0.021$ ), if compared to mild PA patients. The multiple regression analysis evaluating gait parameters and degrees of trunk/neck flexion showed that higher degrees of lumbar anterior trunk flexion were correlated with lower step length (OR -0.244;  $p < 0.014$ ) and lower velocity (OR -0.005;  $p < 0.028$ ).

**Conclusions:** Our results highlight the possible impact of severe anterior trunk flexion on PD patients' gait, with a specific detrimental effect on gait velocity and step length. Personalized rehabilitation strategies should be elaborated based on the different features of PA, aiming to target a combined treatment of postural and specifically related gait pattern alterations.

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## 1. Introduction

Axial symptoms, including gait impairment, postural instability and postural abnormalities (PA), are common disorders in advanced Parkinson's disease (PD) [1]. These symptoms are a leading cause of falls, autonomy loss, and reduced quality of life (QoL), and are largely resistant to dopamine replacement therapy, although some of them can improve with adequate dopaminergic supply and high intensity physical exercise, early in the disease [2].

PA pathophysiology and management have been poorly investigated so far, and their role in influencing gait and balance parameters needs to be elucidated. In fact, only a few pilot studies with a small sample size evaluated the interplay between PA and gait in PD patients, suggesting a role for specific PA on gait performances and balance impairment [3–5]. A consensus agreement recently suggested new cut-off values to classify six PA in PD, envisioning a possible additional category of milder PA added to the classic severe PA category [6].

A deeper knowledge of the correlation between PA and gait performance could offer new insights on the pathophysiology of axial symptoms and provide useful information to improve rehabilitation programs.

Here, we aimed to clarify such correlations by quantitatively analyzing gait and posture of a large cohort of consecutively recruited PD patients.

## 2. Methods

### 2.1. Study design and study participants

A total sample of 326 consecutive PD outpatients attending six movement disorders centers in Italy, Germany, Portugal, Thailand, South Korea, and Saudi Arabia were screened for the presence of PA between May 2019 and May 2021 [7].

We included in the study patients with a diagnosis of idiopathic PD made according to the MDS clinical diagnostic criteria (2015), at least 3 years of disease duration, age less than 80 years, and any kind of abnormal posture defined as a Movement Disorder Society Unified Parkinson Disease Rating Scale (MDS-UPDRS III) item 3.13 posture score  $>0$ . Exclusion criteria included: a) concomitant neurologic diseases known to negatively affect posture (i.e., myopathy, motor neuron disease, myasthenia); b) a history of major spinal surgery or muscle and/or skeletal diseases; c) treatment with drugs potentially able to induce abnormal postures in the 6 months before enrollment (i.e., typical and atypical neuroleptics; tricyclic antidepressants; cholinesterase inhibitors; antiemetic drugs; lithium carbonate); d) clinical features consistent with a diagnosis of atypical parkinsonism.

These data were used in a recent multicenter observational study investigating the prevalence of PA in Asian and Caucasian PD patients [7]. The study was approved by the institutional review boards and all patients provided written informed consent.

### 2.2. Procedures

Patients were assessed on their usual drug treatment, carried out in the practical daily-On therapeutic condition during a single outpatient visit performed by a movement disorder specialist. Photographs, with patients in a standing position in frontal (posterior) and sagittal plane, and video recording were used to capture and objectively analyze the gait and PA by the Kinovea software, as previously reported [7].

### 2.3. Outcome measures

All patients underwent an extensive cross-sectional clinical assessment including demographic and clinical data, levodopa equivalent daily dose (LEDD), Hoehn and Yahr Stage (HY), MDS-UPDRS part II-III, and Parkinson's disease questionnaire 8 (PDQ-8) for QoL. PD phenotype

has been defined in agreement with the algorithm of Stebbins and colleagues as tremor dominant (TD) Postural instability/gait difficulty (PIGD) or mixed type [8]. Clinical asymmetry has been defined when right-left differences in resting tremor, bradykinesia and rigidity were  $\geq 5$  points on the MDS-UPDRS items 3.3 (neck rigidity), 3.4 (right and left finger tapping), 3.6 (right and left hand pronation and supination movements), 3.8 (right and left leg agility), 3.15 (right and left hand postural tremor), 3.16 (right and left hand kinetic tremor), and 3.17 rest tremor amplitude (RUE/LUE/RLE/LEL) [9].

A new consensus on cut-off values based on expert opinions for mild and severe PA of the MDS Task Force on Postural Abnormalities in Parkinsonism was adopted to define PA categories [6]. Accordingly, antecollis (AC) was diagnosed in patients with anterior neck flexion (ANF)  $> 45^\circ$ ; camptocormia (CC) with anterior trunk flexion ("ATF") with lumbar fulcrum  $> 30^\circ$  or ATF with thoracic fulcrum  $> 45^\circ$ ; Pisa syndrome (PS) with lateral trunk flexion  $> 10^\circ$ . A suggested milder entity of "Anterior neck flexion (ANF)" was diagnosed in patients with  $> 35$  to  $\leq 45^\circ$  of flexion; ATF in patients  $\geq 25$  to  $\leq 45^\circ$  (thoracic fulcrum) or  $> 15$  to  $\leq 30^\circ$  (lumbar fulcrum), "lateral trunk flexion (LTF)" in patients with  $\geq 5$  to  $\leq 10^\circ$  [6]. The degrees of PA were evaluated following the previous studies [7,10].

Regarding gait performance analysis, step length, step variability, velocity and cadence have been chosen, as the most commonly impaired gait parameters in PD patients if compared to healthy controls and as they could be calculated in Off-line video recording by the Kinovea software [7,10].

### 2.4. Statistical analysis

Group comparisons were performed by a Chi-square test for categorical data and Mann-Whitney U and Kruskal-Wallis with Bonferroni adjustment for continuous variables. Pearson's coefficient was used to analyze correlations between gait parameters and degrees of trunk/neck flexion. Univariate linear regression models with step length, step variability, velocity, and cadence as dependent variables and the postural angles as well as the main demographic and clinical features as independent variables were used to calculate unadjusted odds ratio (OR; 95% confidence interval [CI]). Multiple linear regression models, adjusted for sex, disease duration, and age, using step length, step variability, velocity, and cadence as dependent variables and clinical features with  $p < 0.2$  at the univariate linear regression analyses, were used to calculate an adjusted OR (95% CI) for all possible confounding effects. All tests were two-tailed with a P-value set at 0.05. Statistical analyses were performed using SPSS statistical software.

## 3. Results

We included in the study 225 from 326 screened PD patients according to the inclusion criteria. Fifty-seven patients had severe PA (AC, CC, or PS) and 149 patients mild PA (ANF, ATF, or LTF), while 19 patients did not meet criteria for PA and were excluded from further analyses. Thirty-eight out of 225 PD patients had isolated severe PA (16.9%), namely AC ( $n = 31$ ; 13.8%), CC ( $n = 7$ ; 3.1%), of which 1 with lumbar fulcrum (0.4%) and 6 with thoracic fulcrum (2.7%), and 0 isolated PS. Nineteen patients (8.4%) showed a combination of two or more severe PA, with six patients showing PS combined with another PA (Table S1). Among patients with mild PA, 41 had isolated mild PA (27.5%), namely ANF ( $n = 5$ ; 2.2%), ATF ( $n = 30$ ; 13.3%), of which 0 with lumbar fulcrum and 30 with thoracic fulcrum (13.3%), and 6 LTF (2.7%) (Table S1); 108 patients (48%) showed a combination of two or more mild PA (Table S1).

PD patients with severe PA were significantly older ( $p: 0.001$ ), with longer disease duration ( $p: 0.007$ ), worse MDS-UPDRS II and III scores and axial sub-scores ( $p < 0.0005$ ), lower motor complications ( $p: 0.026$ ), higher LEDD ( $p: 0.002$ ) and H&Y stage ( $p < 0.0005$ ) compared to patients with mild PA (Table 1). Moreover, severe PA group had a

**Table 1**

Comparison of demographic and clinical findings between patients with mild and severe axial postural abnormalities.

	Mild PA	Severe PA	P-value mild vs. severe
<b>Patients, n</b>	149	57	
<b>Gender, n (%)</b>			<b>&lt;0.0005</b>
Male	78 (52.3%)	41(71.9%)	
Female	71(47.7%)	16 (28.1%)	
<b>Age, years, mean (SD)</b>	62.93 (9.16)	68.04 (7.51)	<b>0.001</b>
<b>Age of PD onset, years, mean (SD)</b>	55.5 (10.47)	58.75 (9.33)	
<b>Disease duration, years, mean (SD)</b>	7.41 (4.2)	9.3 (4.89)	<b>0.007</b>
<b>H&amp;Y stage, mean (SD)</b>	2.24 (0.78)	2.79 (0.67)	<b>&lt;0.0005</b>
<b>MDS-UPDRS score, mean (SD)</b>			
II	10.93 (6.21)	16.42 (9.65)	<b>&lt;0.0005</b>
III	26.58 (12.77)	35.68 (13.67)	<b>&lt;0.0005</b>
Axial	8.07 (3.83)	13.21 (5.17)	<b>&lt;0.0005</b>
IV	4.31 (4.15)	2.84 (3.46)	<b>0.026</b>
<b>Dominant phenotype, n (%)</b>			<b>0.028</b>
PIGD	63 (42.3%)	37 (64.9%)	
Tremor	71 (47.7%)	15 (26.3%)	
Mixed	15(10.1%)	5 (8.8%)	
<b>Lateral of PD onset, n (%)</b>			0.952
Right	87 (58.4%)	31 (54.4%)	
Left	55 (36.9%)	24 (42.1%)	
Bilateral	7 (4.7%)	2 (3.5%)	
<b>Clinical asymmetry, n (%)</b>			0.254
Symmetry	93 (62.4%)	42 (73.7%)	
Asymmetry	56 (37.6%)	15 (26.3%)	
<b>PDQ-8, mean (SD)</b>	21.54 (15.55)	27.25 (17.43)	0.1
<b>LEDD, mg, mean (SD)</b>	673.43 (410.4)	868.46 (385.12)	<b>0.002</b>
<b>LEDD, mg, mean (SD)/DAAs</b>	182.75 (123.58)	214.64 (152.79)	0.309
<b>LEDD, mg, mean (SD)/L-dopa</b>	495.74 (360.81)	621.74 (376.70)	<b>0.024</b>
<b>Ongoing Pharmaceutical treatment, n (%)</b>			0.465
L-dopa	45 (30.2%)	23 (40.4%)	
DAAs	2 (1.3%)	1 (1.8%)	
L-dopa+DAAs	35 (23.5%)	9 (15.8%)	
L-dopa+ MAO-B Is/COMT-Is	67 (45%)	24 (42.1%)	

PA: postural abnormalities. Mild PA: Axial PA with Anterior neck flexion (ANF) in patients with  $>35$  to  $\leq 45^\circ$  of flexion; Anterior trunk flexion (ATF) in patients with  $\geq 25$  to  $\leq 45^\circ$  (thoracic fulcrum) or  $>15$  to  $\leq 30^\circ$  (lumbar fulcrum), Lateral trunk flexion (LTF) in patients with  $\geq 5$  to  $\leq 10^\circ$ .

Severe PA: Axial PA with antecollis (AC) in patients with anterior neck flexion (ANF)  $> 45^\circ$ ; camptocormia (CC) with anterior trunk flexion ("ATF") with lumbar fulcrum  $>30^\circ$  or ATF with thoracic fulcrum  $>45^\circ$ ; Pisa syndrome (PS) with lateral trunk flexion  $>10^\circ$ . DAA: dopamine-agonist; MAO-B Is: Monoamine oxidase-B inhibitors; COMT-I: catechol-o-methyl-transferase inhibitors.

PA: postural abnormalities; H&Y: Hoehn and Yahr scale; MDS-UPDRS: International Parkinson and Movement Disorder Society – Unified Parkinson's Disease Rating Scale; PIGD: Postural instability/gait difficulty; PDQ-8: Parkinson's Disease Questionnaire-8; LEDD: L-dopa equivalent daily dose. Bold indicates significant p-values.

significantly higher number of PIGD phenotypes and higher rate of male patients (p: 0.028 and p < 0.0005, respectively) (Table 1).

### 3.1. Gait parameters and correlations with PA

PD patients with severe PA had a significantly lower velocity (p < 0.001) and cadence (p: 0.021) than patients with mild PA (Table 2). Correlations and univariate analysis results for gait parameters and degree of trunk and neck flexion are reported in Supplementary material (Table S2-S3-S4).

The multiple regression analysis correlating gait parameters with

**Table 2**

Posture and gait characteristic of Parkinson's disease patients with mild and severe axial postural abnormalities.

	Mild PA	Severe PA	P-value mild vs severe
<b>Anterior Neck Flexion (<math>^\circ</math>), mean (SD)</b>	36.91 (5.56)	53.93 (9.84)	<b>&lt;0.001</b>
<b>Anterior Trunk Flexion (LF) (<math>^\circ</math>), mean (SD)</b>	13 (5.51)	21.19 (11.57)	<b>&lt;0.001</b>
<b>Anterior Trunk Flexion (TF) (<math>^\circ</math>), mean (SD)</b>	31.13 (6.01)	39.32 (11.95)	<b>&lt;0.001</b>
<b>Lateral Trunk Flexion (<math>^\circ</math>), mean (SD)</b>	3.13 (1.99)	4.74 (4.41)	<b>0.04</b>
<b>Step length, cm, mean (SD)</b>	50.8 (10.19)	43.58 (12.54)	<b>&lt;0.001</b>
<b>Step variability, %CV, mean (SD)</b>	6.21 (3.96)	10.69 (17.92)	0.108
<b>Velocity, m/s, mean (SD)</b>	0.94 (0.22)	0.76 (0.24)	<b>&lt;0.001</b>
<b>Cadence, steps/min, mean (SD)</b>	111.2 (13.49)	106.89 (19.17)	<b>0.021</b>
<b>Latency to develop PA, y, mean (SD)</b>	3.94 (3.96)	4.5 (4.27)	0.461
<b>PA duration, y, mean (SD)</b>	3.92 (14.85)	3.86(4.75)	0.144

PA: Postural abnormalities. Step length was obtained after calibrating the height of the patient. The distance between the point of initial contact of one foot and the point of initial contact of the opposite foot as step length. Step variability was defined by using coefficient of variation of step lengths. Velocity was the product of cadence and step length. Cadence was calculated in steps per minute. Bold indicates significant p-values.

degrees of trunk and neck flexion showed that only the degrees of anterior trunk flexion with lumbar fulcrum correlated with gait features, with higher degrees of flexion being correlated with lower step length (adjusted OR -0.244; p: 0.014) and lower velocity (adjusted OR -0.005; p: 0.028) (Table 3). The same analysis showed that other demographic/clinical aspects were significantly correlated with gait parameters: male gender was associated with lower step length and velocity, an older age with lower step length and lower step variability, and a higher axial sub-score with lower step length, higher step variability and lower velocity (Table 3).

## 4. Discussion

We analyzed 225 PD patients with different degrees of PA by a video-assisted gait and posture analysis and found that higher degrees of ATF with lumbar fulcrum were associated with lower step length and lower gait velocity. On the contrary, degrees of LTF, ANF, and ATF with a thoracic fulcrum did not have a direct impact on gait parameters. Factors like male gender, an older age, and a higher clinical axial score also correlated with specific gait features. Finally, dividing PD patients in severe vs. mild PA according to the newly proposed MDS Task Force consensus based on expert opinion, we observed that those with severe PA were significantly older, with longer disease duration, more advanced disease stage, worse motor scores, and a higher load of dopaminergic therapy, but a lower degree of motor complications.

Our findings suggest that patients with severe forms of isolated or combined PA, namely AC, CC, and PS, have a more impaired gait, particularly velocity, step length, and cadence. However, these patients also have a more advanced and severe form of PD, probably accounting for worse gait performances than the severity of PA itself. Indeed, the multivariate regression analysis adjusted for demographic and clinical confounders, showed that only the degree of ATF with lumbar fulcrum is significantly correlated with step length and velocity.

These results provide a better characterization of patients with severe PA, indicating a complex interplay between PA and gait. While confirming that gait can be more impaired in a subgroup of patients with

**Table 3**  
Multivariate regression analysis results for the correlation between gait parameters, degrees of trunk and neck flexion, and clinical/demographic data.

	Step length			Step variability			Velocity			Cadence		
	Adjusted OR			Adjusted OR			Adjusted OR			Adjusted OR		
	OR	95% CI	P-value	OR	95% CI	P-value	OR	95% CI	P-value	OR	95% CI	P-value
Gender, Male vs Female	-6.286	-9.042 - (-3.531)	<0.001	1.257	-1.48 - 3.993	0.366	-0.068	-0.132 - (-0.005)	0.035	3.998	-0.508 - 8.503	0.082
Age	0.17	-0.322 - (-0.018)	0.028	-0.185	-0.335 - (-0.034)	0.016	-0.001	-0.005 - 0.002	0.507	0.213	-0.032 - 0.458	0.088
Disease duration	0.102	-0.213 - 0.417	0.524	-0.055	-0.372 - 0.261	0.731	0.002	-0.005 - 0.009	0.504	0.307	-0.16 - 0.775	0.197
H&Y stage	0.004	-2.112 - 2.12	0.997	-0.774	-2.857 - 1.37	0.489	0.017	-0.03 - 0.065	0.476	1.096	-2.287 - 4.478	0.524
MDS-UPDRS score												
II	-0.234	-0.448 - (-0.02)	0.032	0.156	-0.059 - 0.372	0.154	-0.004	-0.009 - 0.001	0.136	0.049	-0.297 - 0.395	0.779
III	-0.063	-0.187 - 0.062	0.321	-0.043	-0.168 - 0.083	0.506	-0.002	-0.005 - 0.001	0.235	-0.137	-0.338 - 0.063	0.179
Axial	-0.513	-0.941 - (-0.084)	0.019	0.904	0.477 - 1.331	<0.001	-0.012	-0.022 - (-0.003)	0.012	-0.216	-0.886 - 0.454	0.526
Dominant phenotype <sup>a</sup>	1.057	-0.993 - 3.108	0.311	0.187	1.857 - 2.23	0.857	0.016	-0.03 - 0.062	0.494			
LEDD	-0.001	-0.005 - 0.003	0.623	-4E-04	-0.004 - 0.003	0.857	-9E-06	-0.00009 - 0.00008	0.838			
ANF	-0.007	-0.159 - 0.146	0.931	0.057	-0.085 - 0.2	0.428	-0.002	-0.006 - 0.002	0.272	-0.21	-0.453 - 0.033	0.089
ATF (LF)	-0.244	-0.438 - (-0.049)	0.014				-0.005	-0.01 - (-0.001)	0.028			
ATF (TF)				0.209	-0.257 - 0.676	0.377	-0.001	-0.004 - 0.003	0.763	-0.176	0.442 - 0.091	0.195
LTF	-0.074	-0.543 - 0.394	0.755				-0.002	-0.013 - 0.008	0.669	-0.34	-1.089 - 0.41	0.372

ANF: Anterior Neck flexion; ATF (LF): Anterior trunk flexion (lumbar fulcrum); ATF (TF): Anterior trunk flexion (thoracic fulcrum); LTF: Lateral trunk flexion.

Bold indicates significant p-values.

<sup>a</sup> Dominant phenotype, PIGD vs Tremor vs Mixed.

severe PA if compared to those with milder PA, our study endorses the hypothesis that multiple factors may contribute to the correlation between posture and gait alterations; in fact, this correlation could be partly due to a phenotypic predisposition to a more aggressive PD phenotype and partly due to specific pathophysiological changes resulting in both abnormal trunk flexion and gait parameters [10,11].

Similar findings were observed in other studies, where patients with an abnormal ATF showed a slower walking speed and a shorter step length than PD patients without PA [3,4]. Moreover, a previous pilot study compared data of stabilometric and gait analysis of PD patients with and without PS, showing that PS patients had a greater body sway velocity for the anteroposterior and medial-lateral directions to maintain postural uprightiness, despite being able to maintain their ability to walk thanks to compensatory strategies [5]. Here, we found specifically that the degrees of ATF with lumbar fulcrum is inversely correlated with the step length and gait velocity. It can be hypothesized that the lower trunk flexion has a biomechanical impact on the iliopsoas muscle function, reducing the hip extension, which in turn impacts the step length and gait speed [3]. Another not-mutually exclusive explanation of this association is related to the center of pressure forward displacement, leading to an imbalance with an obvious impact on gait. The recent separation of camptocormia into two possibly different entities (upper/thoracic and lower/lumbar) still does not have its clinical underpinnings. We believe that the association we found between lumbar ATF and gait performance can be useful to foster studies on the different impact of these two entities and to provide useful information on specific rehabilitation programs.

The main strengths of this study are the large sample size, the quantitative assessment of both PA and gait with the same standardized methodology in a multicenter study, and the adoption of the recently proposed cut-off values for PA definition [6]. The main limitations are 1) a video software for gait assessment, which is not as accurate as the gold standard represented by infrared cameras, in spite of having been already used for gait analysis in PD; 2) the lack of a control group of PD patients without clinically defined PA. However, the fact that we have enrolled only patients with a probable clinical diagnosis of PA (i.e., MDS-UPDRS posture item 3.13  $\geq$  1) allowed us to analyze the impact of PA on gait in a group of patients with different severity of PA (severe and mild forms of PA) likely emphasizing the finding related to the impact of CC on gait. Additionally, comparing the two PA groups, we observed a sort of parallel “progression” of PA and gait alterations, in spite of the cross-sectional study design. 3) We have found a remarkably small number of patients with PS probably hampering the analysis of the impact of lateral trunk flexion on gait. Additionally, this low prevalence may question the real representativity of our cohort. However, we had a prevalence of 2.6% of combined PS, which could be still in line with the heterogeneous literature data [7]. Finally, it must be emphasized that the cut-off values used here are currently based on expert judgment and not on empirical validation. The only exception is the cut-off criterion for anterior trunk flexion with lumbar fulcrum [12].

These limitations notwithstanding, our results highlight the interplay between severe PA and PD patients’ gait, with a specific detrimental effect on gait velocity and step length related to the severity of CC with lumbar fulcrum. These findings contribute to a better comprehension of the disability provided by severe PA and underline the importance of finding adequate therapeutic and prevention strategies for these disabling PD symptoms. Personalized rehabilitation approaches should be elaborated based on the different features of PA, aiming to target both gait and posture to improve reactive postural control with ad-hoc approaches encompassing motor-cognitive dual tasking and sensorimotor retraining [13,14].

**Authorship disclosure**

All authors have materially participated in the research and/or article preparation (see roles, below). All authors have approved the

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## Ethical compliance statement

The patient gave written consent to participate in the study. We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this work is consistent with those guidelines.

## Declaration of conflict of 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.

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## Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.parkreldis.2022.10.026>.

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