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ORIGINAL ARTICLE

Gait Instability and Compensatory Mechanisms in Parkinson's Disease Patients With Camptocormia: An Exploratory Study

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ABSTRACT

Objective Camptocormia contributes to vertical gait instability and, at times, may also lead to forward instability in experimental settings in Parkinson's disease (PD) patients. However, these aspects, along with compensatory mechanisms, remain largely unexplored. This study comprehensively investigated gait instability and compensatory strategies in PD patients with camptocormia (PD+CC).

Methods Ten PD+CC patients, 30 without camptocormia (PD-CC), and 27 healthy controls (HCs) participated. Self-paced gait tasks were analyzed using three-dimensional motion capture systems to assess gait stability as well as spatiotemporal and kinematic parameters. Unique cases with pronounced forward gait stability or instability were first identified, followed by group comparisons. Correlation analysis was performed to examine associations between trunk flexion angles (lower/upper) and gait parameters. The significance level was set at 0.05.

Results Excluding one unique case, the PD+CC group presented a significantly lower vertical center of mass (COM) position (p=0.019) increased mediolateral COM velocity (p=0.004) and step width (p=0.013), compared to the PD-CC group. Both PD groups presented greater anterior-posterior margins of stability than did the HCs (p<0.001). Significant correlations were found between lower/upper trunk flexion angles and a lower vertical COM position (r=-0.690/-0.332), as well as increased mediolateral COM velocity (r=0.374/0.446) and step width (r=0.580/0.474).

Conclusion Most PD+CC patients presented vertical gait instability, increased fall risk, and adopted compensatory strategies involving greater lateral COM shift and a wider base of support, with these trends intensifying as trunk flexion angles increased. These findings may guide targeted interventions for gait instability in PD+CC patients.

Keywords Parkinson's disease; Postural abnormalities; Camptocormia; Gait instability; Motion analysis.

INTRODUCTION

Camptocormia, a postural abnormality that can develop in individuals with Parkinson's disease (PD), is characterized by excessive thoracolumbar flexion while standing and walking.[1-10] PD patients with camptocormia (PD+CC) often experience in-

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stability while standing and walking,[1-4] which increases their reliance on walking aids.[5] Studies have shown that the center of mass (COM) in PD+CC tends to lower during standing and walking,[4,6] indicating proximity to the ground and a higher risk of falls. While prior article studies have suggested that both camptocormia and freezing of gait (FOG) contribute to forward gait instability in PD patients,[11] the specific role of camptocormia in this context has not been objectively validated. Additionally, PD+CC show greater lateral instability while standing.[1] These observations underscore the need for experimental investigations of gait instability in PD+CC.

Objective evaluations of gait instability involve 1) using a three-dimensional motion analysis system to measure the COM position and velocity during walking and 2) calculating the distance between the COM and base of support (COM-BOS distance) and the margin of stability (MOS).[12-14] Two phenotypes of forward gait instability in PD patients have been proposed: one associated with FOG and the other due to forward-leaning postures such as camptocormia.[11] Our recent results demonstrated that PD patients with FOG have reduced COM-BOS distances and reduced MOS, indicating greater forward gait instability than those without FOG.[12] However, gait instability in PD+CC, as well as related spatiotemporal and kinematic parameters, have not been studied. Camptocormia can be classified into lower and upper types,[8] but their relationships with flexion angles remain unclear.

Forward gait instability is often undetected in clinical or experimental settings, even in patients who frequently fall forward. A recent review of MOS in patients with neurological disorders revealed variability in gait stability,[15] potentially due to compensatory strategies influenced by the experimental environment or psychological state. Considering the variability in gait instability related to compensatory strategies, a comprehensive examination of these factors could enhance the understanding of gait instability in PD+CC. This study aimed to provide the first comprehensive and individualized analysis of gait instability and compensatory strategies in PD+CC.

MATERIALS & METHODS

Study design and participants

This multicenter, cross-sectional study enrolled 35 consecutive inpatients with PD who were examined at Osaka Medical and Pharmaceutical University Hospital from June 2018 to November 2023 and five consecutive inpatients with both PD and camptocormia assessed at Mihara Memorial Hospital from April 2022 to November 2023. Recruitment was paused from February 2020 to December 2021 because of the COVID-19 pandemic.

The inclusion criteria for PD patients were as follows: 1) idiopathic PD based on the International Parkinson and Movement Disorder Society clinical diagnostic criteria,[16] 2) ability to walk independently for more than 10 m without the use of a walking aid, 3) absence of visual, vestibular, muscular, spinal orthopedic, or neurological disorders other than PD that could affect ambulation, 4) no significant dyskinesia, and 5) ability to understand verbal instructions. Twenty-seven age-matched healthy controls (HCs) were recruited from the vicinity of Kio University to serve as a control group for comparison. The inclusion criteria for HCs were as follows: 1) ability to walk >10 m independently without a walking aid, 2) no visual, vestibular, muscular, spinal orthopedic, or neurological disorders that could affect ambulation, and 3) ability to understand verbal instructions.

The study was approved by the Ethics Committees of Osaka Medical and Pharmaceutical University Hospital (approval no. 2440-2), Kio University (approval no. H30-37), and the Institute of Brain and Blood Vessels (approval no. 112-07). All participants provided written informed consent in accordance with the Declaration of Helsinki.

Clinical assessments

The following data were collected for all of the participants: age, gender, height, body mass index (BMI), cognitive function assessed by the Mini-Mental State Examination (MMSE),[17] and fall history over the previous 6 months, including the fall direction(s). For PD patients, additional assessments included disease duration, Hoehn and Yahr stages, and severity of motor symptoms according to the Movement Disorders Society-sponsored revision of the Unified Parkinson's Disease Rating Scale motor disability scale (MDS-UPDRS-III) score, which was administered by certified researchers.[18] Subscores were calculated for tremor (subitems 3.15–3.18), bradykinesia (subitems 3.2, 3.4–3.8, and 3.14), rigidity (subitem 3.3), and postural instability and gait difficulty (subitems 3.9–10, 3.12, excluding subitem 3.13, which indicates posture).[19] An assessment of FOG severity was also conducted for PD patients with the New Freezing of Gait Questionnaire (NFOG-Q) score,[20] and the levodopa equivalent daily dose (LEDD) of each participant with PD was calculated.[21]

Procedures

The participants wore tight-fitting attire and walked a 5-m distance at their preferred speed while barefoot, and the trial was repeated three times. At the start of each trial, the participant stood still for at least 3 seconds before walking. Gait analyses were conducted using a motion capture system (Vicon, Oxford Metrics) at each center for the PD patients and at Kio University for the HCs. Forty-one reflective markers, including the fifth lumbar spinous process and thoracic flexion fulcrum, were

placed by well-trained specialists (H.U. and Y.O.) on specific landmarks on the participant's body according to the VICON Plug-in Gait model.[22,23] The first two and last two steps of each trial were excluded from the analysis. Data were captured at a sampling frequency of 100 Hz and smoothed using a fourth-order Butterworth filter with zero lag and a cutoff frequency of 10 Hz.[12,24,25] The average values from the three trials were taken as the representative values.

Group classification of PD patients

We divided the PD patients into two groups, PD+CC (n=10) and those without camptocormia (PD-CC) (n=30), and used a cutoff value for diagnosing camptocormia. Camptocormia was diagnosed as lower-trunk flexion exceeding 30° or upper-trunk flexion exceeding 45°. These cutoff values were established on the basis of an expert consensus issued by the Movement Disorders Society Task Force on Postural Abnormalities in Parkinsonism.[8] On the basis of this expert consensus, the lower-trunk flexion angle was calculated using the "malleolus method," and the upper-trunk flexion angle was calculated using the "upper-camptocormia method" for the participant's standing posture angle. The PD participants were assessed during the practical ON medication state, approximately 60–90 min after their last dose of dopaminergic medication.

Outcome measures

The following gait stability parameters were quantified: COM positions in the anterior-posterior (AP), mediolateral (ML), and vertical (VT) directions; COM-BOS distance; COM velocity (COM vel); and MOS values in the AP and ML directions. The whole-body COM location was calculated by Nexus software (ver. 2.7, Oxford Metrics) as the weighted sum of the COMs of the 15 body segments. The BOS values in the AP/ML directions were defined by four markers attached to the second metatarsal head and the lateral malleoli of both feet.

To evaluate the COM's location relative to the BOS at heel contact, we measured the percentage distribution of the COM's AP position toward the front and rear and that of the ML position toward the medial and lateral BOS (COM position$_{AP/ML}$). Larger values indicate that the COM is closer to the leading limb's BOS. For the VT direction (COM position$_{VT}$), we measured the distance from the COM to the floor at heel contact and normalized it by the participant's height.

The COM-BOS distances in the AP/ML directions were calculated as the distance from the COM to the former second metatarsal head/lateral malleolus marker at heel contact.[12,25]

Similarly, the MOS values in the AP/ML directions were determined as the distance from the extrapolated COM (xCOM) to the same marker at heel contact via the following formula[14]:

$$MOS_{AP/ML}=BOS_{AP/ML}-xCOM_{AP/ML}.$$

The xCOM, reflecting the COM vel, was calculated as follows:

$$xCOM_{AP/ML}=COM_{AP/ML}+\frac{COM\ vel_{AP/ML}}{\sqrt{\frac{g}{l}}}.$$

Here, $COM_{AP/ML}$ and $COM\ vel_{AP/ML}$ represent the position and velocity of the COM in the AP/ML directions at heel contact, respectively. The value "l" is the distance between the COM and the lateral malleolus, and "g" represents gravitational acceleration. The COM-BOS distance indicates the risk of a loss of balance, whereas the MOS represents instantaneous mechanical gait instability.[13] Figure 1 shows a schematic diagram of the MOS.

We measured each participant's gait speed, step length, step width, cadence, and percentage of single and double support times within the gait cycle as spatiotemporal gait parameters. Nexus software was used to define gait events (i.e., heel contact and toe-off) and to calculate the spatiotemporal gait parameters. For kinematic parameters, we calculated the angles of the pelvis and each joint of the leading and trailing limbs at heel contact. We also measured the range of motion throughout the gait cycle. These angles were calculated with Nexus software. The upper-trunk flexion angle during gait, as well as during standing, was calculated by the "upper-camptocormia method." For the lower-trunk flexion angle, the "perpendicular method" was used instead of the "malleolar method" because of foot movement.[22,26]

Statistical analysis

Demographic and clinical characteristics among the three groups (PD+CC, PD-CC, and HCs) were compared using the Kruskal–Wallis test and Fisher's exact test, plus the Mann–Whitney U test for comparisons between PD groups. Fisher's exact test was used to compare the proportions of males and females and those of fallers/nonfallers across the three groups. Comparisons of the side of PD symptom onset, the Hoehn and Yahr stages, and the proportions of freezers and nonfreezers in the two PD groups were also performed via Fisher's exact test. The Crawford-Howell modified t-test was used to compare the MOS_AP values between the PD patients and the HCs,[27] aiming to detect any unique patients with significant forward gait stability or instability. We used the Kruskal–Wallis H test and Quade's rank analysis of covariance (ANCOVA) to compare gait parameters. FOG severity, previously identified as a confounder of gait stability and as measured by the NFOG-Q score,[12] was included as a covariate. Other demographic and clinical characteristics that were significantly different between groups were also incorporated as covariates. We conducted partial correlation analysis adjusted for the NFOG-Q score to investigate the impact of trunk flexion an-

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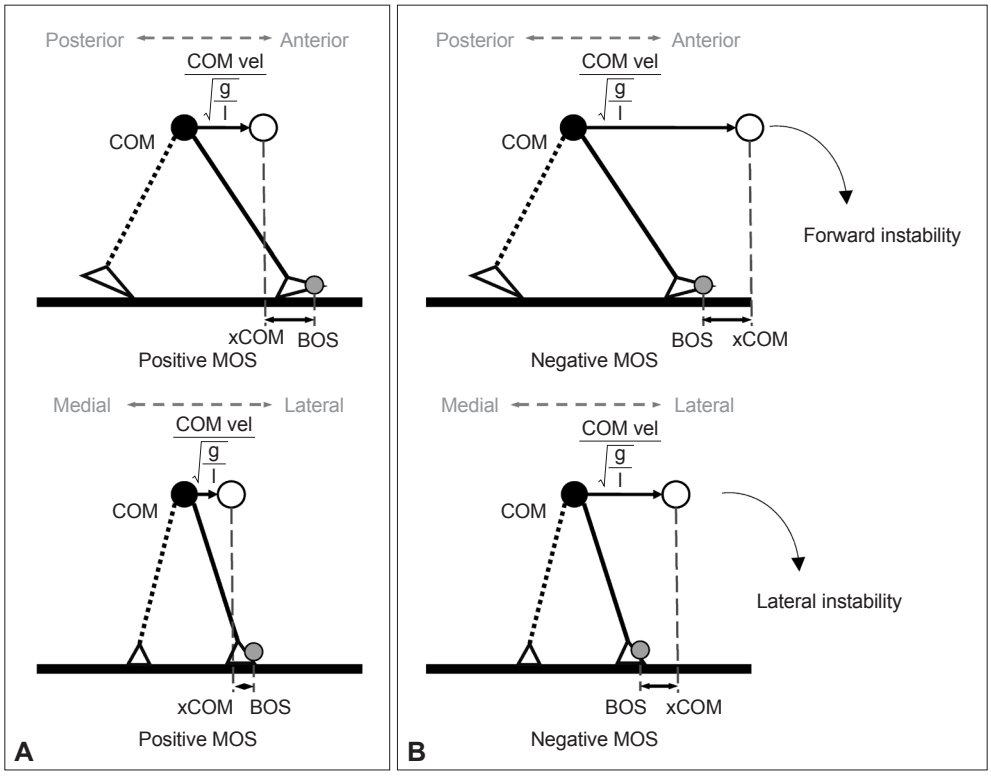

Figure 1. The MOS in the AP and ML directions (bottom). $MOS_{AP/ML}$ was calculated as the distance from the extrapolated xCOM to the boundary of the BOS. Note the positional shifts of the xCOM with varying COM vel values. A: When the xCOM is within the boundary of the positive MOS (BOS), it indicates mechanical stability. B: A deviation of the xCOM beyond the BOS (negative MOS) indicates mechanical instability in the inverted pendulum mpdel. COM, center of mass; vel, velocity; xCOM, extrapolated center of mass; BOS, base of support; MOS, margin of stability; AP, anterior-posterior; ML, mediolateral.

gle, including subdiagnostic camptocormia, on gait stability and spatiotemporal parameters. All the statistical analyses were conducted using SPSS Statistics (ver. 29; IBM Corp.) for most analyses and with open-source R software (https://www.r-project.org/) for the Crawford-Howell modified *t*-tests. The significance level was set at $p<0.05$ for all analyses.

RESULTS

All of the participants successfully completed the experimental trials with no instances of FOG or falls. The Crawford–Howell modified *t*-tests revealed no significant difference in MOS_{AP} values between the 39 PD patients and the HCs ($p>0.05$). However, one participant in the PD+CC group (patient 3 from Supplementary Video 1 [in the online-only Data Supplement]) exhibited a significantly lower MOS_{AP} than did the HCs ($p=0.041$). Supplementary Video 1 (in the online-only Data Supplement) shows his gait alongside that of other PD+CC and PD-CC for comparison. Because of this participant's unique forward gait instability, we conducted group comparisons, and partial correlation analyses were performed, excluding this participant.

Demographic and clinical characteristics

Table 1 summarizes the demographic and clinical characteristics of the three groups. There were no significant differences in age, gender, height, BMI, or MMSE scores among the three groups. The clinical characteristics of the PD+CC and PD-CC groups were not significantly different, except for the LEDD. A significant difference in fall history was observed among the three groups. Falls within the past 6 months were reported by 33.3% of the PD+CC patients, 26.7% of the PD-CC patients, and 3.7% of the HCs. Compared with the PD-CC and HC groups, the PD+CC group presented significantly greater lower- and upper-trunk flexion angles during standing, with the exception of the lateral trunk flexion angle.

Gait stability parameters

Significant between-group differences were observed in COM $position_{VT}$ ($\chi^2=7.921$), COM $position_{ML}$ ($\chi^2=16.203$), COM-BOS $distance_{AP}$ ($\chi^2=30.621$), COM vel (AP: $\chi^2=42.3381$; ML: $\chi^2=13.576$), and MOS_{AP} ($\chi^2=27.350$) (Table 2). Compared with the HCs, both the PD+CC and PD-CC groups presented a significant reduction in COM-BOS $distance_{AP}$ ($p<0.001$) and a significant increase in MOS_{AP} ($p<0.001$). COM vel_{AP} was sig-

Table 1. Demographic and clinical characteristics of the patients and the controls

	PD+CC (*n*=9)	PD-CC (*n*=30)	HCs (*n*=27)	*p* value group comparison
Age (yr)	71 (69, 75)	76 (73, 79)	72 (68, 74)	0.138†
Gender, male/female	7/2	20/10	12/15	0.124‡
Height (m)	1.65 (1.57, 1.67)	159.0 (1.54, 1.65)	1.58 (1.55, 1.66)	0.561†
BMI (kg/m^2)	21.8 (21.4, 22.8)	21.56 (20.0, 24.1)	20.5 (19.5, 25.1)	0.695†
MMSE score	28.0 (25.0, 29.0)	28.0 (25.0, 30.0)	28.0 (27.0, 29.5)	0.763†
Disease duration (months)	84.0 (39.0, 150.0)	45.5 (25.5, 79.3)	N/A	0.064§
LEDD (mg/day)	600.0 (375.0, 750.0)	300.00 (162.5, 614.7)	N/A	0.042*§
Hoehn and Yahr stage, II/III/IV	3/4/2	12/13/5	N/A	>0.999‡
MDS-UPDRS-III score	41.0 (28.0, 43.0)	34.0 (25.3, 40.8)	N/A	0.348§
Tremor-upper limb	0.0 (0.0, 5.0)	2.0 (0.0, 3.0)	N/A	1.000§
Tremor-lower limb	0.0 (0.0, 2.0)	0.0 (0.0, 0.8)	N/A	0.588§
Rigidity-neck	2.0 (1.0, 2.0)	1.0 (1.0, 2.0)	N/A	0.159§
Rigidity-upper limb	3.0 (2.0, 3.0)	3.0 (2.0, 4.0)	N/A	0.857§
Rigidity-lower limb	3.0 (2.0, 4.0)	3.0 (2.0, 4.0)	N/A	0.501§
Bradykinesia-upper limb	8.0 (6.0, 11.0)	8.0 (6.3, 11.0)	N/A	0.987§
Bradykinesia-lower limb	6.0 (3.0, 6.0)	6.0 (4.0, 7.0)	N/A	0.366§
PIGD, excluding subitem 3.13 (posture)	5.0 (3.0, 6.0)	5.0 (2.0, 7.0)	N/A	0.909§
PD symptoms onset side, R/L	5/4	13/17	N/A	0.706‡
Participants with freezing of gait	6 (66.7)	16 (53.3)	N/A	0.471‡
NFOG-Q score	12.0 (0.0, 24.0)	7.5 (0.0, 18.0)	N/A	0.384§
Participants with falls	3 (33.3)	8 (26.7)	1 (3.7)	0.026*‡
Falling direction				
Forward	1 (11.1)	6 (20.0)	1 (3.7)	N/A
Other direction	2 (22.2)	2 (6.7)	0 (0.0)	N/A
None	6 (66.7)	22 (73.3)	26 (96.3)	N/A
Standing posture angle				
Lower trunk flexion (degree)	51.91 (23.14, 57.95)	13.26 (9.25, 20.26)	3.55 (-0.01, 6.95)	<0.001*†
Upper trunk flexion (degree)	41.26 (38.38, 45.85)	23.84 (19.02, 27.88)	14.80 (11.67, 20.32)	<0.001*†
Lateral trunk flexion (degree)	3.18 (1.92, 3.74)	2.18 (1.13, 3.30)	2.99 (1.18, 4.29)	0.535†

Values are median (interquartile 25%, 75%) or number of participants (%).
*p<0.05; †Kruskal–Wallis H Test; ‡Fisher's exact Test; §Mann–Whitney U Test.
PD+CC, PD patients with camptocormia; PD-CC, PD patients without camptocormia; HCs, healthy controls; BMI, body mass index; MMSE, Mini-Mental State Examination; LEDD, levodopa equivalent daily dose; MDS-UPDRS-III, Movement Disorder Society-sponsored revision of the Unified Parkinson's Disease Rating Scale motor disability scale; PIGD, postural instability and gait difficulty; PD, Parkinson's disease; R/L, right/left; NFOG-Q, New Freezing of Gait Questionnaire; N/A, not applicable.

nificantly lower in both the PD+CC and PD-CC groups than in the HCs (both p<0.001).

After adjusting for the NFOG-Q score and LEDD values, Quade's rank ANCOVA revealed that the PD+CC group had significantly lower COM position$_{VT}$ (F=6.060) and COM position$_{AP}$ (F=4.630), as well as a higher COM vel$_{ML}$ (F=9.583), than did the PD-CC group. No significant differences were found in COM-BOS distance$_{AP}$ (F=0.455) or MOS$_{AP}$ (F=3.656) between the PD+CC and PD-CC groups.

Spatiotemporal gait parameters

We observed significant differences among the three groups in all spatiotemporal gait parameters (Table 2): gait speed (χ^2=41.032), step length (χ^2=42.493), step width (χ^2=10.039), cadence (χ^2=14.281), single support time (χ^2=13.153), and double support time (χ^2=14.628). Both PD groups presented significant decreases in gait speed, step length, cadence, and single support time, along with a significant increase in the double support time. Compared with the PD-CC group, the PD+CC group presented a significantly larger step width, whereas no significant differences in the other spatiotemporal parameters were found between the PD groups.

Quade's rank ANCOVA, adjusting for the NFOG-Q and LEDD, revealed a significantly wider step width (F=6.792) in the PD+CC group than in the PD-CC group. No significant differences in gait speed (F=1.255), step length (F=1.022), cadence

Table 2. Gait stability and spatiotemporal gait parameters

	PD+CC (*n*=9)	PD-CC (*n*=30)	HCs (*n*=27)	*p* value† group comparison	*p* value‡ (PD+CC vs. PD-CC)	*p* value‡ (PD+CC vs. Control)	*p* value‡ (PD-CC vs. Control)	*p* value§ adjusting for NFOG-Q and LEDD (PD+CC vs. PD-CC)
Gait stability parameters at heel contact								
COM position$_{VT}$ (% of height)	51.10 (48.85, 52.58)	54.13 (53.44, 55.16)	54.23 (53.24, 55.21)	0.019*	0.021*	0.030*	>0.999	0.019*
COM position$_{AP}$ (% of BOS)	51.49 (45.45, 53.63)	52.10 (49.84, 55.39)	53.27 (51.98, 55.80)	0.155	N/A	N/A	N/A	0.038*
COM position$_{ML}$ (% of BOS)	43.61 (39.17, 43.92)	42.86 (41.80, 44.50)	45.86 (42.18, 47.31)	<0.001*	1.000	0.020*	0.001*	0.320
COM-BOS distance$_{AP}$ (mm)	222.46 (188.13, 227.77)	273.35 (227.55, 294.21)	331.07 (302.01, 354.04)	<0.001*	0.787	<0.001*	<0.001*	0.504
COM-BOS distance$_{ML}$ (mm)	117.36 (111.01, 124.99)	101.04 (93.40, 121.65)	102.95 (98.57, 119.92)	0.190	N/A	N/A	N/A	0.189
COM vel$_{AP}$ (mm/sec)	389.58 (361.10, 607.11)	663.95 (488.12, 805.98)	1107.39 (1033.03, 1224.03)	<0.001*	0.625	<0.001*	<0.001*	0.210
COM vel$_{ML}$ (mm/sec)	109.17 (103.25, 125.47)	87.5 5 (73.05, 89.02)	76.01 (65.88, 89.02)	0.001*	0.014*	<0.001*	0.580	0.004*
MOS$_{AP}$ (mm)	88.03 (58.45, 95.69)	60.55 (42.67, 82.25)	-7.49 (-27.65, 27.23)	<0.001*	0.773	<0.001*	<0.001*	0.064
MOS$_{ML}$ (mm)	84.99 (76.55, 94.30)	78.02 (69.22, 92.63)	81.10 (77.60, 88.83)	0.441	N/A	N/A	N/A	0.603
Spatiotemporal gait parameters								
Gait speed (m/s)	0.40 (0.35, 0.60)	0.64 (0.49, 0.81)	1.09 (1.05, 1.23)	<0.001*	0.779	<0.001*	<0.001*	0.270
Step length (m)	0.30 (0.26, 0.35)	0.39 (0.32, 0.45)	0.59 (0.56, 0.62)	<0.001*	0.630	<0.001*	<0.001*	0.319
Step width (m)	0.12 (0.10, 0.18)	0.08 (0.06, 0.11)	0.07 (0.06, 0.10)	0.007*	0.008*	0.011*	>0.999	0.013*
Cadence (steps/min)	96.59 (91.96, 109.86)	102.39 (96.16, 111.10)	113.75 (108.73, 119.94)	<0.001*	>0.999	0.011*	0.003*	0.203
Single support time (% of gait cycle)	34.03 (30.92, 37.32)	35.83 (32.91, 37.27)	38.32 (36.82, 39.99)	0.001*	>0.999	0.020*	0.004*	0.897
Double support time (% of gait cycle)	34.40 (26.54, 37.59)	28.53 (25.18, 32.61)	22.39 (20.92, 25.49)	<0.001*	>0.999	0.013*	0.002*	0.834

Values are median (interquartile 25%, 75%).
*p<0.05; †Kruskal–Wallis H Test; ‡Mann–Whitney U Test; §Quade's rank ANCOVA.
PD+CC, PD patients with camptocormia; PD-CC, PD patients without camptocormia; HCs, healthy controls; NFOG-Q, New Freezing of Gait Questionnaire; LEDD, levodopa equivalent daily dose; COM, center of mass; VT, vertical; AP, anterior-posterior; BOS, base of support; ML, mediolateral; vel, velocity; MOS, margin of stability; N/A, not applicable; ANCOVA, analysis of covariance; PD, Parkinson's disease.

(F=1.682), single support time (F=0.017), or double support time (F=0.044) were detected between the PD groups.

Kinematic parameters

Significant differences were revealed among the three groups in lower-trunk flexion (χ^2=27.770) and upper-trunk flexion (χ^2=22.447), but no significant difference was detected in the lateral-trunk flexion angle (χ^2=5.172) (Table 3). Significant differences were also observed for the pelvis and lower-limb joint angles at heel contact: pelvis tilt (χ^2=6.233), leading-limb knee flexion (χ^2=23.890), trailing-limb hip flexion (χ^2=21.175), and trailing-limb knee flexion (χ^2=23.548). No significant differences were found in pelvis obliquity (χ^2=5.855), pelvis rotation (χ^2=1.051), leading-limb hip flexion (χ^2=5.442), or leading-limb hip abduction (χ^2=2.716).

The range of motion for upper-trunk flexion (χ^2=10.694) and lateral-trunk flexion (χ^2=8.374) during the gait cycle was significantly different among the groups. Significant differences were also demonstrated for pelvis obliquity (χ^2=27.523), pelvis rotation (χ^2=13.759), hip flexion/extension (χ^2=48.398), hip abduction/adduction (χ^2=6.590), and knee flexion/extension (χ^2=25.150) but not for lower-trunk flexion (χ^2=1.401) or pelvis tilt (χ^2=1.104). Compared with the PD-CC group, the PD+CC group presented significantly larger lower- and upper-trunk flex-

Table 3. Kinematic parameters at heel contact and joint range of motion in the gait cycle

	PD+CC (*n*=9)	PD-CC (*n*=30)	HCs (*n*=27)	*p* value† group comparison	*p* value‡ (PD+CC vs. PD-CC)	*p* value‡ (PD+CC vs. Control)	*p* value‡ (PD-CC vs. Control)	*p* value§ adjusting for NFOG-Q and LEDD (PD+CC vs. PD-CC)
At heel contact								
Lower trunk flexion (degree)	56.57 (20.02, 60.61)	10.83 (8.44, 15.03)	6.81 (6.16, 10.30)	<0.001*	0.003*	<0.001*	0.015*	<0.001*
Upper trunk flexion (degree)	44.21 (40.92, 46.08)	23.70 (15.82, 25.94)	17.45 (15.31, 20.95)	<0.001*	0.001*	<0.001*	0.215	<0.001*
Lateral trunk flexion (degree)	6.01 (3.18, 15.27)	1.31 (0.52, 2.26)	2.81 (0.91, 4.64)	0.075				0.194
Pelvis tilt (degree)	6.57 (-0.63, 14.53)	7.05 (3.64, 10.80)	11.04 (7.95, 14.07)	0.044*	>0.999	0.614	0.041*	0.951
Pelvis obliquity (degree)	2.79 (1.41, 3.01)	1.85 (0.97, 3.83)	1.07 (0.44, 2.09)	0.054	N/A	N/A	N/A	0.920
Pelvis rotation (degree)	1.98 (1.59, 5.19)	2.26 (1.10, 3.10)	1.64 (1.09, 2.84)	0.591	N/A	N/A	N/A	0.426
Leading limb hip flexion (degree)	22.96 (17.55, 35.50)	21.09 (18.36, 26.35)	27.87 (23.19, 29.67)	0.066	N/A	N/A	N/A	0.376
Leading limb hip abduction (degree)	0.11 (-2.66, 2.88)	2.74 (-0.36, 4.96)	1.02 (-0.86, 3.26)	0.257	N/A	N/A	N/A	0.324
Leading limb knee flexion (degree)	18.28 (11.23, 19.27)	10.59 (3.07, 15.58)	2.14 (-0.21, 5.03)	<0.001*	0.132	<0.001*	0.001*	0.144
Trailing limb hip flexion (degree)	4.31 (0.42, 18.98)	-2.12 (-8.08, 3.41)	-10.77 (-16.08, -6.82)	<0.001*	0.371	<0.001*	0.001*	0.146
Trailing limb knee flexion (degree)	21.82 (15.24, 25.27)	14.57 (9.45, 17.81)	5.73 (3.83, 11.08)	<0.001*	0.334	<0.001*	0.001*	0.180
Range of motion in the gait cycle								
Lower trunk flexion (degree)	3.24 (3.14, 3.81)	3.38 (2.82, 3.80)	3.69 (3.13, 4.26)	0.403	N/A	N/A	N/A	0.307
Upper trunk flexion (degree)	1.24 (0.96, 1.49)	1.78 (1.41, 3.16)	2.72 (1.90, 3.89)	0.005*	0.342	0.009*	0.088	0.108
Lateral trunk flexion (degree)	5.56 (1.98, 6.09)	3.22 (2.39, 3.84)	4.51 (3.23, 5.07)	0.016*	0.167	>0.999	0.023*	0.284
Pelvis tilt (degree)	3.57 (2.95, 4.79)	3.14 (2.77, 3.67)	3.19 (2.85, 3.60)	0.576	N/A	N/A	N/A	0.233
Pelvis obliquity (degree)	5.31 (2.91, 6.99)	3.54 (2.68, 4.74)	7.30 (6.57, 8.46)	<0.001*	0.624	0.055	<0.001*	0.029*
Pelvis rotation (degree)	7.77 (5.37, 7.98)	6.53 (4.79, 7.89)	9.90 (7.68, 11.01)	0.001*	>0.999	0.115	0.001*	0.337
Hip flexion/extension (degree)	21.10 (19.63, 23.91)	30.94 (26.50, 34.20)	41.54 (39.10, 47.50)	<0.001*	0.199	<0.001*	<0.001*	0.021*
Hip abduction/adduction (degree)	7.43 (6.18, 8.17)	8.84 (5.95, 10.33)	9.51 (8.25, 10.75)	0.037*	>0.999	0.082	0.129	0.960
Knee flexion/extension (degree)	35.54 (29.99, 39.31)	45.65 (39.83, 48.03)	53.21 (51.10, 58.56)	<0.001*	0.306	<0.001*	<0.001*	0.050*

Values are median (interquartile 25%, 75%).
*p<0.05; †Kruskal–Wallis H Test; ‡Mann–Whitney U Test; §Quade's rank ANCOVA.
PD+CC, PD patients with camptocormia; PD-CC, PD patients without camptocormia; HCs, healthy controls; NFOG-Q, New Freezing of Gait Questionnaire; LEDD, levodopa equivalent daily dose; N/A, not applicable; ANCOVA, analysis of covariance; PD, Parkinson's disease.

ion angles at heel contact but not other kinematic parameters.

Quade's rank ANCOVA revealed a significant increase in the lower (F=18.766) and upper-trunk flexion angles (F=17.273) at heel contact but no significant difference in lateral-trunk flexion (F=1.748) between the two PD groups. The PD+CC group also exhibited a significantly decreased range of hip flexion/extension (F=5.810) and knee flexion/extension angles (F=4.123) during the gait cycle, along with increased pelvis obliquity (F=5.175).

The other kinematic parameters at heel contact and the range of motion during the gait cycle were not significantly different between the PD+CC and PD-CC groups.

Relationships between trunk flexion angle and gait instability and spatiotemporal gait parameters

Table 4 presents the partial correlation results (adjusted for the NFOG-Q score) between the lower and upper trunk flexion angles and gait stability and spatiotemporal parameters in the PD group. Both the lower and upper trunk flexion angles were significantly negatively correlated with COM position$_{VT}$ and positively correlated with COM vel$_{ML}$ and step width. The lower trunk flexion angle did not significantly correlate with COM-BOS distance$_{AP}$ or MOS$_{AP}$ but had significant negative correlations with COM position$_{AP}$ and COM position$_{ML}$.

The upper trunk flexion angle was not significantly correlated with COM position$_{AP/ML}$ but was significantly negatively correlated with COM-BOS distance$_{AP}$, step length, COM vel$_{AP}$, and gait speed. Additionally, the upper trunk flexion angle was positively correlated with MOS$_{AP}$.

A participant with camptocormia and marked forward gait instability

One participant with camptocormia (patient 3 from Supplementary Video 1 [in the online-only Data Supplement]) presented a significantly lower MOS$_{AP}$ than did the HCs. This male participant in his 60s, classified as Hoehn and Yahr stage II with mild FOG (NFOG-Q score, 2), presented pronounced lower- and upper-trunk flexion angles. However, his step length and gait speed were similar to those of the HCs. Although his COM position$_{AP}$ was more anterior and his COM-BOS distance$_{AP}$ was smaller than those of the HCs, his COM vel$_{AP}$ was comparable to that of the HCs and notably greater than that of the PD+CC group. Consequently, his MOS$_{AP}$ was notably lower than that of the HCs. Unlike the PD+CC group, he did not exhibit a lower COM position$_{VT}$, a reduced sagittal range of motion in the lower extremity joints, increased frontal pelvic obliquity during gait, or a wider step width; his parameters closely resembled those of the HCs.

With respect to his personal background, he lived alone, experienced frequent forward falls (nearly daily) and reported no fear of falling. Despite intact cognitive function (MMSE score, 29), he appeared indifferent to fall prevention, stating, "It's okay if I fall." His gait parameters are presented in Supplementary Table 1 and Supplementary Table 2 (in the online-only Data Supplement), showing differences from the medians of both the PD+CC group and HCs.

DISCUSSION

This study represents the first comprehensive and objective

Table 4. Partial correlation, adjusted for freezing of gait severity, between lower or upper trunk flexion angle, gait stability parameters, and spatiotemporal gait parameters in the PD group (*n*=39)

	Correlation with lower trunk flexion angle at heel contact†	*p* value	Correlation with upper trunk flexion angle at heel contact*	*p* value
Gait stability parameters at heel contact				
COM position$_{VT}$ (% of height)	-0.690	<0.001*	-0.332	0.041*
COM position$_{AP}$ (% of BOS)	-0.479	0.002*	-0.131	0.431
COM position$_{ML}$ (% of BOS)	-0.464	0.003*	-0.190	0.254
COM-BOS distance$_{AP}$ (mm)	-0.160	0.336	-0.338	0.038*
COM-BOS distance$_{ML}$ (mm)	0.171	0.305	0.362	0.025*
COM vel$_{AP}$ (mm/sec)	-0.238	0.151	-0.428	0.007*
COM vel$_{ML}$ (mm/sec)	0.374	0.021*	0.446	0.005*
MOS$_{AP}$ (mm)	0.250	0.131	0.325	0.047*
MOS$_{ML}$ (mm)	-0.015	0.929	0.205	0.216
Spatiotemporal gait parameters				
Gait speed (m/s)	-0.201	0.226	-0.406	0.011*
Step length (m)	-0.200	0.228	-0.433	0.007*
Step width (m)	0.580	0.001*	0.474	0.003*
Cadence (steps/min)	-0.184	0.268	-0.127	0.448
Single support time (% of gait cycle)	-0.211	0.203	-0.182	0.273
Double support time (% of gait cycle)	0.205	0.217	0.233	0.160

*p<0.05; †partial correlation coefficients (adjusted for the New Freezing of Gait Questionnaire).
PD, Parkinson's disease; COM, center of mass; VT, vertical; AP, anterior-posterior; BOS, base of support; ML, mediolateral; vel, velocity; MOS, margin of stability.

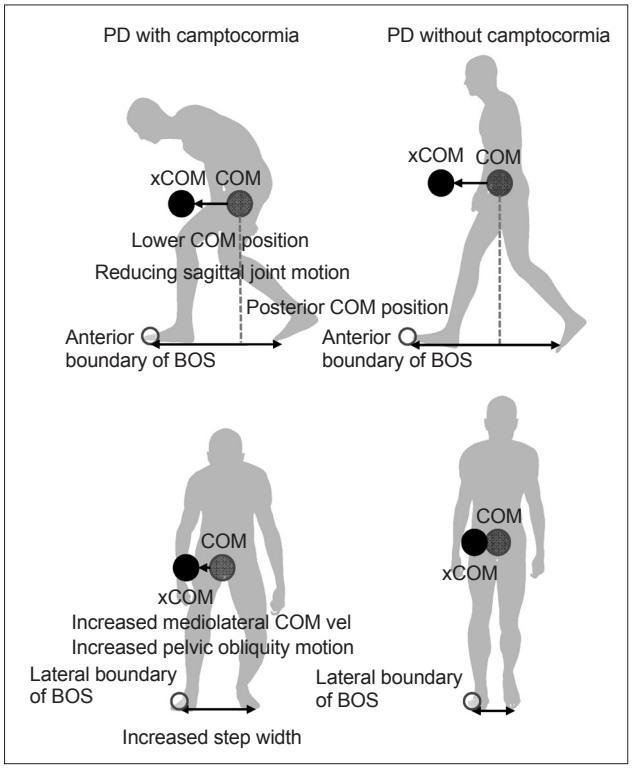

Figure 2. Gait characteristics of PD patients with and without camptocormia. The figure illustrates the COM, xCOM, and BOS. Compared with PD patients without camptocormia, PD patients with camptocormia exhibit a lower and more posterior COM position, reflecting vertical instability and a compensatory strategy for forward instability. They also demonstrated reduced sagittal joint motion, increased mediolateral COM vel, and a widened BOS. PD, Parkinson's disease; xCOM, extrapolated center of mass; COM, center of mass; BOS, base of support.

examination of the biomechanical mechanisms underlying gait instability and compensatory strategies in PD+CC patients. Figure 2 illustrates the key characteristics of PD+CC patients. We identified common control characteristics related to VT gait instability and compensatory strategies in PD+CC patients, excluding a unique patient with pronounced forward gait instability. First, we discuss the common characteristics of most PD+CC patients on the basis of group comparisons and correlation analysis with trunk flexion angles. Subsequently, we discuss the unique cases separately.

Common characteristics of gait instability and compensatory strategies in most PD+CC patients

Our findings quantitatively demonstrated, for the first time, that PD+CC patients exhibit a lower VT COM position during walking, bringing them closer to the ground. A fall is defined as an event resulting in a person unintentionally coming to rest on the ground or another lower level.[28] Additionally, we observed associations between increased trunk flexion angles and lower VT COM position. Therefore, these results suggest that PD+CC patients experience VT gait instability and an increased fall risk, with this risk intensifying as trunk flexion angles increase.

Camptocormia has also been considered to be associated with forward gait instability in PD patients,[11] leading us to expect that both the COM-BOS$_{_AP}$ and the MOS$_{_AP}$ in the present PD+CC patients would be lower than those in the PD-CC patients. However, the results showed that both measures were comparable between the two groups. The COM-BOS$_{_AP}$ was similarly lower in both the PD+CC and PD-CC patients than in the HCs, which is likely due to the narrowing of the anterior BOS caused by a decrease in step length, as suggested in an earlier investigation.[12] The effect of trunk flexion in PD+CC patients may have been offset by increased joint flexion angles in the trailing limbs. Although the increases in the participants' hip and knee flexion angles and the decrease in the ankle dorsiflexion angle in the trailing limb were not statistically significant, we speculate that the combination of these joint angle changes resulted in a significantly greater posterior position of the COM. These compensatory mechanisms may play crucial roles in maintaining forward stability despite pronounced trunk flexion.

The MOS$_{_AP}$ in our PD+CC group, similar to that in the PD-CC group, was greater than that in the HCs, indicating that PD+CC patients also tend to exhibit greater dynamic stability in the AP direction than HCs do. Kinematic analysis in this study revealed that in PD+CC participants, the range of motion in the sagittal plane of the hip and knee joints decreased, whereas the range of pelvic obliquity increased. The step width and COM lateral velocity in the PD+CC participants also increased. These results suggest that PD+CC participants adopt compensatory strategies not only to mitigate forward dynamic instability but also to manage the lateral instability caused by their forward flexion posture. By reducing joint motion in the sagittal plane and increasing lateral joint movement, they probably use inertial forces to reduce the flexion moment of the trunk, thereby stabilizing their gait.

The partial correlation analysis revealed significant negative correlations between trunk flexion angles (both upper and lower) and COM position$_{_VT}$, as well as positive correlations between trunk flexion angles and both COM vel$_{_ML}$ and step width. These results suggest that in PD patients, including those in the prediagnostic stage of camptocormia, greater trunk flexion angles increase VT gait instability and fall risk, prompting reliance on lateral COM shift as a compensatory strategy. Additionally, the analysis revealed different associations between the lower and upper trunk flexion angles. The negative correlation observed only between the lower trunk flexion angle and COM position$_{_AP, ML}$ indicates that greater lower trunk flexion angles are associated with a tendency to maintain the COM position in the trailing limb at heel contact. Conversely, the upper trunk flex-

ion angle was negatively correlated with only COM-BOS distance$_{AP}$, COM vel$_{AP}$, and step length and positively correlated with both MOS$_{AP}$ and COM-BOS$_{ML}$. These findings suggest that greater upper trunk flexion angles are associated with reduced step length and an increased risk of forward balance loss but also indicate a tendency to lower COM vel and enhance dynamic stability. Overall, these results imply that upper and lower trunk flexion angles may have common and distinct effects on gait instability and compensatory strategies in PD patients.

Unique case with prominent forward gait instability in PD+CC

One individual in the PD+CC group differed significantly from the other participants, exhibiting a marked reduction in MOS$_{AP}$ compared with the HCs. This finding indicates that the participant experienced pronounced forward gait instability, which we were able to objectively measure for the first time in the PD+CC condition. Unlike the other PD+CC participants, this unique individual did not adopt compensatory strategies such as reducing sagittal joint motion in the lower limbs or increasing pelvic movement in the frontal plane and step width. These results support the idea that compensatory strategies play a role in mitigating excessive forward gait instability in PD+CC patients. Notably, this participant showed preserved cognitive function and, despite frequent forward falls, had a low fear of falling and did not perceive the need to prevent forward falls. Additional research is needed to evaluate factors such as fear of falling and gait self-efficacy and to further investigate the determinants of the application of compensatory strategies for forward gait instability in PD+CC patients.

Study limitations and future perspectives

Our study has several limitations and suggests future research directions. First, the small sample size limits the representativeness of the PD+CC group and precludes subgroup analyses on the basis of fulcrum locations (upper and lower). A larger sample size may enable a more detailed investigation of differences between fulcrum locations and clarify gait instability characteristics in each group.

Additionally, qualitative factors such as fear of falling and psychosocial influences, which are closely tied to behavior, were underexplored. Gait measurements in an experimental environment aim to objectively analyze gait instability and compensatory strategies, ensuring robust results. In such controlled settings, subjects can more easily focus their attention on movement control. PD patients often maintain motor control when they are focused but control falters when attention is diverted.[29,30] Thus, in daily life, PD+CC patients may struggle to intentionally apply the compensatory strategies identified in this study, potentially worsening their gait instability.

Another limitation is that the measurements were conducted only during the ON phase of antiparkinsonian medication. In the OFF phase, delays in reactions necessary for controlling dynamic stability during gait may occur, potentially worsening gait instability.[31] Future research should incorporate wearable devices, such as multiple inertial measurement unit sensors, to perform long-term gait measurements in daily life,[32] including during the OFF phase. Examining MOS under limited attention could clarify differences between daily life and experimental conditions.

Conclusions

The findings of this study demonstrated that most PD+CC patients presented VT gait instability associated with a lower COM position and compensatory strategies involving increased lateral COM shift and a wider BOS, with these trends intensifying as trunk flexion angles increased. Additionally, certain cases were identified where patients with concurrent FOG, influenced by psychosocial factors impacting behavioral control, did not adopt compensatory strategies and exhibited significant forward gait instability, even in experimental settings. Understanding common abnormalities and compensatory characteristics of gait instability in PD+CC patients, along with individual variability influenced by factors such as psychosocial conditions, underscores the importance of targeted, multidisciplinary rehabilitation interventions.

Supplementary Video Legends

Video 1. This video shows the gait patterns of 3 patients with Parkinson's disease (PD). The top section shows a lateral view of gait, the bottom left shows a posterior view, and the bottom right presents the trajectory of the center of mass (COM) movement. Patient 1 was a PD patient without camptocormia. Patient 2, with camptocormia, presented decreased sagittal plane joint motion, increased step width, and greater lateral COM displacement. Patient 3 also has camptocormia and exhibits marked forward gait instability, without decreased sagittal plane joint motion or increased frontal plane joint movement.

Supplementary Materials

The online-only Data Supplement is available with this article at https://doi.org/10.14802/jmd.24226.

Conflicts of Interest

The authors have no financial conflicts of interest.

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