

Gait dynamics in Pisa syndrome and Camptocormia: The role of stride length and hip kinematics

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Abstract

This is an observational cross-sectional study evaluating gait dynamics in patients with Parkinson's Disease (PD) and severe postural deformities, PD without axial deviations and healthy subjects. Ten PS individuals with Pisa syndrome (PS) and nine subjects with Camptocormia (CC) performed 3-D Gait Analysis and were evaluated with walking and balance scales. Correlations with clinical and functional scales were investigated. Spatio-temporal and kinematic data were compared to ten PD subjects without postural deformities (PP) and ten healthy matched individuals (CG). Data obtained showed decreased walking velocity, stride and step length in PP, PS and CC groups compared to controls. The correlation analysis showed that stride and step length were associated with reduced functional abilities and disease severity in PS and CC groups. Kinematic data revealed marked reduction in range of movements (ROMs) at all lower-extremity joints in PS group. While, in CC group the main differences were pronounced in hip and knee joints. PS and CC groups presented a more pronounced reduction in hip articular excursion compared to PP subjects, revealing an increased hip flexion pattern during gait cycle. Moreover, the increased hip and knee flexion pattern adversely affected functional performance during walking tests. Results obtained provide evidence that step length, along with stride length, can be proposed as simple and clear indicators of disease severity and reduced functional abilities. The reduction of ROMs at hip joint represented an important mechanism contributing to decreased walking velocity, balance impairment and reduced gait performance in PD patients with postural deformities.

Keywords: Functional disability; Gait analysis; Hip kinematics; Parkinson's disease; Postural deformities.

1. Introduction

Patients with Parkinson's Disease (PD) often present with abnormal posture, with a subset of patients showing severe postural deformities, including Pisa syndrome and camptocormia [1]. Pisa syndrome refers to a marked lateral trunk flexion of more than 10°, which is typically mobile and it resolves by passive mobilization or lying down [2]. It presents a prevalence of 8,8%, affecting predominantly patients with significantly longer disease duration, more severe disease and worse quality of life [3]. Camptocormia is used to describe a marked flexion of the thoraco-lumbar spine of at least 40–45°, appearing in standing position, increasing while walking and resolving in supine position [2]. At first, these abnormalities were described as truncal dystonia, analogous to that occurring in non-parkinsonian patient as a side-effect of neuroleptics' treatment [2,4]. Nowadays, the pathophysiology is very probably multifactorial, even if the bulk of the data supports central, rather than peripheral, hypotheses. Peripheral hypotheses take into account unspecific myopathic changes of paraspinal

muscles: a focal myopathy has been demonstrated by EMG, CT and MRI scans and biopsy [5]. The central hypotheses, supported by both animal studies and clinical data, include an asymmetric functioning of basal ganglia output, leading directly to asymmetric regulation of postural muscle tone, inducing a misperception of body orientation and the adoption of an asymmetric posture [4]. Anyway, as far as the underlying pathophysiology of these postural deformities is still largely unknown and debated, their management remains difficult. Marked alterations of gait are common in advanced PD, while perturbations of postural reflexes may be responsible for postural instability. Gait impairments, together with turning and balance disturbances, are the most important determinants of falls among people with PD. The negative impact of gait disorders on QoL is related to immobility and increased risk of falling [6]. Postural instability represents a disabling feature associated with difficulties in postural transfers, progressive loss of independence, immobility and high risk of sudden falls [7]. In this panorama, it is widely recognized that postural deformities may cause and worsen gait impairments, postural imbalance and functional disability [8], independently of other motor symptoms [1]. Therefore, the management of such abnormalities have recently attracted attention, even if it still remains a challenge. Recently, suggestions for clinical practice have been put forward, addressing the importance to consider both pharmacological and nonpharmacological interventions as integral parts of a comprehensive rehabilitative plan [4]. Current literature present a plenty of study aiming at objectively quantifying gait pattern in subjects with PD [9–11]. To our knowledge, only one study evaluated gait abnormalities in PD patients with Pisa syndrome, focusing almost on static postural control and balance [8]. Therefore, the characterization of gait abnormalities in PD patients with postural deviations could be relevant in order to describe distinctive walking patterns and direct specific rehabilitative strategies. The purpose of our study is to quantitative assess gait dynamics in PD patients affected by postural deformities, Pisa syndrome and camptocormia, compared to a group of PD subjects without axial deviations and to a group of age-matched healthy subjects. Moreover, we try to reveal if disease severity is related to spatio-temporal and kinematic data in PS and CC groups. In the end, we evaluate if functional abilities during walking tests are related to spatio-temporal and kinematic data in the two groups of PD individuals with postural abnormalities.

2. Patients & methods

Twenty-nine subjects diagnosed with Parkinson's disease from the Unit of Neurology (University Hospital of Pisa, Italy) were recruited. Patients presented the following postural deformities: Pisa syndrome, defined as at least 10° of lateral trunk flexion, or camptocormia, defined as an anterior trunk flexion of at least 40°, reducible by passive mobilization or supine positioning. All participants (mean age 71 ± 6,4 years) were divided into three groups: Group PS, including 10 subjects with Pisa syndrome; Group CC, including 9 subjects with camptocormia; Group PP, including 10 subjects diagnosed with Parkinson's disease without postural deformities. Moreover, 10 healthy subjects (CG) were individualized to participate as a control group. Inclusion criteria were:

- diagnosis of Parkinson's disease, according to the United Kingdom Parkinson's Disease Society Brain Bank criteria [12]
- postural deformities (Pisa syndrome or camptocormia);
- walking ability for a short distance (10 m) without use of device;
- age over 18 years.

Exclusion criteria were:

- severe dyskinesia or "on-off" fluctuations;
- need for assistive devices to rise from a chair or bed;
- severe cardiopulmonary disease;
- severe lower limbs arthritis;
- severe motor disability due to other neurological or orthopedic diseases;
- other vertebral diseases (scoliosis, spondyloarthritis) evaluated with X-ray;
- important cognitive deficit (MMSE < 24).

Anthropometric data of parkinsonian patients are summarized in Table 2. All subjects underwent functional evaluations and gait analysis, after giving their informed consent to participate in the study. The disease duration (DD) and the time of postural deviation's onset (PO) were determined for all parkinsonian patients with axial deviations, (Table 2). Moreover, all PD patients were classified with the Hoehn and Yahr disease rating scale (HY) [13] and examined according to the motor section of the Unified Parkinson's Disease Rating Scale (UPDRSIII) [14], (Table 2). Patients also performed the following functional evaluations: Six-minute walk test (6MWT), 10-m Walking Test (10mWT), Timed Up and Go Test (TUG) and Berg Balance Scale (BBS) [15–17].

The study was carried out according to the Declaration of Helsinki and was approved by the Local Ethics Committee.

2.1. Gait analysis

All subjects underwent 3-D Gait Analysis performed at Motion Analysis Laboratory of Neurorehabilitation Unit of the University Hospital of Pisa, using the ELITE System (BTS Bioengineering, Milan, Italy). Before the recordings, general and anthropometric data were collected. The gait tests were performed during the on-phase of the medication cycle (1–2 h after intake of their morning dose). Then, spatio-temporal and kinematics data were acquired through six photogrammetric system infrared cameras acquiring at a sampling frequency of 100 Hz 18 reflective markers were placed on definite anatomical landmarks for kinematic acquisitions, according to Davis protocol [18]. Acquisitions were made in standing position and during barefoot walking at self-selected speed, recording at least three trials for each limb.

2.2. Data analysis

Spatio-temporal and kinematic parameters analysis for the hip, knee, and ankle joints were performed by using BTS Elite Clinic software. The following spatio-temporal parameters were analyzed: walking velocity, cadence, stride time, stride length, step time, step length, step width and the percentages of stance and swing phases compared to the total duration of gait cycle. As regard the kinematic pattern, a body model accounting for each body segments was used to calculate articular angular excursions along the gait cycle. In the sagittal plane, the value of the maximum and the minimum angle reached in each joint (hip, knee, ankle) was calculated. Furthermore, the dynamic range of motion (ROM) of each joint was determined. Further analysis were performed using MATLAB.

2.3. Statistical analysis

Variables across groups were tested for normality with Shapiro-Wilk test. The four groups were matched for anthropometric variables such as age, sex, height, weight and BMI as confirmed with Kruskal-Wallis H test for non-parametric samples (Table 1). Data were analyzed nonparametrically with the Mann-Whitney U-test for independent group comparisons. Further, Spearman's coefficient was used to analyze the correlations between disease severity and spatio-temporal data (step and stride length) and kinematic data (knee ROM, hip ROM, hip flexion in stance phase of gait cycle) in PS and CC groups. Moreover, the correlations between functional tests (6MWT, 10 MWT, TUG, BBS) and spatio-temporal data (step and stride length) and kinematic data (hip ROM, knee ROM, hip flexion in stance phase of gait cycle) were analyzed. Bonferroni correction was performed.

Results are described in relation to the significance of $p < 0,05$ and 95% confidence interval. All statistical procedures were performed with SPSS/PC (version 20.0) software program.

Table 1
Groups' characteristics.

	PS	CC	PP	CG	<i>p</i>
Age (years)	67,7 ± 4,3	72,2 ± 5,2	73,2 ± 8,1	68,8 ± 10	0,24
Sex (F/M)	5F/5M	2F/7M	4F/6M	7F/3M	0,21
Height (m)	1,67 ± 1,07	1,73 ± 0,09	1,66 ± 0,08	1,65 ± 0,08	0,29
Weight (Kg)	72,6 ± 10,8	77,6 ± 15,2	61,4 ± 11,8	66,4 ± 8,8	0,06
BMI	25,8 ± 3,1	25,6 ± 3,8	22,1 ± 3,1	24,3 ± 4,3	0,07

NOTE. Values are mean ± standard deviation (SD). Abbreviations: BMI, Body Mass Index; PS, Pysa syndrome; CC camptocormia; PP, Parkinson disease without postural deformities; CG, healthy subjects. *Significant at $p < 0.05$.

Table 2
Anthropometric and clinical data for PD patients.

Patient	Syndrome	Age	Sex	Height	Weight	BMI	DD years	PO months	Tilt side	HY stage	UPDRSIII
1	PS	67	F	1,66	62	22,49	15	12	R	3	20
2	PS	64	F	1,5	50	22,22	9	84	R	4	51
3	PS	70	M	1,67	82	29,4	4	3	R	2	12
4	PS	75	M	1,65	80	29,38	3	6	R	4	44
5	PS	63	F	1,67	79	28,32	2	1	L	2,5	27
6	PS	72	F	1,65	70	25,71	5	6	R	3	36
7	PS	72	M	1,75	73	23,83	10	12	R	3	40
8	PS	62	M	1,72	70	23,66	8	48	L	3	34
9	PS	67	M	1,74	72	23,78	1	12	L	1,5	8
10	PS	65	F	1,71	88	30,09	7	12	R	2	29
11	CC	65	M	1,8	77	23,76	3	12	anterior	2	28
12	CC	80	M	1,74	65	21,47	7	12	anterior	4	38
13	CC	72	M	1,7	74	24,31	4	12	anterior	2	49
14	CC	75	M	1,91	95	26,04	3	24	anterior	2	18
15	CC	63	F	1,63	69	25,97	5	12	anterior	3	15
16	CC	74	M	1,76	90	29,05	11	24	anterior	3	28
17	CC	76	F	1,6	60	23,43	7	6	anterior	2	29
18	CC	73	M	1,75	104	33,95	2	24	anterior	2	14
19	CC	72	M	1,68	65	23,03	2	12	anterior	2	19
20	PP	62	M	1,72	81	27,37	3	-	-	1	12
21	PP	74	M	1,68	75	26,57	5	-	-	2	15
22	PP	59	M	1,72	68	22,98	6	-	-	1	21
23	PP	80	F	1,52	42	18,17	10	-	-	3	27
24	PP	79	F	1,55	57	23,72	7	-	-	3	26
25	PP	70	F	1,6	51	19,92	6	-	-	4	46
26	PP	85	M	1,65	50	18,36	12	-	-	3	50
27	PP	70	M	1,75	64	20,89	10	-	-	4	21
28	PP	74	M	1,78	65	20,51	5	-	-	2	16
29	PP	79	F	1,64	61	22,67	6	-	-	3	27

Abbreviations: BMI, Body Mass Index; DD, Disease duration; PO, time of postural deviation onset; HY stage, Hoehn & Yahr stage; UPDRS III, Unified Parkinson's Disease Scale-motor part; PS, Pysa syndrome; CC camptocomia; PP, Parkinson disease without postural deformities; F, female; M, male; R, right; L, left.

3. Results

3.1. Spatio-temporal data

All Parkinsonian patients, belonging to either PS, CC or PP groups, showed decreased walking velocity, stride and step length compared to controls, whereas cadence did not differ significantly. Moreover, the double-limb support and the stance phase of gait were prolonged.

Compared to PP group, PS and CC groups showed no significant differences, (Table 3).

3.2. Kinematic data

PP group presented reduced ROMs at hip and knee joints compared to controls (Fig. 1). They presented a reduced hip flexion during time of initial contact and in the swing phase, increased knee flexion in the stance phase and reduced knee flexion in the swing phase (Fig. 2).

The ROMs were reduced at all lower-extremity joints in the PS group compared to the CG group (Fig. 1). A reduced hip extension

during the mid-stance and a reduced knee flexion during the swing phase were evident (Fig. 2). Moreover, PS subjects walked with an increased

knee flexion during the single support phase, even if not significantly different. No differences were found in ankle kinematics between

the two groups. The PS group showed a significant reduction in hip ROMs and walked with an increased hip flexion during the stance

phase and during the swing phase compared to the PP group (Figs. 1 and 2). In the CC group the main differences were pronounced in the proximal joints, as far as the ROMs were reduced at hip and knee

compared to the CG group (Fig. 1). CC subjects walked with increased hip flexion during the stance phase and increased knee flexion during the single support phase compared to controls. No difference were found

in ankle kinematics between the two groups. The CC group showed a significant reduction in hip ROMs and presented an increased hip flexion pattern during the different phases of gait cycle and an increased

knee flexion during the single support phase and in the swing phase compared to the PP group (Figs. 1 and 2).

3.3.

Table 3
Spatio-temporal data.

Parameters	PS	PP	p	CG	p
Walking velocity (m/s)	0,62 ± 0,23	0,7 ± 0,24	0,5	0,95 ± 0,12	0,001**
R Stride Length (m)	0,75 ± 0,22	0,83 ± 0,23	0,54	1,09 ± 0,09	0,001**
L Stride Length (m)	0,75 ± 0,23	0,82 ± 0,24	0,73	1,10 ± 0,10	0,001**
R Stance duration (% GC)	61,62 ± 4,66	60,17 ± 4,92	0,5	55,75 ± 2,33	0,001**
L Stance duration (% GC)	61,97 ± 5,71	60,76 ± 3,88	0,7	55,42 ± 2,25	0,002**
Cadence (step/min)	98,13 ± 23,2	100,37 ± 9,44	0,6	103,9 ± 7,73	0,4
R Double support (% GC)	11,84 ± 4,58	10,69 ± 4,61	0,65	5,92 ± 1,82	0,003**
L Double support (% GC)	12,18 ± 6,66	9,82 ± 4,40	0,4	5,58 ± 2,18	0,001**
Step width (m)	0,13 ± 0,04	0,13 ± 0,02	0,4	0,09 ± 0,03	0,07

Parameters	CC	PP	p	CG	p
Walking velocity (m/s)	0,76 ± 0,11	0,7 ± 0,24	0,5	0,95 ± 0,12	0,004**
R Stride Length (m)	0,88 ± 0,14	0,83 ± 0,23	0,4	1,09 ± 0,09	0,004**
L Stride Length (m)	0,89 ± 0,14	0,82 ± 0,24	0,37	1,10 ± 0,10	0,003**
R Stance duration (% GC)	58,70 ± 3,29	60,17 ± 4,92	0,46	55,75 ± 2,33	0,1
L Stance duration (% GC)	58,94 ± 3,87	60,76 ± 3,88	0,32	55,42 ± 2,25	0,03*
Cadence (step/min)	104,06 ± 10,57	100,37 ± 9,44	0,1	103,9 ± 7,73	0,5
R Double support (% GC)	9,61 ± 3,45	10,69 ± 4,61	0,74	5,92 ± 1,82	0,01*
L Double support (% GC)	8,43 ± 2,83	9,82 ± 4,40	0,62	5,58 ± 2,18	0,02*
Step width (m)	0,12 ± 0,04	0,13 ± 0,02	0,56	0,09 ± 0,03	0,1

NOTE. Values are mean ± standard deviation (SD). Abbreviations: PS, Pisa syndrome; CC camptocormia; PP, Parkinson disease without postural deformities; CG, healthy subjects; R, right; L, left; GC, gait cycle. *Significant at $p < 0.05$; **Significant at $p < 0.01$.

Correlation analysis

In the PS group, the correlation analysis between clinical scales and spatio-temporal data revealed that higher HY stage was related to decreased step and stride length. Moreover, step and stride length negatively influenced 10MWT, TUG, 6 MWT and BBS. The correlation analysis between clinical scales and kinematic data revealed that UPDRSIII negatively affected knee and hip ROMs and worsened the hip flexion pattern during stance time of gait cycle. While, the hip flexion pattern during stance phase of gait cycle negatively influenced TUG and 6 MWT. In the CC group, the correlation analysis between clinical scales and spatio-temporal data showed that disease severity (HY) was related to reduced step and stride length. Furthermore, step and stride length negatively influenced BBS. The correlation analysis between functional scales and kinematic data revealed that disease severity, assessed by HY stage, negatively influenced hip ROM and worsened the hip flexion pattern during stance time of gait cycle. Furthermore, the reduced hip ROM and the hip flexion pattern during stance phase of gait cycle adversely affected 6 MWT, 10 MWT ad BBS.

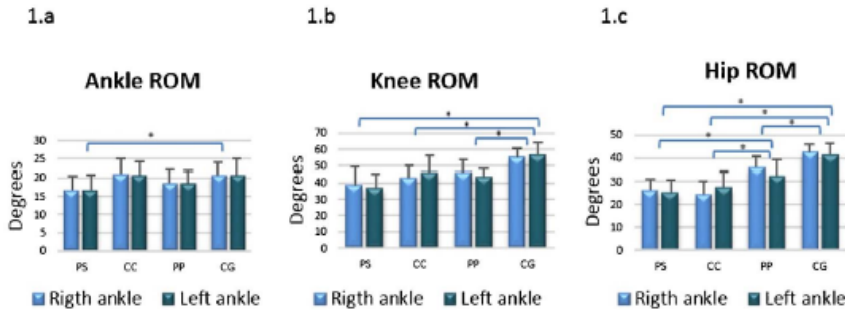


Fig. 1. Right and left ankle (1.a), knee (1.b) and hip (1.c) ROMs over the gait cycle in four groups.
NOTE: Values are mean ± SD. Abbreviation: PS, Pisa syndrome group; CC, Camptocormia group; PP, Parkinson's disease without postural deformities; CG, control group. *Significant at $p < 0.05$.

4. Discussion

Gait disturbances are frequently observed in PD patients and are usually consistent with functional disabilities, loss of independence and increased risk of falling [6]. Postural deformities may worsen gait impairments, postural instability and overall individual functional capacities [8]. Our data provide detailed informations regarding gait dynamics in PD patients with Pisa syndrome and camptocormia. Furthermore, we focus on the relationship between these parameters and clinical and functional scales.

The key spatiotemporal findings in P, CC and PP groups revealed that gait was characterized by short steps and lower walking velocity compared to healthy subjects. These results were consistent with most previous studies on time-distance parameters in Parkinsonian patients [19,10,11]. Moreover, similar to previous reports, we also did not find any difference in cadence between PD subjects and controls [20]. Cadence decrease was evidenced in particular on initial stages in PD subjects as a contributing factor to velocity reduction [21]. In the present study, the three PD groups presented moderate to severe stages of the disease. Therefore, the decrease of gait velocity may be related to stride length shortening, since these two parameters are often associated [19]. We also documented a tendency to a longer duration of the double-support phase of gait cycle. Usually, people increase double limb support time to compensate for fear of falling or postural instability, both of which are common in advanced PD [6] and are aggravated by axial deformities [8]. Then, extending the duration of double limb support times increases the time available for restabilization, thus minimizing the demands on postural control system [19].

Contrary to our findings, Geroin et al. [8] did not find similar spatiotemporal modifications in PD patients with Pisa syndrome. This is quite controversial, even if subjects recruited presented less severe impairment compared to ours, as evidenced by functional scales.

Very interestingly, as regard functional tests in PS and CC groups, the stride and step length reduction were associated with increased disease severity, assessed by HY stage. These two parameters were also both related to reduced performance during walking tests in PS group, such as 10 MWT, TUG, 6 MWT, and with impaired balance during functional tasks in PS and CC groups, as measured by BBS. Similarly, De Melo Roiz et al. [11] revealed the negative relation between stride length and motor-functional tests, in particular UPDRS, TUG and BBS in Parkinsonian subjects. As currently evidenced, PD severity [22] together with BBS [23] are both recognized as significant risk factors for future falls in PD subjects. Furthermore, our patients presented typical biomechanical features evidenced in PD fallers: shorter strides, slower walking and increased double-support time [24]. Therefore, we confirm that stride and step length may represent high-reproducible measures of disease severity and reduced functional abilities even in PD patients with postural deformities. The main finding of our study is related to kinematic evaluations: ROM values were reduced for all major joints of the lower extremity in the PS group, while in the CC and PP groups the differences were more pronounced in hip and knee joints compared to controls. Moreover, data showed that both PS and CC groups presented a significant reduction in hip ROMs even compared to PP subjects. These findings have been previously described in Parkinsonian patients [10,11], suggesting a possible contribution to shortened stride length [25]. In fact, stride length regulation and angular excursions seem to be two faces of a single coin: an internal motor misregulation of basal ganglia in the setting of stride length may cause a mismatch in amplitude of movement occurring across all joints [20]. Furthermore, gait pattern in the PS and CC groups was characterized by increased hip flexion during the stance phase compared to healthy subjects. Moreover, an increased knee flexion during the single support phase was also appreciable in both groups. Interestingly, PS and CC subjects walked with increased hip flexion over almost gait cycle even compared to PP patients. CC patients presented also increased knee flexion during the single support phase and in the swing phase compared to PP patients. Previously, Morris et al. [20] pointed out that hip extension range in stance phase was the most affected in a PD patient. On this line, Lewis et al. [21] defined that similar features represented the most frequent biomechanical findings in Parkinsonian individuals. Interestingly, Albani et al. [26] discussed dynamic characteristics of Parkinsonian gait related to disease severity: patients with greater disease severity, as evidenced by HY stage, presented a prevalently proximal involvement, while alterations in ankle kinematics were evident in patients at earlier stages of the disease. Anyway, we did not find any kinematic modifications at the ankle joint during maximal plantarflexion and dorsiflexion times. This is quite singular, as far as different studies [19,20,9] evidenced a reduced plantarflexion associated with underscaling of power generation at push-off in PD patients [27].

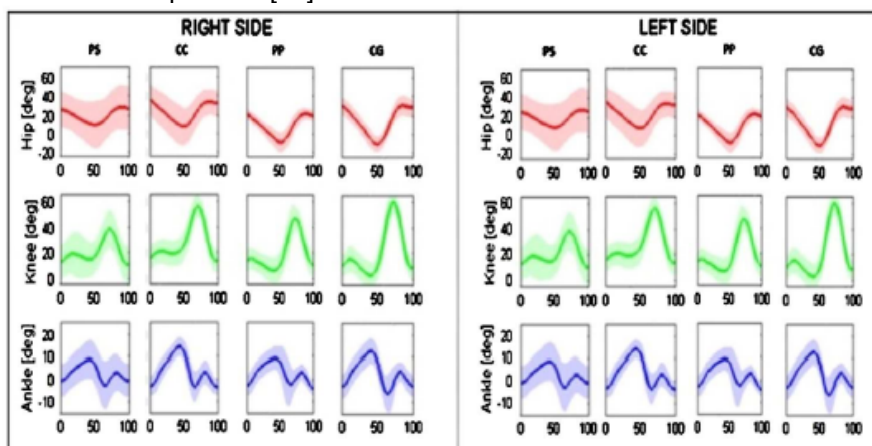


Fig. 2. Right and left ankle, knee, and hip kinematics in the four groups. Abbreviation: PS, Pisa syndrome group; CC, Camptocormia group; PP, Parkinson's disease without postural deformities; CG, control group; deg, degrees.

A reason for such a difference might be that patients in either PS group or CC group presented moderate-severe disease progression, then supporting previous considerations of predominantly proximal joint impairment. Interestingly, Sofuwa et al. [9] pointed out that the inadequate hip extension in the stance phase of Parkinsonian gait represented one of the most important factor limiting hip power generation. In fact, the normal compensatory dynamic strategies, such as an increase in hip flexion power in the late stance phase, seems to be not fully exploited in PD patients [9] or accompanied by an increased stiffness at the hip [28]. As known, the increased axial stiffness of PD patients, especially at the hip [29], may influence the different response between ankle and hip joints in static and dynamic conditions. Therefore we could hypothesize that PD patients with postural deformities present typical misregulations, worsening hip kinematics in dynamic situations. As regard correlation analysis in the PS group, we evidenced that the degree of motor impairment, assessed by UPDRSIII, was associated with reduced knee and hip ROMs and worsened the hip flexion pattern during stance time of gait cycle. While, in the CC group, disease severity,

assessed by HY stage, negatively influenced hip ROM, and worsened hip flexion pattern during stance time of gait cycle. In line with our findings, a strong negative linear relationship seems to exist between disease severity and movement amplitude across multiple joints, in particular the hip [25]. Then, our data evidence that impaired hip kinematics seems to be one of the main characteristic of PD gait with axial deviations, able to influence gait abilities and performance during balance tasks. These results may have important implications even in the treatment approach of such patients. Rehabilitation trials considered visual cues and treadmill walking programs to improve time-distance and kinematic parameters: normal stride length can be elicited in PD subjects using attentional strategies and visual cues [30]. In this regard, we can suggest that similar attentional strategies may be proposed in subjects with PD and axial deformities, as a tool to normalize stride length and gait pattern [30]. Furthermore, training interventions improving hip extension strength as well as techniques focusing on hip muscle stiffness might be valuable rehabilitation strategies, correcting kinematic gait abnormalities. A limitation of the current study is represented by the small sample size. Using a larger sample size may strengthen the statistics of the study. Another limitation to consider is that recordings were made in a setting of short-medium dimensions. Future perspectives should be aimed at the evaluation of joint moments of forces and joint power generation and absorptions (kinetics) during walking.

References

- [1] R. Ashour, J. Jankovic, *Joint and skeletal deformities in Parkinson's disease, multiple system atrophy, and progressive supranuclear palsy*, *Mov. Disord.* 21 (2006) 1856–1863.
- [2] K.M. Doherty, B.P. Van de Warrenburg, M.C. Peralta, L. Silveira-Moriyama, J.P. Azulay, O.S. Gershanik, B.R. Bloem, *Postural deformities in Parkinson's disease*, *Lancet Neurol.* 10 (6) (2011) 538–549.
- [3] M. Tinazzi, A. Fasano, C. Geroin, F. Morgante, R. Ceravolo, S. Rossi, A. Thomas, G. Fabbrini, A. Bentivoglio, F. Tamma, G. Cossu, N. Modugno, M. Zappia, M.A. Volontè, C. Dallochio, G. Abbruzzese, C. Pacchetti, R. Marconi, G. Defazio, M. Canesi, A. Cannas, A. Pisani, R. Mirandola, P. Barone, C. Vitale, *Pisa syndrome in Parkinson disease: an observational multicenter Italian study*, *Neurology* 85 (2015) 1–12.
- [4] M. Tinazzi, C. Geroin, M. Gandolfi, N. Smania, S. Tamburin, F. Morgante, A. Fasano, *Pisa syndrome in Parkinson's disease: an integrated approach from pathophysiology to management*, *Mov. Disorder* 31 (12) (2016) 1785–1795.
- [5] T. Lenoir, N. Guedj, P. Boulu, P. Guigui, M. Benoist, *Camptocormia the bent spine syndrome, an update*, *Eur. Spine J.* 19 (2010) 1229–1237.
- [6] N. Smania, A. Picelli, C. Geroin, P. Ianes, E. La Marchina, A. Zenorini, M. Gandolfi, *Diagnosis and Treatment of Parkinson's Disease*, (2011).
- [7] Y.A. Grimbergen, M. Munneke, B.R. Bloem, *Falls in Parkinson's disease*, *Curr. Opin. Neurol.* 17 (4) (2004) 405–415.
- [8] C. Geroin, N. Smania, F. Schena, E. Dimitrova, E. Verzini, F. Bombieri, F. Nardello, M. Tinazzi, M. Gandolfi, *Does the Pisa syndrome affect postural control, balance, and gait in patients with Parkinson's disease? An observational cross-sectional study*, *Parkinsonism Relat. Disord.* 21 (2015) 736–741.
- [9] O. Sofuwa, A. Nieuwboer, K. Desloovere, A.M. Willems, F. Chavret, I. Jonkers, *Quantitative gait analysis in Parkinson's disease: comparison with a healthy control group*, *Arch. Phys. Med. Rehabil.* 86 (2005) 1007–1013.
- [10] M. Švehlík, E.B. Zwick, G. Steinwender, W.E. Linhart, P. Schwingenschuh, P. Katschnig, E. Ott, C. Enzinger, *Gait analysis in patients with Parkinson's disease off dopaminergic therapy*, *Arch. Phys. Med. Rehabil.* 90 (2009) 1880–1886.
- [11] R. De Melo Roiz, E.W. Azevedo Cacho, M. Macedo Pazinato, J.G. Reis, A. Cliquet Jr., E.M.A. Barasnevicius-Quagliato, *Gait analysis comparing Parkinson's disease with healthy elderly subjects*, *Arq. Neuropsiquiatr.* 68 (1) (2010) 81–86.
- [12] D.J. Gelb, E. Oliver, S. Gilman, *Diagnostic criteria for Parkinson disease*, *Arch. Neurol.* 56 (1999) 33–39.
- [13] M.M. Hoehn, M.D. Yahr, *Parkinsonism onset, progression, and mortality*, *Neurology* 5 (1967) 427–442.
- [14] S. Fahn, R. Elton, *Unified Parkinson's Disease Rating Scale*, *MacMillan Healthcare Information*, Florham Park, NJ, 1987.
- [15] T. Steffen, M. Seney, *Test-retest reliability and minimal detectable change on balance and ambulation tests, the 36-item short-form health survey, and the unified Parkinson disease rating scale in people with parkinsonism*, *Phys. Ther.* 88 (6) (2008) 733–746.
- [16] K.J. Brusse, S. Zimdars, K.R. Zalewski, T.M. Steffen, *Testing functional performance in people with Parkinson disease*, *Phys. Ther.* 85 (2) (2005) 134–141.
- [17] J. MacGregor, *The evaluation of patient performance using long-term ambulatory monitoring technique in the domiciliary environment*, *Physiotherapy* 67 (2) (1981) 30–33.
- [18] R.B. Davis, S. Ounpuu, D. Tyburski, J.R. Gage, *A gait analysis data collection and reduction technique*, *Hum. Mov. Sci.* 10 (5) (1991) 575–587.
- [19] M.E. Morris, J. McGinley, F. Huxham, J. Collier, R. Robert Iansek, *Constraints on the kinetic, kinematic and spatiotemporal parameters of gait in Parkinson's disease*, *Hum. Movement Sci.* 18 (1999) 461–483.

- [20] G.N. Lewis, W.D. Byblow, S.E. Walt, *Stride length regulation in Parkinson's disease: the use of extrinsic, visual cues*, *Brain* 123 (2000) 2077–2090.
- [21] I. Carpinella, P. Crenna, E. Calabrese, et al., *Locomotor function in the early stage of Parkinson's disease*, *Trans. Neural Syst. Rehabil. Eng.* 15 (2007) 543–551.
- [22] B.R. Bloem, Y.A. Grimbergen, M. Cramer, M. Willemsen, A.H. Zwiderman, *Prospective assessment of falls in Parkinson's disease*, *J. Neurol.* 248 (11) (2001) 950–958.
- [23] R.P. Duncan, A.L. Leddy, J.T. Cavanaugh, L.E. Dibble, T.D. Ellis, M.P. Ford, K.B. Foreman, G.M. Earhart, *Accuracy of fall prediction in Parkinson disease: six-month and 12-month prospective analyses*, *Parkinson's Dis.* (2012) 1–7.
- [24] M.H. Cole, P.A. Silburn, J.M. Wood, C.J. Worringham, G.K. Kerr, *Falls in Parkinson's disease: kinematic evidence for impaired head and trunk control*, *Mov. Disord.* 25 (14) (2010) 2369–2378.
- [25] M. Morris, R. Iansek, J. McGinley, T. Matyas, F. Huxham, *Three-dimensional gait biomechanics in Parkinson's disease: evidence for a centrally mediated amplitude regulation disorder*, *Mov. Disord.* 20 (1) (2005) 40–50.
- [26] G. Albani, V. Cimolin, A. Fasani, C. Trotti, M. Galli, A. Mauro, *Masters and servants in parkinsonian gait: a three-dimensional analysis of biomechanical changes sensitive to disease progression*, *Funct. Neurol.* 29 (2) (2014) 99–105.
- [27] V. Dietz, W. Zijlstra, T. Prokop, W. Berger, *Leg muscle activation during gait in Parkinson's disease: adaptation and interlimb coordination*, *Electroencephalogr. Clin. Neurophysiol.* 97 (6) (1995) 408–415.
- [28] T.A. Boonstra, A.C. Schouten, J.P.P. Van Vugt, B.R. Bloem, H. Van der Kooij, *Parkinson's disease patients compensate for balance control asymmetry*, *J. Neurophysiol.* 112 (2014) 3227–3239.
- [29] M.G. Carpenter, J.H.J. Allum, F. Honegger, A.L. Adkin, B.R. Bloem, *Postural abnormalities to multidirectional stance perturbations in Parkinson's disease*, *J. Neurol. Neurosurg. Psychiatry* 75 (2004) 1245–1254.
- [30] M.E. Morris, R. Iansek, T.A. Matya, J.J. Summers, *Stride length regulation in Parkinson's disease Normalization strategies and underlying mechanisms*, *Brain* 119 (1996) 551–568.