

Kinematic signatures underlying freezing of gait: a possible pathophysiological bridge between Parkinson's Disease phenotypical spectrum and Progressive Supranuclear Palsy

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Title page

Title

Kinematic signatures underlying freezing of gait: a possible pathophysiological bridge between Parkinson's Disease phenotypical spectrum and Progressive Supranuclear Palsy.

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Abstract

Introduction: Gait analysis has emerged as a valuable tool for the objective assessment of motor dysfunction in neurodegenerative diseases. While spatiotemporal gait parameters are well established as markers of disease and progression, the combination of kinematic assessment may aid to disentangle subtle differences in motor control strategies, especially when assessing episodic symptoms, like freezing of gait (FOG). Although FOG represents one of the most disabling symptoms in Parkinson Disease (PD) and Progressive Supranuclear Palsy (PSP), the extent to which the presence of FOG alters gait patterns, even beyond FOG episodes, remains overlooked.

Objective: To quantitatively compare gait patterns among PD patients with (PD+FOG) and without FOG (PD-FOG) and PSP patients, combining spatiotemporal and kinematic analyses with the aim of offering insight about both the nature of such phenomenon and the issue of the phenotypical heterogeneity in parkinsonism.

Methods: Seventy-six patients with parkinsonism (32 PD-FOG, 20 PD+FOG, and 24 PSP, all comparable on both gender and age) underwent optoelectronic gait analysis during a single-task walking and a dual cognitive task. Spatiotemporal and kinematic parameters were extracted using a standardized motion capture protocol. Group differences were investigated using ANCOVA, controlling for disease duration.

Results: Almost all spatiotemporal parameters significantly differentiated PSP from both PD groups, whereas no differences emerged between PD+FOG vs PD-FOG. Kinematic analysis revealed significant alterations at the pelvic, knee and ankle levels, distinguishing again PSP from PD, but also highlighting several differences between PD+FOG and PD-FOG. In particular, kinematic variables stood along a spectrum spanning across PD-FOG, PD+FOG and PSP, where pelvic tilt, hip flexion-extension, knee rotation and ankle plantarflexion emerged as key discriminative features. Dual-task acquisitions seem to provide information that is coherent, but for fewer features and of reduced magnitude, highlighting the potentially flattening effect of interferences in patients with neurodegenerative disorders.

Conclusions: Our findings suggest that spatiotemporal gait parameters primarily reflect relevant disease-related differences between PD and PSP, while kinematic variables capture more subtle alterations, hypothetically related to motor control strategies. Therefore, kinematic

65 analysis may offer insight about the pathophysiological mechanism underlying FOG in PD
66 patients, whose gait strategy would resemble an approach more similar to that of PSP patients.
67 These results, combined with a deeper anamnestic profiling, could allow for developing
68 integrated biomarkers and a novel phenotyping approach.

69
70 **Keywords:** Gait analysis; kinematic parameters; Parkinson's Disease; freezing of gait; Progressive
71 Supranuclear Palsy.

1. Introduction

Gait impairment represents a core clinical feature of several neurodegenerative disorders, including Parkinson's disease (PD) and progressive supranuclear palsy (PSP). While clinical assessment scales provide an essential overview of motor disability, they could be limited by subjectivity and inter-rater variability or by the presence of undetectable subclinical alterations in the case of early stages of disease

In recent years, technological advances have enabled quantitative measurements [1] to support observation-based assessment, such as motion capture systems, inertial measurement systems, sensorised insoles, force plates, and video analysis systems. Therefore, quantitative gait analysis has gained increasing attention as an objective approach to characterize locomotor dysfunctions, especially the subtler ones [2, 3, 4, 5]. Owing also to these instrumental innovations, it has become clearer that similar neurodegenerative conditions could be differentiate (especially, in early stages) thanks to subclinical features only detectable by Movement Analysis, such as gait slowing or instability, while being driven – apparently – by distinct etiological mechanisms [6]. Nevertheless, different etiologies do not necessarily imply different pathophysiological mechanisms, hence, clinical–phenomenological similarities may arise from analogous pathophysiological responses to distinct etiological factors and may also demand comparable rehabilitative and therapeutic responses or approaches.

Mainly in the field of neurodegenerative diseases, gait analysis has progressively evolved from a descriptive tool to a powerful framework able to help the detection of the underlying pathophysiology, allowing a quantitative characterization of motor dysfunction beyond and, probably, before clinical observation. In Parkinson's disease (PD) and atypical parkinsonisms, gait disturbances represent a core clinical feature and often precede the emergence of explicit disability, making subtle or early locomotion alterations a privileged investigational object into disease mechanisms.

While spatiotemporal gait parameters are well established as markers of disease and progression [7], they predominantly capture the global and comprehensive motor performance, possibly failing to disentangle subtle differences in motor control strategies, especially when assessing early stage of disease or episodic symptoms. This limitation is particularly evident when addressing complex gait phenomena [8, 9] such as freezing of gait (FOG), a disabling, episodic and heterogeneous manifestation [10, 11] whose pathophysiological substrate remains only partially understood [12, 13, 14, 15, 16].

FOG represents one of the most disabling symptoms in Parkinsonism [17] and is associated with increased fall risk and reduced quality of life [18].

FOG is common in PD [19], mainly in advanced stage [20], whereas it occurs early in PSP, representing a key diagnostic clue [21, 22].

The early occurrence of FOG in PSP suggests that PSP gait pattern might represent a reference model for interpreting gait abnormalities observed in PD patients with FOG [6].

Previous gait analysis studies focused on FOG phenomenon or on gait phases immediately preceding or following such phenomenon [2]. However, the extent to which the presence of FOG alters gait patterns, even beyond FOG episodes, like inter-critical walk or sway [23, 24], remains overlooked.

The present study aims to quantitatively compare gait patterns among PD patients with and without FOG and PSP patients, combining spatiotemporal and kinematic analyses. We hypothesize that, while spatiotemporal parameters may primarily aid to discriminate different diseases, kinematic variables could capture subtler alterations related to motor control and symptom-specific mechanisms, such as FOG, hypothetically offering insight about both the nature of such phenomenon and the issue of the phenotypical heterogeneity of parkinsonism.

From these considerations, kinematic variables [25, 26, 27] – particularly those related to axial and appendicular alignment – may reflect motor or cognitive [28] strategies rather than mere consequences of global motor impairment. However, kinematic analysis remains unexplored in comparative studies between PD patients with and without FOG and PSP patients, despite its potential to reveal predisposing motor patterns related to gait initiation, turning, postural control and motor strategy, particularly in terms of environmental adaptability.

Within this conceptual framework, the present study also addresses whether kinematic gait variables can identify a spectrum of motor control strategies spanning PD without FOG, PD with FOG, and PSP, thereby supporting the hypothesis of a phenotypical and pathophysiological bridge between PD phenotypical spectrum and PSP

2. Materials and methods

2.1. Study population and clinical assessment

The study population included 76 patients of which 52 were diagnosed with PD, according to the Movement Disorder Society (MDS) clinical diagnostic criteria [29], and 24 were PSP, in accordance with the MDS PSP criteria [22]. All patients were consecutively enrolled at the Centre for Neurodegenerative Diseases (CEMAND) at the University of Salerno, Italy, after excluding those with dementia, gait requiring support, significant orthopaedic conditions, severe cardiovascular or respiratory diseases, additional neurological disturbances. PD patients were assessed with complete MDS-UPDRS (Unified Parkinson's Disease Rating Scale), Hoehn & Yahr scale, whereas PSP patients were assessed using the PSP rating scale (PSP-RS). All patients underwent the Montreal Cognitive Assessment (MoCA). In addition, PD population was subclassified

according to the presence or absence of FOG experience, self-reported by answers to dichotomous questions, as previously seen in literature [30]. In particular, based on a score greater than or equal to 1 on the thirteenth item of the MDS-UPDRS clinical scale part II, 20 PD patients were classified as freezing-experienced patients (PD+FOG), while 32 patients with a score equal to zero were classified as not freezing-experienced (PD-FOG).

Specifically, it was decided to include in the study only patients reporting anamnestic – i.e., self-reported – freezing of gait, with the aim of ensuring homogeneity of inclusion criteria and capturing individuals with disease burden and gait impairment not severe enough to act as major confounding factors. In this respect, it should be noted that this criterion corresponds, according to recent consensus definitions, to ‘possible’ freezing of gait, representing the lowest level of diagnostic certainty, but offering high sensitivity [11].

The study received approval from the local ethics committee, and all patients signed written informed consent at the time of enrolment.

2.2. Gait analysis and variables

Patients performed gait assessment using the BTS SMART DX-400 optoelectronic system (BTS Bioengineering Corp., Milan, Italy). The system involves six infrared cameras (100 Hz sampling rate), 22 infrared markers (14 mm diameter), a force plate, two video cameras, and a computer with dedicated software. The markers were placed on the patient's body according to the Davis protocol [31]. Each patient performed two tasks: a normal gait, consisting of walking along a 10-metre path at her/his natural speed (single task) and a cognitive dual task, walking on the same path while serial subtracting 7 starting from 100. The tasks were repeated at least 4 times per subject, and the average of 4 trials was taken. The walking paths of the three groups of patients were evaluated using both spatiotemporal and kinematic variables. The spatiotemporal variables were obtained from semi-automatic processing using dedicated BTS system software, while the kinematic variables were calculated from signal processing using a pipeline developed and described in a previous study [28]. Table 1 illustrates the spatiotemporal and kinematic variables assessed in the study.

Table 1. List of spatiotemporal and kinematic parameters used for gait patterns assessment. Kinematic variables were grouped for different joints.

Spatiotemporal parameters	Kinematic	
Cycle duration (s)	Joint	Parameters
Stance duration (s)	Trunk	Anterior, Posterior and ROM Tilt
Swing duration (s)		Internal, External and ROM Rotation
Stance phase (% gait cycle time)		High, Low and ROM Obliquity
Swing phase (% gait cycle time)	Pelvis	Antiversum, Retroversum and ROM Tilt
Single support phase (%)		Internal, External and ROM Rotation
Double support phase (%)		High, Low and ROM Obliquity
Speed (m/s)	Hip	Internal, External and ROM Rotation
Cadence (steps/minute)		Flexion, Extension and ROM flexion/extension

Cycle length (%height)	Knee	Internal, External and ROM Rotation
Step length (m)		Flexion, Extension and ROM flexion/extension
Step width (m)	Ankle	Dorsiflexion, Plantarflexion and ROM Dors-Plantarflexion

Abbreviations: ROM=Range of Motion

2.3. Statistical analysis

Statistical analysis was carried out to find differences among PD patients with FOG, PD patients without FOG and patients with PSP. The ShapiroWilk test was conducted to assess the normality of the data distribution. Descriptive statistical analysis and the parametric or non-parametric test – according to the data distribution – were performed for continuous clinical demographic variables, including age, disease duration, Levodopa Equivalent Daily Dose (LEDD), H&Y, MDS-UPDRS (parts I, II, III, IV and total), MoCA and PSP-RS, while a chi-square test was applied for the categorical variable of gender. For spatiotemporal and kinematic variables, the analysis of covariance (ANCOVA) was performed to examine group differences while controlling for the effect of one or more covariates, as appropriate. For variables showing statistically significant differences, post hoc pairwise comparisons with Bonferroni correction were conducted. The study workflow is summarized in Figure 1.

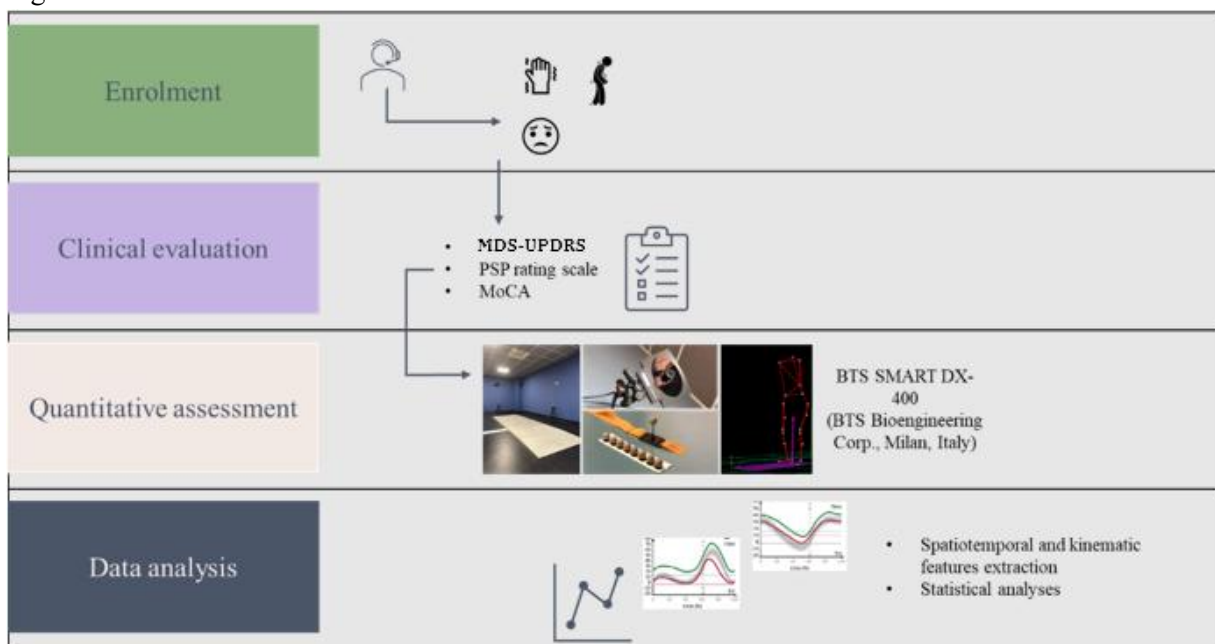


Figure 1. Study workflow.

3. Results

Descriptive statistics and test results, where applicable, for clinical demographic variables are presented in Table 2. Comparisons among three groups were performed using the Kruskal-Wallis test with post-hoc

analysis, whereas two-group comparisons were conducted using the Mann-Whitney U test. The study groups were comparable for gender and age, while statistically significant differences emerged for disease duration between PD and PSP, due to the more rapid disease progression with early stability impairment of patients with PSP, which leads us to select them at an earlier disease stage and, consequently, with shorter disease duration, when they still retain walking independence. LEDD was significantly different among the three groups due to greater mean value for PD+FOG, with post hoc pairwise comparisons showing significant difference between PD+FOG and PSP and a statistical trend between PD+FOG and PD-FOG. As regards the MoCA score, the three groups showed a trend to significance with comparisons revealing difference between PSP patients towards both PD subpopulations. When comparing MDS-UPDRS scores between PD+FOG and PD-FOG, PD+FOG revealed higher mean scores in all parts, except Part III, with strong significative differences for Part I, Part II, Part IV e total score. Descriptive statistics for quantitative variables (kinematic and spatiotemporal) were reported in Table S1 and Table S2 for the single-task walking condition and in Table S3 and Table S4 for the dual cognitive task, in the Supplementary Materials file.

Table 2. Clinical and demographical variables of PD patients with and without FOG and PSP patients. Mean and standard deviation were reported for each group. *p*-values were reported if applicable.

Variables	PD – FOG	PD + FOG	PSP	<i>p</i> -value	PD-FOG vs PD+FOG	PD-FOG vs PSP	PD+FOG vs PSP
Gender (M/F)	19/13	15/5	17/7	0.42	-	-	-
Age (years)	66.88 ± 7.99	65.00 ± 7.49	68.63 ± 6.96	0.37	-	-	-
Disease duration (years)	5.02 ± 2.87	5.80 ± 2.33	1.75 ± 0.44	<0.01*	0.65	0.00	0.00
LEDD	493.10 ± 318.18	720.50 ± 354.55	303.85 ± 243.64	<0.01*	0.08	0.22	0.00
MoCa	22.15 ± 4.31	21.72 ± 3.71	18.63 ± 5.08	0.06	-	-	-
Hoehn & Yahr	1.78 ± 0.42	2.02 ± 0.50	NA	-	0.13	-	-
MDS-UPDRS part I	6.62 ± 4.29	12.60 ± 8.08	NA	-	0.01 [†]	-	-
MDS-UPDRS part II	7.22 ± 6.05	14.40 ± 6.44	NA	-	<0.001 [†]	-	-
MDS-UPDRS part III	22.94 ± 9.08	25.85 ± 10.34	NA	-	0.27	-	-

MDS-UPDRS part IV	1.59 ± 2.98	3.40 ± 3.33	NA	-	0.03 [†]	-	-
Total MDS-UPDRS	38.38 ± 17.18	56.25 ± 21.04	NA	-	0.00 [†]	-	-
PSP-rs IV	NA	NA	7.38 ± 3.21	-	-	-	-
PSP-rs V	NA	NA	5.58 ± 1.91	-	-	-	-
PSP-rs total	NA	NA	32.13 ± 9.85	-	-	-	-

Abbreviations: LEDD=Levodopa Equivalent Daily Dose; MDS-UPDRS= Movement Disorder Society-Unified Parkinson's Disease Rating Scale; PSP-rs=Progressive Supranuclear Palsy rating scale. *Statistically significance level <0.05. Three-group comparisons were assessed using the Kruskal-Wallis test with post-hoc analysis. † Two-group comparisons were performed using the Mann-Whitney U test. Statistical significance was set at $p < 0.05$.

With regard to spatiotemporal and kinematic variables, disease duration was included as a covariate in the ANCOVA. The results for the spatiotemporal parameters extracted from both the single-task and dual cognitive walking conditions are presented as heatmaps in Figure 2 and 3, respectively. For each parameter, the overall p-value for group differences is reported, followed by the p-values for post-hoc pairwise comparisons. The colour scale ranges from green (statistically significant difference) to red (no statistically significant difference), with statistical trends indicated in yellow.

Concerning the single walking task, excluding step length (in meters), swing phase (as % of gait cycle time) and single support phase (as %), all the spatiotemporal parameters resulted statistically significant different in the comparison among the three groups. Such differences were primarily driven by contrasts between PD and PSP, as highlighted by post-hoc analysis, with both PD-FOG and PD+FOG groups demonstrating several significant differences compared with the PSP group. In particular, PSP as compared with both PD+FOG and PD-FOG showed similar statistically significant increase in cycle duration, stance duration, swing duration, stance phase (as % of gait cycle time), double support phase (as % gait of cycle time); and statistically significant decrease towards PD patients in cadence (steps/minute) and cycle length (height corrected).

Similarly, in the dual cognitive task condition, fewer significant differences were observed between patients with PSP and those with PD, regardless of the presence or absence of FOG. Specifically, PSP as compared with both PD+FOG and PD-FOG showed similar statistically significant increase in cycle duration, stance duration, swing duration, double support phase (as % gait cycle time), while exhibiting statistically significant decrease towards both PD+FOG and PD-FOG in speed, cadence (steps/minute), cycle length (height corrected).

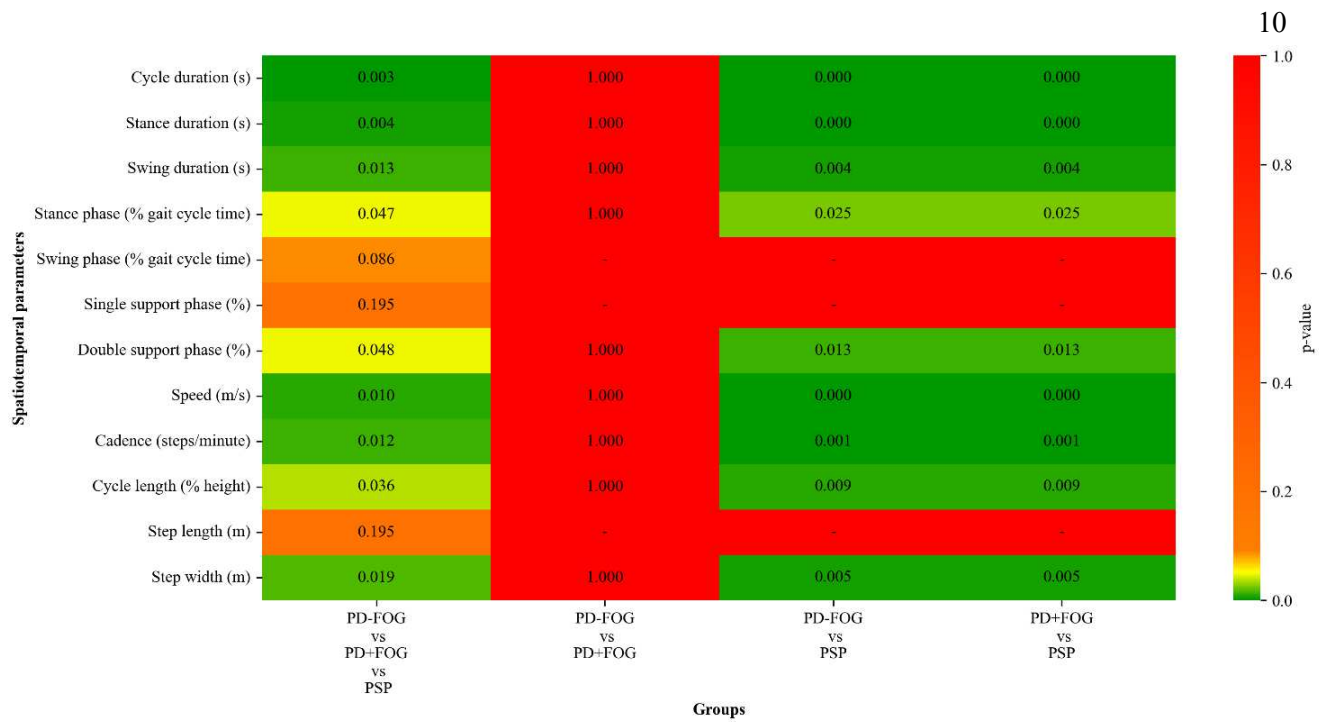


Figure 2. Heatmap of p-values for spatiotemporal parameters of single walking task obtained from the comparison between three clinical groups (PD-FOG, PD+FOG, and PSP). The rows show the spatiotemporal parameters, while the columns show the comparisons between groups and pairs. The colour scale indicates the level of statistical significance. For variables that did not show statistical significance between the three groups, no post hoc tests were performed.

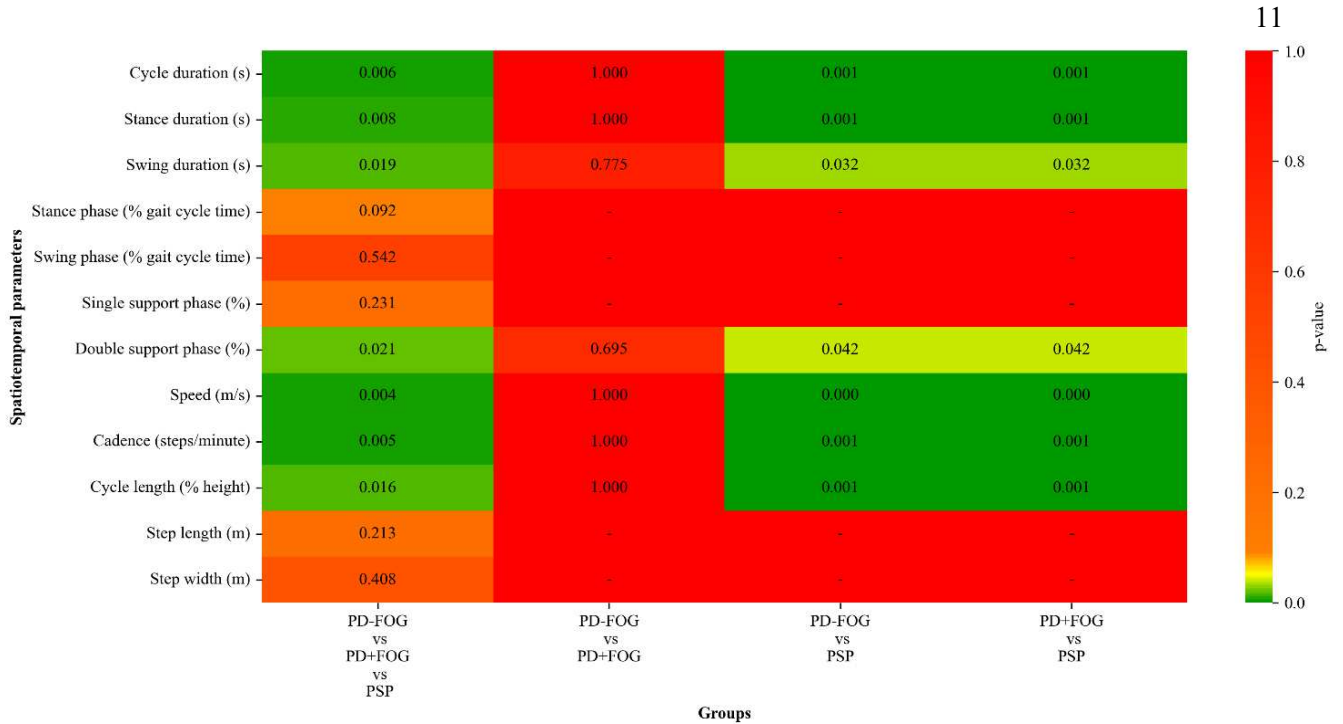


Figure 3. Heatmap of p-values for spatiotemporal parameters of dual cognitive task obtained from the comparison between three clinical groups (PD-FOG, PD+FOG, and PSP). The rows show the spatiotemporal parameters, while the columns show the comparisons between groups and pairs. The colour scale indicates the level of statistical significance. For variables that did not show statistical significance between the three groups, no post hoc tests were performed.

Concerning kinematic variables, Figure 4 shows the violin plots of the three groups (PD-FOG vs PD+FOG vs PSP) of the statistically significant variables of the different joints in the single task. Additionally, for each general shape of the distribution, the actual points were overlapped. Similarly, Figure 5 shows the violin plots of the statistically significant kinematic variables of different joints in the comparison among the three groups in the cognitive dual task. For kinematic variables the heatmaps containing the p-values for each variable of a specific joint were reported in Supplementary materials file (Figures S1 to S5 refer to variables extracted from the single task gait; Figures S6 to S10 refer to variables extracted from the cognitive dual task).

In particular, kinematic analysis in single task revealed significant differences at the pelvic, knee, and ankle levels, distinguishing again PSP from PD, but also highlighting several differences between PD+FOG and PD-FOG. In particular, kinematic variables stood along a spectrum spanning across PD-FOG, PD+FOG and PSP where, at one end, PD-FOG showed reduced trunk obliquity (in terms of ROM), pelvic tilt characterized by more positive antiversum and retroversum angles, a greater hip flexion with decreased hip extension, a greater knee ROM rotation, especially due to wider external rotation, and an increased plantarflexion. At the other end, PSP showed a reduced and posteriorized pelvic tilt (of both anteversum and retroversum), a reduced knee flexion and a peculiar knee rotation, with greater internal rotation and reduced external rotation (intrarotated configuration) and a lower plantarflexion (with a statistical trend towards PD patients). While PD+FOG showed in-between values on such joints. When analysing kinematic variables in

dual task, significant differences emerged at both the pelvic and the knee level in the comparison between PD population and PSP patients, whereas the only difference between PD-FOG versus PD+FOG was the hip extension which was increased in PD+FOG (extended posture).

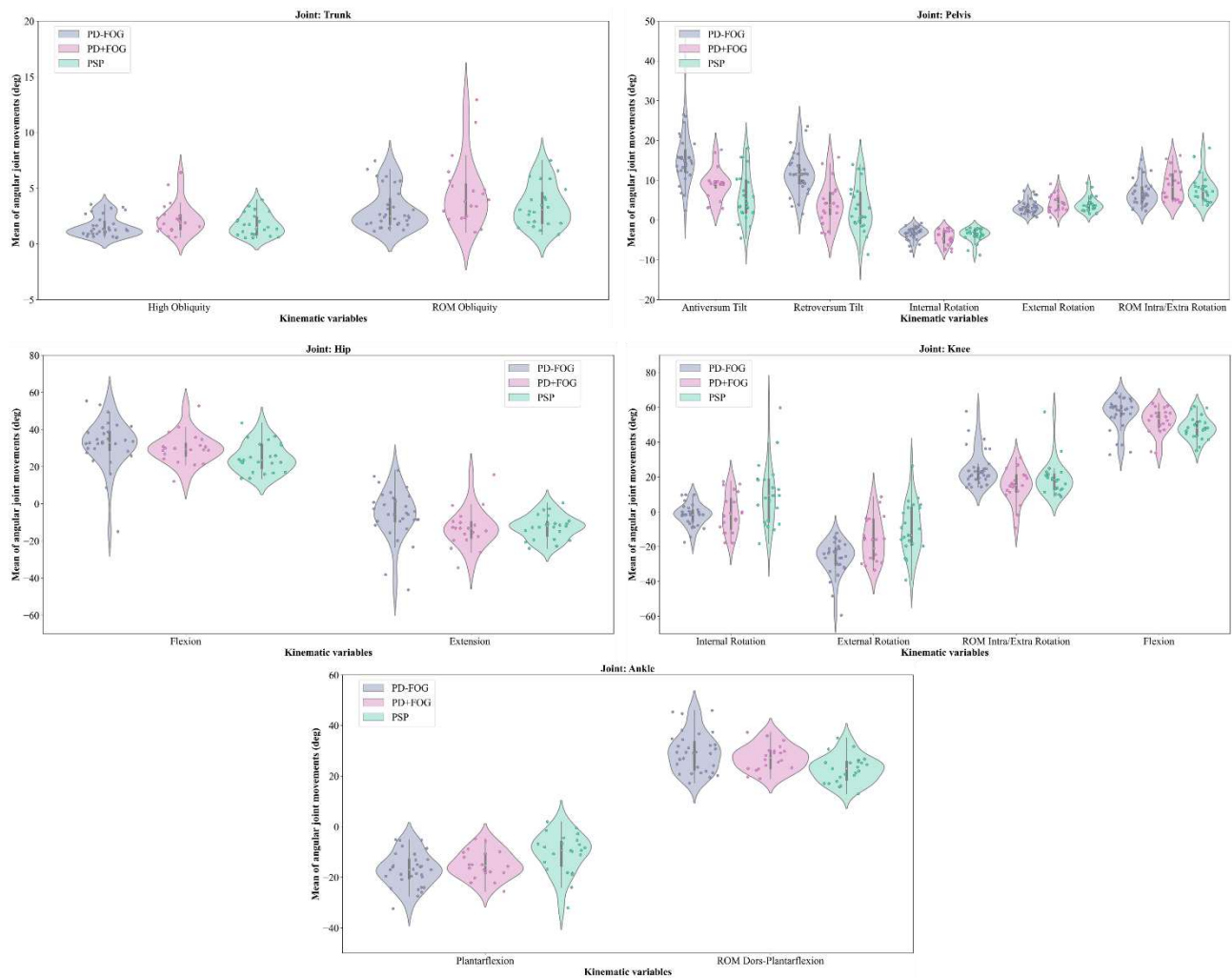


Figure 4. Violin plots of statistically significant kinematic parameters for different joint angle. Violin plots show the variability and density of the measured values for each variable, with overlapping points representing individual observations.

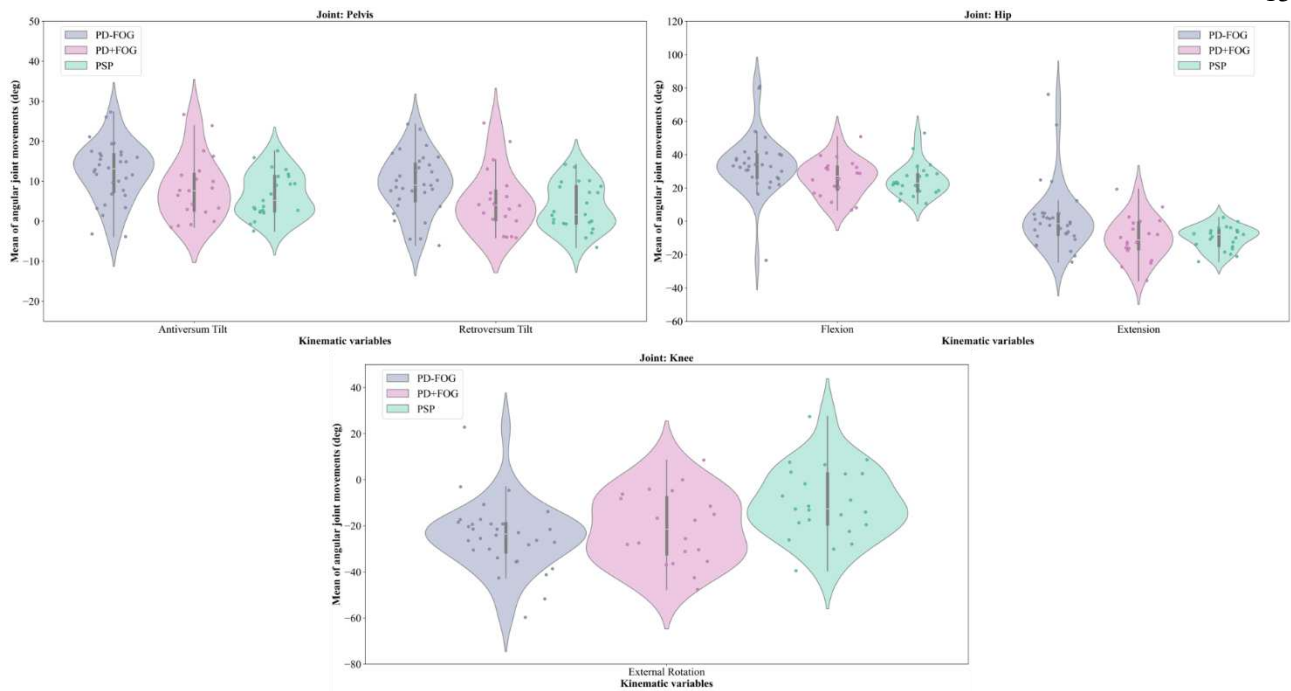


Figure 5. Violin plots of statistically significant kinematic parameters for different joint angle extracted from dual cognitive task. Violin plots show the variability and density of the measured values for each variable, with overlapping points representing individual observations.

4. Discussion

The current study indicates a possible kinematic gait metrics spectrum encompassing PD-FOG, PD+FOG and PSP patients. Specifically, PD+FOG patients would exhibit a kinematic pattern posing them in the middle between PD-FOG and PSP patients suggesting that the presence of FOG might identify a peculiar PD phenotype sharing kinematic features with PSP patients.

It is worth noting that in the present study, PD+FOG and PD-FOG patients were comparable for demographic characteristics and disease duration and exhibited overlapping global cognitive performance on the Montreal Cognitive Assessment (MoCA), showing similar motor scores on the MDS-UPDRS Part III. This occurred in the context of a predictable significantly greater dopaminergic load in the PD+FOG group, as reflected by higher levodopa equivalent daily dose (LEDD). In contrast, the remaining MDS-UPDRS domains consistently revealed a more severe clinical burden in PD+FOG patients. Although Part IV differences may plausibly be driven by treatment-related factors, the systematic divergence observed in Parts I and II further raises the possibility of distinct phenotypic trajectories. Within this framework, PD+FOG patients appear to be characterized by a broader and more disabling spectrum of motor and non-motor impairments, with a disproportionate impact on activities of daily living and overall quality of life, closely resembling those usually referred to other parkinsonism, such as PSP. Importantly, these alterations seem likely to remain partially underdetected in routine clinical evaluations if not specifically addressed, as they predominantly emerge during daily living activities rather than at the time of examination. The more pervasively ecologic and patient-centered (given the self-reported) nature of the information derived from Parts I and II further suggests that the differences observed between PD+FOG and PD-FOG are not confined to episodic or anecdotal events – for instance, those typically associated with an ictal and unpredictable phenomenon, like the freezing of gait – but rather extend broadly across the patient’s lived experience. Concurrently and accordingly with these results, quantitative gait analysis was designed to identify potential subclinical alterations that might characterize this population that, therefore, would represent a distinct phenotype even when FOG is still mild and only referred after a specific question (item 13 of the MDS-UPDR part II).

In line with earlier research [7], in the present study, spatiotemporal factors successfully distinguished PSP patients from both PD groups, demonstrating their sensitivity to axial dysfunction and disease-related severity. In contrast, there were no spatiotemporal differences between PD patients with and without FOG, possibly underscoring the limited discriminatory power of these parameters for capturing the complexity of FOG-related phenomenon and its ancillary motor dysfunction. Interestingly, kinematic analysis revealed a more nuanced, informative pattern. Alterations at the pelvic, knee, and ankle levels not only distinguished PSP from both PD groups but also highlighted differences between PD+FOG and PD-FOG patients. Hence, specific kinematic variables seem to fall on a continuum, with PD-FOG at one end (with more physiological features), PSP at the other (with more dysfunctional changes) and PD+FOG occupying an intermediate position. This spectrum could support the hypothesis that FOG in PD patients may reflect that their motor control strategies are more closely resembling those observed in PSP.

Id est, pelvic tilt changes in PD+FOG – with a more posteriorized kinematic configuration, resulting in concordantly shifting of anteversum and retroversum tilt, similarly to PSP patients – may indicate an early impairment in axial control and trunk–lower limb coordination, which both are critical for alterations in gait ignition and dynamic balance. Similarly, changes in knee kinematic – showing a reduced range of movement and a greater tendency toward internal rotation, in analogous directions of PSP features – could suggest compensatory – often necessary but maladaptive – strategies to sustain walking in the presence of poorer supraspinal regulation. In summary, PD+FOG and PSP patients appear to share a distinctive kinematic postural pattern, characterized by a posteriorly shifted pelvic girdle and a more constrained configuration of the knees and ankles. Actually, the study of kinematic variables captured peculiar motor configurations (and related cognitive strategies) and/or secondary adaptations to specific disease phenotypes: some PD patients – in the present case, those with FOG – may exhibit a latent PSP-like gait signature. From a pathophysiological outlook, these results enhance the hypothesis that FOG in PD may emerge within/from an early impairment of axial and appendicular coordination, resembling the motor strategy observed in PSP. Theoretically, this interpretation aligns with models [10, 13, 14] that describe FOG as a network-level disorder involving frontal, brainstem, and cerebellar circuits rather than a purely dopaminergic phenomenon, echoing the concept that this phenomenon arises from a complex, anatomically and functionally higher disruption in the integration of posture, balance, locomotor planning and ecological adaptability, that resemble the one responsible for the motor features of PSP patients.

It is of particular interest to note that similar results are observed under cognitive dual-task conditions, although certain previously significant findings are attenuated or gone. Accordingly, it could be hypothesized the presence of a specific dual-task effect, whereby the cognitive overload induced by an interfering task asymptotically flattens performances throughout groups, especially for some parameters – both spatiotemporal and kinematic – to the point of abolishing differences previously significant in single-task.

Notably, the kinematic parameters that remain discriminative under dual-task conditions may correspond to the features more robustly preserved in a phenotype (PD-FOG) and more/early vulnerable in the other (PD+FOG); consequentially, the more characteristic of a given disease or phenotype. In this perspective, the negative imprint emerging from this “condensed” spectrum of significances obtained by dual-task acquisitions (compared to the ones from single-task acquisitions) may, to some extent, also highlight the gradual and consequent gait alterations occurring over the analogous pathogenetic course of different phenotypes of the same diseases [32] ; in this regard, a more precise understanding of the sequence of preservation / adaptation / loss of certain features may also support the development of specific, preventive rehabilitative interventions.

Consistent with our initial premises, quantitative movement analysis has demonstrated its ability to uncover subclinical differences capable of disentangling otherwise indistinguishable conditions, namely gait patterns of PD patients with and without FOG beyond FOG episodes, thus revealing different pattern underlying presumed different phenotypes. In addition, our findings, in a manner equally plausible and conceptually compelling, also revealed convergent patterns across distinct disease entities, pointing toward partially overlapping pathophysiological mechanisms and shared compensatory strategies in distinct conditions such as PD+FOG and PSP.

The present study shows some limitation: first, the sample size remains relatively modest, and further validation in larger cohorts will be necessary to confirm the robustness and generalizability of these findings. Second, as outlined in the Methods section, inclusion criteria relied on the presence of self-reported freezing of gait (FOG); notably, according to the most recent consensus definitions, anamnestic FOG is currently classified only as a “possible” rather than definitive phenomenon. However, this choice was intentional and aligned with the specific aims of the study, targeting a cohort of patients with minimal functional impairment, that could be assessed under conditions that are intercritical, capturing only early or subclinical alterations. Within this framework, the observed kinematic differences appear to support the existence of a distinct motor phenotype in patients reporting anamnestic FOG, suggesting that even self-reported episodes may reflect underlying pathophysiological divergence.

Conclusion

The present findings might suggest that while spatiotemporal gait parameters primarily reflect disease-related differences between PD and PSP, kinematic analysis could reveal more subtle alterations in motor control strategies. The identification of a kinematic continuum extending from PD without FOG to PSP – with PD with FOG in the middle – could support the notion that parkinsonian syndromes share phenotypical manifestations, potentially representing similar underlying physiopathological drive: from this perspective, the presence of freezing of gait may represent a phenotypical bridge between PD and PSP.

Clinically, this highlights the potential of kinematic gait analysis as an early, objective biomarker for identifying PD patients at risk of developing FOG or with subtle motor profile already suggestive of or equivalent to early freezer; more ambitiously, it may prompt a phenomenological and descriptive reconsideration of our current conceptualization of nosographic entities, clinical phenomena and their underlying pathophysiological mechanisms.

Future longitudinal studies are required to determine whether such kinematic signatures could even precede the clinical onset of FOG and whether they can be integrated into predictive models of disease progression. Furthermore, it would be valuable to harmonize findings derived from our optoelectronic recordings with data obtained from more ecologic, affordable, scalable – wearable or wearless – systems [33], in order to facilitate multicentric studies or secondary analyses on already existing external datasets.

379 **Statement and Declaration**

380 The authors declare that the research was conducted in the absence of any commercial or financial
381 relationships that could be construed as a potential conflict of interest.

382 No funding was received to assist with the preparation of this manuscript.

383 The authors have no competing interests to declare that are relevant to the content of this article.

384

385 **Authors' contribution**

386 M.A., P.B., M.P., F.A., N.P., F.D. conceived the presented idea. M.A. C.R., N.P., F.D. developed the
387 theory. M.A., C.R., M.R., N.P., F.D. verified the analytical methods. F.D., M.R., N.P., D.B., A.V.,
388 G.D., L.S. carried out the planned experiments. N.P., C.R., M.R. performed the computations. All
389 authors discussed the results, contributed to the final manuscript and reviewed it.

390

391 **Data access statement**

392 The data that support the findings of this study are not openly available due to reasons of sensitivity
393 and are available from the corresponding author upon reasonable request. Data are located in
394 controlled access cloud shared among the authors of the present study.

395

396 **Ethical standards**

397 All people recruited for the study gave their informed consent prior to their inclusion. All acts were
398 performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and
399 its later amendments and with the guidelines agreed for research protocols by the Center for
400 Neurodegenerative Diseases of Salerno with the Ethics Committees of “South Naples” and
401 “Campania 2”.

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