Parkinsonism and Related Disorders 147 (2026) 108081

Contents lists available at ScienceDirect

Parkinsonism and Related Disorders

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

Short communication

Differential gait disturbances in patients with Parkinson's disease and normal pressure hydrocephalus

Carolin Semmler [a,*], Veronika Wunderle [a], Taylan D. Kuzu [a], Oezguer A. Onur [a], Christian Grefkes [b], Gereon R. Fink [a,c], Michael T. Barbe [a], Peter H. Weiss [a,c]

[a] University of Cologne, Faculty of Medicine and University Hospital Cologne, Department of Neurology, Cologne, Germany
[b] Department of Neurology, University Hospital Frankfurt, Goethe University Frankfurt, Frankfurt (Main), Germany
[c] Institute of Neuroscience and Medicine (INM-3), Cognitive Neuroscience, Forschungszentrum Jülich, Jülich, Germany

ARTICLE INFO

Keywords:
Levodopa
CSF
Force
Pressure-distribution
Instrument-supported locomotion assessment

ABSTRACT

Introduction: Gait disturbances are a key symptom in normal pressure hydrocephalus (NPH) and Parkinson's disease (PD). We employed instrument-supported gait analysis to characterize commonalities and differences in gait patterns among PD and NPH patients, as well as gait changes following disease-specific interventions (i.e., spinal tap and dopaminergic medication).

Methods: Gait and balance analyses were conducted i) before and after NPH patients (n = 19) received a spinal tap, and ii) before and after PD patients (n = 19) received soluble dopaminergic medication. Change scores were calculated relative to each patient's baseline performance. Group differences were analyzed using independent sample t-tests or Mann-Whitney U tests and within-group changes using paired sample t-tests or Wilcoxon signed-rank tests.

Results: Static postural control (i.e., balance) did not differ between patient groups, but dynamic postural control did, with PD patients showing significantly reduced mediolateral sway and gait width compared to NPH patients. These parameters did not change after disease-specific interventions. For gait dynamics, PD patients demonstrated significantly higher values in pace and force parameters, which improved significantly in both groups following intervention. Finally, gait regularity showed lower variability and better rhythm-related parameters in PD patients compared to NPH patients, along with differential changes after interventions.

Conclusion: Except for static postural control (i.e., balance), instrument-supported gait analysis revealed several characteristic gait differences between PD and NPH patients. Variability and rhythm, reflecting gait regularity, were best suited to differentiate gait disturbances in patients with PD and NPH.

1. Introduction

Gait disturbances are common in neurological disorders, significantly impairing mobility and quality of life [1]. Nevertheless, accurate assessment of gait disturbances in patients with Parkinson's disease (PD) and idiopathic normal pressure hydrocephalus (NPH) remains challenging, especially in early disease stages and due to high inter-individual variability. Clinical gait assessments often rely on subjective interpretation and lack the precision to detect subtle gait alterations. Instrument-supported gait analysis (iGA) has emerged as a powerful tool for quantifying spatiotemporal and kinetic gait parameters [2].

PD and NPH present with clinically overlapping gait abnormalities, such as reduced gait velocity and stride length, yet differ in pathophysiology and treatment approaches [3]. In PD, gait analyses revealed reduced stride length, increased step-to-step variability, and episodic freezing of gait compared to healthy, age-matched controls [4]. In contrast, NPH is characterized by a distinct "magnetic" gait with widened stance, reduced foot elevation, and impaired anticipatory postural adjustments [5]. In addition, differential gait characteristics were reported, such as relatively prolonged stance and double limb support phases in NPH compared to PD [1].

Moreover, iGA can quantify treatment effects. In NPH, gait improvements following spinal tap predict responsiveness to shunting [5], while in PD objective gait metrics reflect responses to dopaminergic therapy and deep brain stimulation and thus enable individualized

* Corresponding author.
E-mail address: carolin.semmler@uk-koeln.de (C. Semmler).

https://doi.org/10.1016/j.parkreldis.2025.108081
Received 1 August 2025; Received in revised form 9 October 2025; Accepted 10 October 2025
Available online 10 October 2025

treatment strategies [4].

Despite its potential, quantitative iGA is rarely integrated into routine clinical practice. In this study, we used iGA to compare postural control and gait domains in PD and NPH patients before and after disease-specific interventions (i.e., spinal tap in NPH and dopaminergic medication PD). We aimed to disclose balance and gait parameters that differentiate between the two hypokinetic gait disorders and hypothesized that distinct gait patterns would emerge at baseline and would change differentially after disease-specific interventions.

2. Methods

2.1. Participants

This retrospective study included patients with NPH and PD, who were admitted to the Department of Neurology, University Hospital Cologne, for clinical evaluation, including iGA. The study was conducted following the Declaration of Helsinki and was approved by the local ethics committee (study no.: 25-1151-retro).

Cognitive function was assessed using the DemTect in NPH and the MoCA in PD patients. DemTect-scores were converted into MoCA scores for group comparison [6].

2.2. Inclusion and exclusion criteria

NPH diagnosis was based on clinical criteria, including gait disturbance, cognitive decline, urinary incontinence, and structural brain imaging abnormalities [7]. All 19 patients fulfilled the criteria for probable NPH based on NPH guidelines (i.e., gait disorder plus urinary incontinence and/or cognitive impairment; [7]). PD diagnosis was made by experienced neurologists based on neurological examination, clinical history, the presence of cardinal motor symptoms (bradykinesia, rigidity, resting tremor), and the systematic exclusion of alternative causes. Only patients with a clinically established diagnosis of idiopathic PD were included, while patients with (suspected) atypical or secondary Parkinsonian syndromes or deep brain stimulation (DBS) were excluded.

General exclusion criteria for both patient groups were current or previous diseases affecting the central nervous system and the inability to walk the required distances.

2.3. Gait and balance assessment

IGA was performed using ground reaction force plates and pressure distribution platforms, as previously described [2]. In summary, patients performed walking tasks on a Leonardo Mechanograph® Gangway (600 × 77 cm, 400 Hz; Novotec Medical GmbH, Pforzheim, Germany) and two consecutively placed zebris® FDM pressure distribution platforms (total length 300 × 60.5 cm, 50 Hz; zebris Medical Systems, Tübingen, Germany). The pressure distribution platforms were extended by two wooden plates at the beginning and end in order to exclude gait initiation and termination steps from the analysis. For the standing and sitting balance tasks, a Leonardo Mechanograph® Ground Reaction Force Plate (66 × 66 cm, 800 Hz; Novotec Medical GmbH, Pforzheim, Germany). For the latter one, a modular bench was used with a sitting height of 46 cm.

The following gait parameters were calculated and assigned to the respective gait domain (adapted from Lord and colleagues [8]).

(Dynamic) Postural control.

- Gait width: Lateral distance between the feet during walking, given in meters (m)
- Mediolateral sway: Side-to-side movement of the body's center of gravity, given in meters (m)

Pace.

- Gait velocity: Average speed of walking, given in meters per second (m/s)
- Step length: Distance between the heel strikes of two consecutive steps by opposite feet, given in meters (m)

Force.

- Maximum force for heel-strike/toe-off: Peak ground reaction forces during initial contact (heel-strike) and final push-off (toe-off), given in percentage of the patients' body weight (% body weigth)
- Height difference of the body's center of gravity: Vertical displacement of the body's center of gravity throughout the gait cycle, given in centimeters (cm)

Rhythm.

- Relative swing phase: Proportion of the gait cycle during which one foot is in the air, given in percentage (% gait cycle)
- Relative double limb support phase: Proportion of the gait cycle during which both feet are in contact with the ground, given in percentage (% gait cycle)

Variability.

- Intra-individual step length variability: Fluctuations in step length across consecutive steps by the same individual, given as the coefficient of variance in percent (CoV %)
- Gait width variability: Fluctuations in step width across consecutive steps by the same individual, given as standard deviation in centimeters (Δcm)

For both the standing and sitting balance analysis, sway was quantified as the area of an ellipse given in cm^2, while variability in the forces transmitted through the feet onto the force plate, given in Newton per kilogram, additionally reflected fluctuations in balance control.

All patients were assessed before and after disease-specific interventions (i.e., spinal tap in NPH patients, dopaminergic medication in PD patients). In NPH patients, the median time between spinal tap and subsequent gait assessment was 21 h (IQR = 19–32). While the majority of patients (15/19) was tested within 2 days, three patients were assessed within one week after discharge as out-patients and one patient after 12 days due to a COVID-19 infection. The distribution of the individual intervals between spinal tap and subsequent gait assessment are displayed in Fig. S1 of the supplementary material. PD patients were measured off-mediation after ≥14 h withdrawal of dopaminergic medication. For the disease-specific intervention, most PD patients (18/19) received 200 mg of soluble L-Dopa; one patient received 300 mg (mean: 205.6 ± 23.6 mg). On average, motor symptoms improved by 36.9 ± 18.1 % (UPDRS-III assessment: medOFF: 31.0 ± 10.5, medON: 20.5 ± 10.4), indicating moderate symptom severity in the OFF state with a clinically meaningful improvement to milder symptoms in the ON state. The post-intervention gait assessment was conducted within 1 h after L-Dopa onset. At the time of the assessment, the mean levodopa equivalent dose (LEDD) was 914.7 mg (SD = 584.3). Three patients had a levodopa dose of 0 mg at admission; in two cases, the inpatient stay served to initiate the first levodopa treatment following PD diagnosis, while one patient had previously discontinued oral levodopa due to poor tolerability and was re-evaluated during their hospitalization. In this latter case, the levodopa challenge test was performed with 300 mg soluble levodopa, resulting in a robust motor improvement of 32.7 %, which subsequently allowed for successful re-initiation of levodopa therapy.

2.4. Statistical analysis

On average, 42.7 (SD: 7.6) steps were captured on the gangway and 32.3 (SD: 7.8) steps on the pressure walkway per patient and time point (i.e. before and after spinal tap or levodopa intake). Baseline group differences were analyzed using ANCOVAs with age, body height, and cognitive scores as covariates. To examine within-group changes caused by the interventions, absolute pre-post differences (i.e. changes expressed in the original units of each parameter, including %-points for variability measures) were analyzed using paired sample t-tests and Wilcoxon signed-rank tests. Correction for multiple comparisons was performed using the Benjamini-Hochberg method. In NPH patients, a Spearman's rank correlation was applied to evaluate whether the interval between spinal tap and subsequent gait assessment was associated with patients' responsiveness, defined as the improvement in gait velocity. A significance level of $\alpha = 0.05$ was set for all statistical analyses.

3. Results

3.1. Patient samples

Nineteen NPH and 19 PD patients were enrolled with 14 men in both groups ($\chi^2(1) = 0.00$, $p = 1.000$). NPH patients were older (75.4 ± 7.3 years vs. 65.6 ± 10.1 years, $p = 0.002$) and smaller than PD patients (169.5 ± 8.4 cm vs. 176.4 ± 8.4 cm, $p = 0.007$), while their weight (80.2 ± 13.6 kg vs. 87.7 ± 17.7, $p = 0.166$) and body mass index (27.2 ± 4.8 vs. 28.1 ± 5.0, $p = 0.540$) did not differ significantly. NPH patients had lower cognitive scores (i.e., $MoCA_{transformed}$: 20.7 ± 4.1 vs. 23.9 ± 4.0, $p = 0.035$). There was no significant association between the interval between spinal tap and subsequent gait assessment and the responsiveness of the NPH patients to the spinal tap, defined as the change in gait velocity ($\rho = 0.33$, $p = 0.159$).

Table 1
Results of the instrument-supported gait analysis in patients with NPH and PD.

	Domain	Parameter	Comparison between patient groups at baseline (ANCOVA) Mean (SD)	Absolute changes to baseline in patients with PD (paired-sample *t*-test) Mean (SD)	Absolute changes to baseline in patients with NPH (paired-sample *t*-test) Mean (SD)
A - Balance/static postural control	Balance sitting	Sitting ellipse [cm^2]	PD: 0.05 (0.03) NPH: 0.09 (0.08) $p_{corr} = 0.147$	+0.01 (0.05) $p_{corr} = 0.446$	−0.2 (0.10) $p_{corr} = 0.836$
		Sitting force SD [N/kg]	PD: 0.005 (0.003) NPH: 0.006 (0.002) $p_{corr} = 0.147$	−0.001 (0.003) $p_{corr} = 0.573$	0.000 (0.002) $p_{corr} = 0.837$
	Balance standing	Standing ellipse [cm^2]	PD: 9.97 (10.84) NPH: 13.19 (12.82) $p_{corr} = 0.294$	−2.08 (12.17) $p_{corr} = 0.872$	−2.35 (9.78) $p_{corr} = 0.557$
		Standing force SD [N/kg]	PD: 0.023 (0.021) NPH: 0.043 (0.056) $p_{corr} = 0.512$	−0.007 (0.020) $p_{corr} = 0.486$	−0.019 (0.050) $p_{corr} = 0.557$
B - Dynamic postural control	Postural control during gait	Mediolateral sway [m]	PD: 0.09 (0.08) NPH: 0.13 (0.04) **$p_{corr} = 0.037$**	+0.02 (0.06) $p_{corr} = 0.198$	+0.00 (0.03) $p_{corr} = 0.764$
		Gait width [cm]	PD: 13.55 (4.20) NPH: 20.62 (4.33) **$p_{corr} = 0.002$**	−0.27 (1.19) $p_{corr} = 0.453$	−1.69 (2.69) $p_{corr} = 0.058$
C - Gait dynamics	Pace	Gait velocity [m/s]	PD: 0.40 (0.07) NPH: 0.27 (0.09) **$p_{corr} = 0.006$**	+0.05 (0.05) **$p_{corr} < 0.001$**	+0.06 (0.05) **$p_{corr} < 0.001$**
		Step length [m]	PD: 0.49 (0.13) NPH: 0.29 (0.12) **$p_{corr} = 0.014$**	+0.11 (0.11) **$p_{corr} < 0.001$**	+0.10 (0.06) **$p_{corr} < 0.001$**
	Force	Height difference [cm]	PD: 1.74 (0.78) NPH: 0.92 (0.46) **$p_{corr} = 0.015$**	+0.47 (0.60) **$p_{corr} = 0.004$**	+0.23 (0.03) **$p_{corr} = 0.003$**
		Max heel-strike force [% body weight]	PD: 20.96 (12.57) NPH: 8.20 (5.18) **$p_{corr} = 0.015$**	+10.00 (10.57) **$p_{corr} = 0.004$**	+4.27 (6.28) **$p_{corr} = 0.016$**
		Max toe-off force [% body weight]	PD: 17.42 (9.27) NPH: 7.82 (5.82) **$p_{corr} = 0.008$**	+6.34 (6.12) **$p_{corr} = 0.004$**	+2.47 (4.85) **$p_{corr} = 0.045$**
D - Gait regularity	Variability	Intra-individual step length variability [CoV %]	PD: 11.50 (6.21) NPH: 31.01 (18.98) **$p_{corr} = 0.005$**	+0.89 (10.84) $p_{corr} = 0.872$	−12.00 (15.37) **$p_{corr} = 0.008$**
		Gait width variability [Δcm]	PD: 25.21 (16.17) NPH: 9.28 (5.26) **$p_{corr} = 0.001$**	+6.34 (6.12) **$p_{corr} = 0.023$**	+1.99 (5.10) $p_{corr} = 0.185$
	Rhythm	Double limb support phase [% gait cycle]	PD: 36.67 (4.35) NPH: 51.47 (11.38) **$p_{corr} = 0.009$**	−4.02 (6.46) $p_{corr} = 0.072$	−6.27 (9.62) **$p_{corr} = 0.037$**
		Swing phase [% gait cycle]	PD: 31.66 (2.25) NPH: 24.21 (5.73) **$p_{corr} = 0.009$**	+1.97 (3.26) $p_{corr} = 0.072$	+3.14 (4.76) **$p_{corr} = 0.037$**

NPH = Normal Pressure Hydrocephalus, PD = Parkinson's disease, ANCOVA = Analysis of covariance, SD = standard deviation, CoV % = Coefficient of variance given in percent, N = Newton, pcorr = corrected for multiple comparison using the Benjamini-Hochberg method.

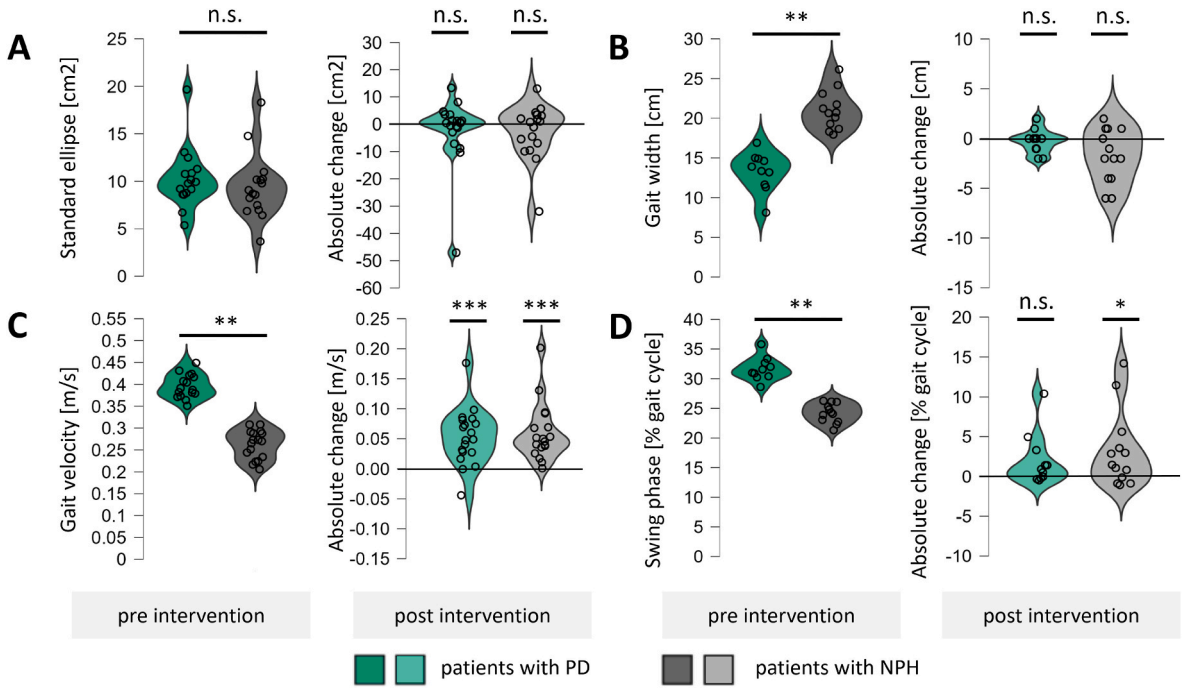

Fig. 1. Overview of the different result patterns with exemplary balance and gait parameters.
A. Static postural control, exemplified by the standing balance, did not show a significant group difference at baseline (left panel) and was not significantly modulated by the disease-specific intervention (right panel).
B. At baseline, the two patient groups differed in their dynamic postural control, represented by the gait width (left panel). However, dynamic postural control was not affected by the disease-specific interventions (right panel).
C. Gait velocity as a representative parameter for the domain pace showed significant group differences at baseline (left panel) and similar changes in both patients in response to the disease-specific interventions (right panel).
D. The gait domain rhythm, reflected by the relative swing phase, not only showed significant group differences at baseline, but also differential changes after the disease-specific interventions (right panel).
Data from Parkinson's disease (PD) patients are depicted in green colors, data from patients with normal pressure hydrocephalus (NPH) are shown in grey colors. n.s. = not significant, * = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

3.2. Balance assessment

Static balance while sitting and standing did not significantly differ between PD and NPH patients at baseline (all $p_{corr} > 0.147$, see Table 1 and Fig. 1A). Likewise, the change scores for the sway area and force were not significant for both groups (all $p_{corr} > 0.446$).

3.3. Gait assessment

For dynamic postural control, NPH patients presented with significantly broader baseline gait width compared to PD patients (20.62 ± 4.33 cm vs. 13.55 ± 4.20 cm, $p_{corr} = 0.002$). However, the absolute differences between pre- and post-intervention gait width did not deviate significantly from zero for both groups (NPH: 1.69 ± 2.69 cm and PD: 0.27 ± 1.19 cm, all $p_{corr} > 0.058$). Similarly, the mediolateral sway significantly differed between groups at baseline (NPH: 0.13 ± 0.04 m vs. PD: 0.09 ± 0.08 m, $p_{corr} = 0.037$), but did not significant change after the disease-specific intervention in NPH or PD patients (+0.00 ± 0.03 m and +0.02 ± 0.06 m, both $p_{corr} > 0.198$). Hence, at baseline, NPH patients presented significantly increased side-to-side movement while walking compared to PD patients. However, both groups did not show statistically significant intervention-induced changes in this parameter.

For the gait domains pace and force, reflecting gait dynamics, both groups differed significantly in their gait velocity, step length, height difference of the body's center of gravity, and maximum force during the heel-strike and toe-off phases at baseline and showed significant improvements after the respective interventions (Table 1, Fig. 1C). This pattern suggests that both interventions were similarly effective in improving gait dynamics across groups.

The gait domains reflecting gait regularity, i.e., variability and rhythm, showed group differences at baseline and differential changes after respective intervention (Table 1). At baseline, NPH patients showed significantly higher step length variability (31.01 ± 18.98 CoV %) than PD patients (11.50 ± 6.21 CoV %, $p_{corr} = 0.005$), which decreased significantly in NPH patients after spinal tap (−12.00 ± 15.37 CoV %, $p_{corr} = 0.008$), but not in PD patients after the intake of dopaminergic medication (+0.89 ± 10.84 CoV %, $p_{orr} = 0.872$). The inverse pattern was observed for the gait width variability (PD: 25.21 ± 16.17 Δcm, NPH: 9.28 ± 5.26 Δcm at baseline, $p_{corr} = 0.001$), which significantly changed after respective interventions in PD patients (+6.34 ± 6.12 Δcm, $p_{corr} = 0.023$) but not in NPH patients (+1.99 ± 5.12 Δcm, $p_{corr} = 0.185$).

The gait domain rhythm showed group differences at baseline (Table 1). PD patients presented with shorter relative double limb support phase (36.67 ± 4.35 % gait cycle) and longer relative swing phase (31.66 ± 2.25 % gait cycle) than NPH patients (51.47 ± 11.38 % gait cycle and 24.21 ± 5.73 % gait cycle, Fig. 1D, both $p_{corr} = 0.009$). Interestingly, after respective interventions, NPH patients showed significant improvement (relative swing phase: +3.14 ± 4.76 % gait cycle, $p_{corr} = 0.037$; relative double limb support phase: 6.27 ± 9.62 % gait cycle, $p_{corr} = 0.037$), while PD patients showed no significant change (both $p_{corr} = 0.072$).

4. Discussion

The instrument-supported gait analysis (iGA) revealed four distinct results patterns relevant for differential diagnosis and assessment of treatment response in PD and NPH. Balance and static postural control showed no baseline group differences (i.e., before the disease-specific intervention) and no significant within-group intervention effects. Dynamic postural control differed between groups at baseline, however, did not change after the disease-specific interventions. Pace and force, representing gait dynamics, also revealed group differences at baseline and improved similarly in both groups after the respective interventions. In contrast, gait regularity, reflected by the gait domains variability and rhythm, showed baseline group differences and revealed differential changes after the disease-specific interventions.

The lack of between-group differences and within-group changes in balance parameters was consistent with previous studies [9]. Thus, balance tests have little value for differential diagnosis or evaluation of treatment response in PD and NPH patients.

In line with previous studies, the gait domains (dynamic) postural control, pace, force, variability, and rhythm demonstrated diagnostic relevance by differentiating PD and NPH patients at baseline [10]. While dynamic postural control confirmed previous reports of a broader gait width and increased mediolateral sway in patients with NPH compared to PD patients [11], this domain was not intervention-sensitive and, thus, less useful diagnostically. In contrast, the gait domains variability and rhythm not only distinguished the groups at baseline but also responded differentially to the interventions. Specifically, step length variability and relative swing/double limb support phases improved in NPH patients following intervention, but not in PD patients. In contrast, dopaminergic medication in PD patients was associated with increased gait width variability. A potential explanation may lie in the discrepancy of improved gait pace and force, whereas measures of dynamic postural control remained unchanged in PD patients. Thus, gait width variability might increase as enhanced forward propulsion occurred without a corresponding stabilization of mediolateral control. These findings suggest a potential imbalance between improved forward-directed gait dynamics and unchanged lateral control and should be further investigated in future studies.

Previous studies in PD and NPH primarily reported treatment effects in the gait domain pace, specifically in gait velocity and step length [10]. Our findings confirmed these effects and underline the clinical relevance of a $\geq$20 % gait velocity improvement after spinal tap in NPH [2]. However, changes in force, variability, and rhythm cannot be detected by standard clinical testing, such as the 10-m walk test. In contrast, iGA revealed differential improvements in these domains after spinal tap in NPH patients, but not after dopaminergic treatment in PD patients, highlighting its added diagnostic value.

This study has some limitations. The sample size was relatively small, but comparable to other studies examining differential gait patterns in PD and NPH [11,12]. Notably, iGA revealed significant differences between PD and NPH patients even at these group sizes. The current static balance test was presumably not demanding enough, since more challenging balance tasks, e.g. standing on a foam surface or in tandem stand, have already revealed differential impairments in NPH compared to PD patients [9]. However, intervention-induced effects on such demanding balance tasks remain unexplored.

In summary, iGA proved valuable for the differential gait assessment in NPH and PD and enabled a fine-grained evaluation of disease-specific treatments responses. The current in-depth gait analysis revealed that the gait domains variability and rhythm, reflecting gait regularity, were best suited to differentiate the gait disturbances caused by the two hypokinetic gait disorders.

CRediT authorship contribution statement

Carolin Semmler: Writing – review & editing, Writing – original draft, Visualization, Methodology, Investigation, Formal analysis, Conceptualization. **Veronika Wunderle:** Writing – review & editing, Investigation. **Taylan D. Kuzu:** Writing – review & editing, Investigation. **Oezguer A. Onur:** Writing – review & editing. **Christian Grefkes:** Writing – review & editing, Resources, Funding acquisition. **Gereon R. Fink:** Writing – review & editing, Resources, Funding acquisition. **Michael T. Barbe:** Writing – review & editing, Supervision, Conceptualization. **Peter H. Weiss:** Writing – review & editing, Supervision, Resources, Project administration, Methodology, Funding acquisition, Conceptualization.

Ethical approval and consent to participate

The data used in this study were acquired during the clinical care of the patients. This retrospective study was conducted under the Declaration of Helsinki and authorised by the local ethics committee (Cologne, study no.: 25-1151-retro).

Consent for publication

Not applicable.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Funding

The Deutsche Forschungsgemeinschaft (DFG, German Research Foundation) – Project-ID 431549029 – SFB 1451 funded this project. CG received additional funding from project GRK 2783. OO received consulting fees from Biogen and Lilly and lecture fees from Eisai, Boston Scientific, and Functional Neuromodulation. This work was also supported by a Marga and Walter Boll-Foundation grant to OO and GRF.

Declaration of competing interest

The authors have no conflicts of interest to declare.

Acknowledgements

Not applicable.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.parkreldis.2025.108081.

References

[1] S. Mermelstein, P. Barbosa, D. Kaski, Neurological gait assessment, practical neurology 24 (1) (2024) 11–21.
[2] C. Semmler, V. Wunderle, T.D. Kuzu, O.A. Onur, C. Grefkes, M.T. Barbe, G.R. Fink, P.H. Weiss, Instrument-supported gait analysis characterizes gait domain changes in patients with suspected normal pressure hydrocephalus, Neurol. Res. Pract 7 (1) (2025) 1–11.
[3] C. Jalles, D. Guerreiro, F. Pona-Ferreira, R.M. Simões, S. Reimão, J.J. Ferreira, Hypokinetic-rigid gait disorders with balance impairment-a walk through clinical and pathophysiological definitions, Park. Relat. Disord. (2025) 107339.
[4] A. Mirelman, P. Bonato, R. Camicioli, T.D. Ellis, N. Giladi, J.L. Hamilton, C.J. Hass, J.M. Hausdorff, E. Pelosin, Q.J. Almeida, Gait impairments in Parkinson's disease, Lancet Neurol. 18 (7) (2019) 697–708.
[5] M. Passaretti, A. Maranzano, B. Bluett, R. Rajalingam, A. Fasano, Gait analysis in idiopathic normal pressure hydrocephalus: a meta-analysis, Mov. Disord. Clin. Pract. 10 (11) (2023) 1574–1584.
[6] J. Scheffels, H. Kräling, E. Kalbe, J. Kessler, Conversions of cognitive screenings: mini-mental state examination vs. Montreal cognitive assessment vs. DemTect, Nervenarzt 89 (2018) 1371–1377.

[7] M.A. Williams, N.R. Relkin, Diagnosis and management of idiopathic normal-pressure hydrocephalus, Neurol. Clin. Pract. 3 (5) (2013) 375–385.
[8] S. Lord, B. Galna, J. Verghese, S. Coleman, D. Burn, L. Rochester, Independent domains of gait in older adults and associated motor and nonmotor attributes: validation of a factor analysis approach, J. Gerontol. A Biol. Sci. Med. Sci. 68 (7) (2013) 820–827.
[9] Y. Nikaido, T. Akisue, H. Urakami, Y. Kajimoto, K. Kuroda, Y. Kawami, H. Sato, Y. Ohta, T. Hinoshita, Y. Iwai, Postural control before and after cerebrospinal fluid shunt surgery in idiopathic normal pressure hydrocephalus, Clin. Neurol. Neurosurg. 172 (2018) 46–50.
[10] P. Bugalho, L. Alves, R. Miguel, Gait dysfunction in Parkinson's disease and normal pressure hydrocephalus: a comparative study, J. Neural Transm. 120 (2013) 1201–1207.
[11] Y. Nikaido, Y. Okada, H. Urakami, N. Ishida, T. Akisue, Y. Kawami, K. Kuroda, Y. Kajimoto, R. Saura, Dynamic stability during gait in idiopathic normal pressure hydrocephalus and Parkinson's disease, Acta Neurol. Scand. 145 (2) (2022) 215–222.
[12] G. Mostile, F. Contrafatto, R. Terranova, C. Terravecchia, A. Luca, M. Sinitò, G. Donzuso, C.E. Cicero, G. Sciacca, A. Nicoletti, Turning and sitting in early Parkinsonism: differences between idiopathic normal pressure hydrocephalus associated with Parkinsonism and Parkinson's disease, Mov. Disord. Clin. Pract. 10 (3) (2023) 466–471.