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# Is frontal gait a myth in normal pressure hydrocephalus?

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ABSTRACT

*Background:* Patients with idiopathic normal pressure hydrocephalus (iNPH) are considered to present a magnetic, slow, wide-based gait, also called frontal gait. However, this gait profile is not specific for iNPH and encountered in patients with other neurological conditions mimicking iNPH (i.e. iNPH mimics), such as vascular dementia. We aimed to characterize the gait profiles in iNPH and their mimics and to compare the prevalence of clinical gait abnormalities between both groups.

*Methods:* This retrospective study included 140 patients suspected of iNPH (76.3 ± 6.8 yo; 30.7% female). Eighty patients (57.1%) were diagnosed with iNPH according to the NPH consensus guidelines criteria; the remaining sixty patients were classified as mimics (23 neurodegenerative conditions, 12 multifactorial conditions, 9 vascular dementia, 7 mixed dementias, 6 toxic conditions, 2 psychiatric conditions, and 1 stroke). Two independent diagnosis-blinded clinicians (kappa, 0.73) evaluated gait according to four categories: frontal gait, parkinsonian gait, other clinical gait abnormalities, and normal gait.

*Results:* iNPH patients and mimics shared similar clinical characteristics. Frontal gait occurred in only 26% of patients (with a similar prevalence for the mimics). Parkinsonian gait was significantly more prevalent among the mimics (32% versus 15%; *p*-value: 0.032). This association between parkinsonian gait and mimics remained significant after adjusting for age, gender, comorbidities and white matter changes (OR: 2.404; 95% CI: [1.03–5.64]; *p* value: 0.044).

*Conclusion:* Frontal gait is not the most prevalent gait abnormality in iNPH and does not discriminate iNPH from its mimics. Parkinsonian gait is more prevalent among the mimics.

## 1. Introduction

Idiopathic normal pressure hydrocephalus (iNPH) is the most frequent cause of reversible dementia in aging, with a prevalence reaching 5.9% after 80 years [1]. Gait disorders in iNPH are classically described as slow, magnetic and wide-based, also known as frontal gait [2] or as higher-level gait disorder [3]. However, motor phenotypes of iNPH are very heterogeneous, in example 20–71% presenting with parkinsonism [4,5]. This heterogeneity makes challenging the diagnosis of iNPH, because neurodegenerative and/or vascular diseases mimic the clinical features of iNPH. Clinical gait abnormalities, especially frontal and parkinsonian gait, have already been compared between iNPH patients and specific conditions such as Parkinson's disease (PD) [5,6] or Alzheimer's disease [7]. However, clinical gait abnormalities have never been compared between iNPH patients and their mimics - patients with alternate neurological conditions mimicking the symptoms of iNPH (i.e. vascular dementia, Parkinson's disease or dementia with Lewy bodies), as encountered in daily clinical activities. Similar to other clinical characteristics, iNPH patients and mimics share similar quantitative gait characteristics, such as reduced walking speed and step height [8]. In order to improve early diagnosis and treatment of iNPH and its mimics, there is a need to extend our knowledge about the prevalence of clinical gait abnormalities between iNPH and mimics.

We conducted a retrospective study on video recording to characterize gait phenotypes between iNPH patients and mimics, while focusing on four major clinical gait phenotypes (frontal, parkinsonian, normal and other gait abnormalities), and to compare their respective prevalence. Based on clinical experience and on previous studies comparing clinical gait abnormalities between iNPH and specific conditions [5–7], we hypothesized that the prevalence of frontal gait will be

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similar between iNPH and its mimics. As the mimics present a high prevalence of parkinsonism [4], we hypothesized that parkinsonian gait would be more prevalent among the mimics in comparison to iNPH.

## 2. Methods

### 2.1. Participants

A total of 140 consecutive patients (76.3 ± 6.8 yo; 30.7% female) referred to the Department of Neurology of the Geneva University Hospitals between January 2008 and February 2017 for a suspicion of iNPH were included in this retrospective study. The suspicion of iNPH was based on the presence of gait and/or cognitive complaints with a ventricular enlargement at brain imaging (Evans ratio > 0.30). Inclusion criteria for this study were patients with suspicion of iNPH with a neurological examination (i), able to walk without assistance (ii) and a video recording of their gait (iii). Exclusion criteria were: the presence of an acute medical illness in the past three months and a diagnosis of secondary NPH. The diagnosis of iNPH was assigned following the international NPH consensus guidelines criteria [9]. A CSF tap test was performed in every patient; the CSF tap test consisted on a lumbar puncture of 40 ml of CSF through a 20-gauge spinal needle. Following comprehensive neurological and neuropsychological assessments, a diagnosis (iNPH versus mimics) was assigned after reviewing all available clinical data, as well as brain imaging and blood/CSF laboratory results at a consensus case conference involving neurologists and neuropsychologists blinded for the clinical gait abnormalities, as previously reported [10]. Eighty patients (57.1%) fulfilled the international NPH consensus guidelines criteria [9]; and the remaining sixty patients were classified as mimics: 23 presented a neurodegenerative condition, 12 multifactorial conditions, 9 a vascular dementia, 7 a mixed dementia, 6 a toxic condition, 2 a psychiatric condition and 1 a stroke (Fig. 1). The study protocol was approved by the Institutional Review Board of the Geneva University Hospital (Protocol 09-160R).

### 2.2. Clinical gait characteristics

The video recording of the gait was standardized, as previously described [10]. Briefly, patients were asked to walk at their self-selected speed on a 10-m walkway, while their gait was video recorded with a 12-camera motion system (Vicon Mx3+, Oxford Metrics, UK). Two independent raters blind for the diagnosis (iNPH versus mimics) evaluated the video recordings to clinically classify gait phenotypes as frontal, parkinsonian, other, or normal, as previously reported [11]. Frontal gait was characterized by short steps, a wide base of support and a magnetic component (reduced step height); parkinsonian gait by short and/or shuffling steps, flexed posture, reduced arm swing and normal base [11]. Patients without any clinical gait abnormalities were defined as normal, while those with any other clinical gait abnormalities were defined as other (i.e., hemiparetic or ataxic gait). It must be emphasized that frontal and parkinsonian gaits share overlapping characteristics such as small steps and slow gait speed; step width and arm swing are the main distinctive features.

### 2.3. Covariates

The Global Health Status Score (GHS; 0–10) summarizes the presence of the following comorbidities: diabetes, chronic heart failure, arthritis, hypertension, depression, stroke, PD, chronic obstructive pulmonary disease, angina, and myocardial infarction [12]. The vascular risk factor score (range 0–5) recorded the presence of diabetes, hypertension, hypercholesterolemia, body mass index > 30 and

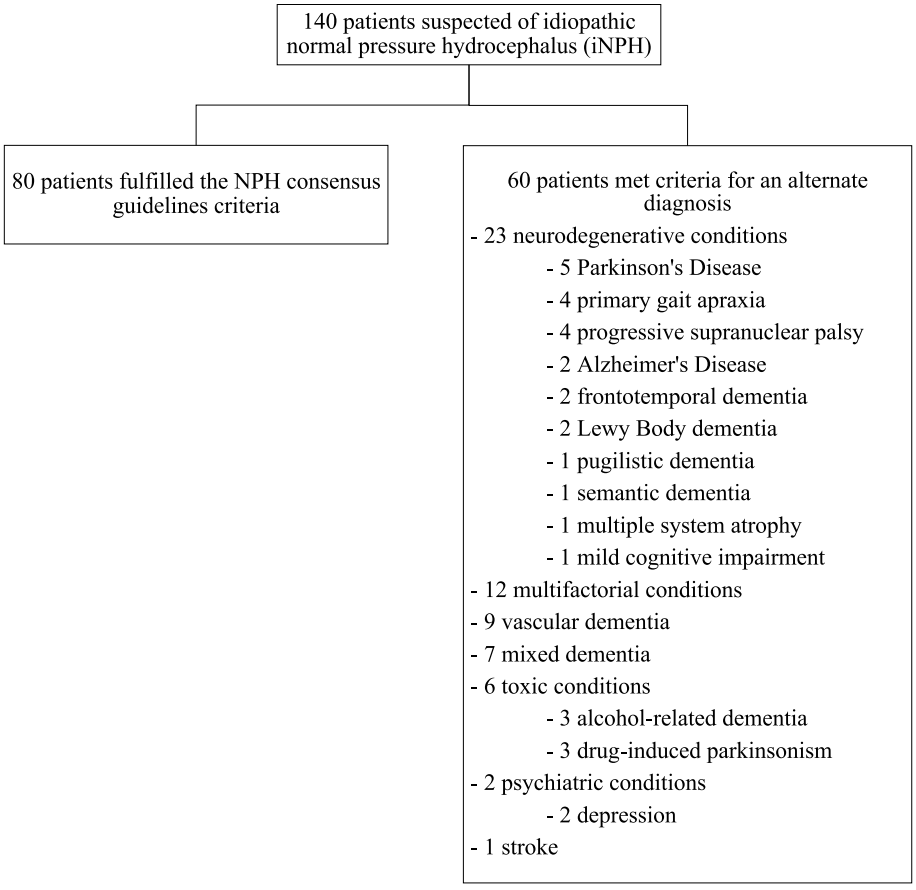


**Fig. 1.** Study profile.

smoking; the cardiovascular risk factor score (range 0–4) the presence of myocardial infarction, angina, arrhythmia and chronic heart failure. NPH symptoms in gait, cognition, and miction were quantified with the NPH grading scale [13]. Age-related white matter changes (ARWMC; range 0–30) were used to quantify the burden of white matter disease in all the brain and its subregions [14].

### 2.4. Statistics

Descriptive statistics compared iNPH and mimics patients with *t*-test, $\chi^2$, or Mann-Whitney as appropriate. Univariable and multivariable (adjusted on age, gender, comorbidities, and white matter changes) logistic regression models were used to assess the association between the diagnosis (iNPH vs. mimics; dependent variable) and each clinical gait characteristic (independent variables). Finally, we conducted sensitivity and specificity analyses of the frontal gait for diagnostic group (i.e., iNPH versus mimics). All statistical analyses were performed with SPSS Version 23 (SPSS Inc., Chicago, IL, USA).

## 3. Results

Baseline characteristics were similar between iNPH and mimics (Table 1). The inter-rater reliability between both assessors (EM and GA) was substantial (kappa, 0.73) [15]. Frontal gait was not the most prevalent gait abnormality in patients with suspicion of iNPH (only 25.7% for both groups; Fig. 2). Both groups shared similar prevalence of frontal gait (*p*-value: 1.000) and normal gait (p-value: 0.324); while parkinsonian gait was less prevalent in the iNPH group than in the mimics group (15% vs. 32%; p-value: 0.032; Fig. 2). This association between parkinsonian gait and the group of mimics remained significant after adjusting on age, gender, comorbidities and white matter changes (OR: 2.404; 95% CI: [1.03–5.64]; *p* value: 0.044) (Table 2).

**Table 1**
Clinical characteristics of participants ($n$ = 140).

| | iNPH ($n$ = 80) | | | Mimics ($n$ = 60) | | | p-Value |
|---|---|---|---|---|---|---|---|
| Age, years | 75.96 | ± | 6.26 | 76.80 | ± | 7.41 | 0.451 |
| Gender, n (% female) | 25 | | (31.25) | 18 | | (30) | 1.000 |
| Disease duration, months | 38.15 | ± | 29.34 | 42.37 | ± | 47.99 | 0.777 |
| Treatment, n | 3.95 | ± | 2.31 | 4.88 | ± | 3.04 | 0.097 |
| Comorbidities (GHS, 0–10) | 1.86 | ± | 1.04 | 2.23 | ± | 1.23 | 0.058 |
| Risk factors | | | | | | | |
| Vascular (0–5)[a] | 1.36 | ± | 0.90 | 1.42 | ± | 1.08 | 0.694 |
| Cardiovascular (0–4)[b] | 0.21 | ± | 0.41 | 0.20 | ± | 0.44 | 0.709 |
| NPH grading scale[c] | | | | | | | |
| Gait (0–4) | 1.95 | ± | 0.39 | 1.95 | ± | 0.47 | 0.960 |
| Cognition (0–4) | 2.13 | ± | 0.51 | 2.15 | ± | 0.76 | 0.319 |
| Urinary (0–4) | 1.22 | ± | 1.07 | 1.12 | ± | 1.06 | 0.577 |
| Gait speed, m/s | 0.70 | ± | 0.25 | 0.66 | ± | 0.30 | 0.365 |
| White Matter Changes[d] | | | | | | | |
| Frontal (0–6) | 2.41 | ± | 1.56 | 2.43 | ± | 1.65 | 0.796 |
| Parieto-occipital (0–6) | 2.15 | ± | 1.89 | 2.03 | ± | 1.92 | 0.704 |
| Temporal (0–6) | 0.60 | ± | 1.22 | 0.47 | ± | 0.96 | 0.785 |
| Basal Ganglia (0–6) | 0.55 | ± | 0.86 | 0.45 | ± | 0.84 | 0.472 |
| Infratentorial (0–6) | 0.22 | ± | 0.57 | 0.26 | ± | 0.59 | 0.382 |
| Total Score (0 − 30) | 5.94 | ± | 4.70 | 5.64 | ± | 4.34 | 0.823 |

*Note.* Results are presented in mean ± standard deviation; iNPH: idiopathic normal pressure hydrocephalus; GHS: Global Health Status Score, summarizes the presence of diabetes, chronic heart failure, arthritis, hypertension, depression, stroke, Parkinson's disease, chronic obstructive pulmonary disease, angina and myocardial infarction.

[a] Diabetes, hypertension, hypercholesterolemia, body mass index > 30 and smoking.

[b] Myocardial infarction, angina, arrhythmia and chronic heart failure.

[c] NPH symptoms in gait, cognition and miction.

[d] Quantifies the burden of white matter disease in the all brain and its subregions.

Sensitivity of frontal gait for iNPH diagnosis was only 30% and specificity 75%; while positive predictive value was 61.5% and negative predictive value 44.6%.

## 4. Discussion

This study aimed to describe the clinical gait phenotypes of iNPH and mimics and to compare their respective prevalence. Corresponding to our initial hypotheses, we found that frontal gait was not the most prevalent gait abnormality among iNPH patients (hypothesis 1); and we found that parkinsonian gait was more prevalent among the mimics (hypothesis 2).

Frontal gait was not the most prevalent gait abnormality among patients with suspicion of iNPH. In contrast with previous results, the prevalence was similar between iNPH and mimics patients [9]. A reason for this discrepancy is that our study combined several neurological diseases in the mimics group and not only patients with PD like in another study [6]. Frontal gait is a non-specific gait abnormality that is found in various neurological conditions affecting the fronto-subcortical loops, such as in iNPH [16] or in other vascular or neurodegenerative conditions found in the group of mimics. Furthermore, the low sensitivity and positive predictive values of frontal gait illustrate that this gait phenotype does not present a reliable parameter to identify iNPH from the mimics.

The prevalence of parkinsonian gait was increased among the mimics in comparison to iNPH patients. This is consistent with a previous study, reporting an increased proportion of parkinsonism in the mimics in comparison to iNPH (40.3% vs. 20.3%; *p*-value: 0.015) [4]. In another study, PD patients are presenting more severe parkinsonian features than iNPH patients [6]. Although the presence of parkinsonian gait and parkinsonism in general have been both reported in patients with iNPH [5,17,18], the presence of parkinsonian gait in patients with suspected iNPH should be considered as a cue pointing to an alternate condition.

A minority of iNPH and mimics presented a normal clinical gait evaluation with a similar prevalence between both groups (respectively, 29% and 20%; *p*-value: 0.324). The presence of clinical gait abnormalities was not an inclusion criterion for the current study. The patients may complain about walking difficulties, but with an intact clinical gait evaluation.

Comparing clinical gait characteristics among this important number of iNPH and mimics patients represents the main strength of this study, as it reflects the daily clinical activity. Furthermore, it must be emphasized that the clinical gait examination can be easily performed during the standard neurological assessment and better fits to the daily reality of clinicians than a spatiotemporal gait analysis requiring a time-consuming assessment and expansive equipment. The generalization of the study findings should be limited to patients with suspicion of iNPH, who are still able to walk. We deliberately selected patients at a very early stage (even without clinical gait abnormalities, but with a complaint of walking difficulty) in order to improve the early management of these conditions. However, this approach may have increased the prevalence of the mimics, by lowering the threshold for NPH ascertainment. A major limitation is the absence of data concerning the proportion of patients who underwent shunting: around two-thirds of the patients diagnosed with iNPH at baseline tend to be diagnosed with an alternate condition in the three following years [19]. Although we apply the international NPH consensus guidelines criteria for diagnosing iNPH [9], we cannot exclude the presence of mimics in the group of iNPH patients. The absence of histopathological confirmation of the diagnoses of iNPH and mimics, or long-term follow-up after shunt surgery represent a main limitation. Finally, although the number of drugs did not differ between both groups, the lack of information about the use of psychoactive drugs that may affect gait represents a limitation and should be taken into account in future studies.

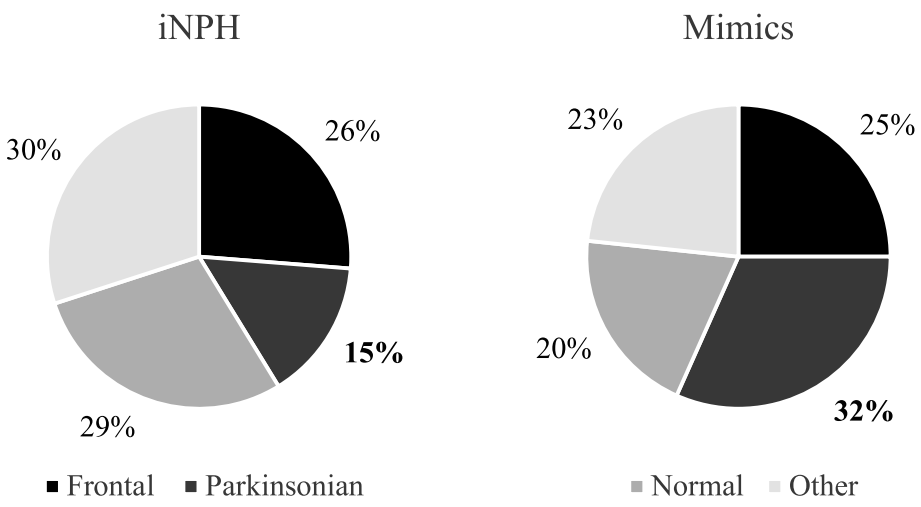


**Fig. 2.** Prevalence of clinical gait characteristics among iNPH and mimics.
The prevalence of frontal gait is similar between iNPH patients and mimics (*p*-value: 1.000) and concerns 26% of the patients. The majority of iNPH patients presents either a normal walk or other clinical gait abnormalities, while parkinsonian gait is the most prevalent gait type among mimics. Among the four types of gait, only the parkinsonian gait was significantly different (*p* value: 0.032).

**Table 2**
Univariable and multivariable (adjusted for age, gender, comorbidities and white matter changes[a]) logistic regressions showing an association between diagnosis (mimics versus iNPH; dependent variable) and clinical gait characteristics (independent variable).

| | Univariable | | | Multivariable | | |
|---|---|---|---|---|---|---|
| | O.R. | (95% CI) | p-Value | O.R. | (95% CI) | p-Value |
| Frontal | 0.937 | (0.44–2.02) | 0.867 | 0.830 | (0.37–1.85) | 0.649 |
| Parkinsonian | 2.626 | (1.16–5.96) | 0.021* | 2.404 | (1.03-5.64) | 0.044* |
| Normal | 0.620 | (0.28–1.37) | 0.239 | 0.753 | (0.33–1.73) | 0.504 |
| Other | 0.710 | (0.33–1.53) | 0.381 | 0.714 | (0.32–1.60) | 0.412 |

*Note.* O.R.: odds ratio; (95% CI): confidence interval.
[a] White matter changes are measured by age-related white matter changes (total score).
* $p < 0.05$.

## 5. Conclusion

This study deconstructed the classical description of the frontal gait in iNPH, as it is present in only 26% of iNPH patients at early stage of the disease. Furthermore, we demonstrated that the presence of parkinsonian gait in patients with a suspicion of iNPH could represent a cue for an alternate diagnosis. In addition to neuroimaging and CSF biomarkers [20,21], these findings suggest that a clinical approach based on gait phenotypes may also contribute to correctly identify iNPH from its mimics. Future studies focusing on the outcome of the shunt placement should determine the prognostic value of clinical gait phenotypes, especially frontal and parkinsonian gait, among patients with iNPH.

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## Conflict of interests

The authors declare that they have no conflict of interest.

## Acknowledgements

None.

## Ethical approval

The study protocol was approved by the Institutional Review Board of the Geneva University Hospital (Protocol 09-160R).

## Informed consent

Informed consent was obtained from all participants.

## Author roles

Research Project: Conception/Organization/Execution: Morel, Allali.
Statistical analysis:

A) Design: Allali
B) Execution: Morel
C) Review and Critique: Allali

Manuscript preparation:

A) Writing of the first draft: Morel
B) Review and Critique: Armand, Assal, Allali

All authors approved the final article.

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