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RESEARCH ARTICLE

Apathy in idiopathic normal pressure hydrocephalus: A marker of reversible gait disorders

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Objective: Apathy—the most common behavioral disturbance in idiopathic normal pressure hydrocephalus (iNPH)—is associated with poor gait, but the role of apathy on gait improvement after cerebrospinal fluid (CSF) tapping has not been studied yet. This study aims to compare gait improvement after CSF tapping in iNPH patients with and without apathy.

Methods: Stride time variability (STV), a marker of higher level of gait control, was measured in 33 iNPH patients (78.4 ± 5.7 years; 36.4% women) with an optoelectronic system during usual walking (single task) and during walking while dual tasking of counting and verbal fluency before and 24 hours after CSF tapping. Apathy was defined by a score ≥14 on the Starkstein apathy scale.

Results: Apathy was present in 60.6% of patients. Cerebrospinal fluid tapping led to greater improvement of STV (ie, decrease) during dual-task walking (and more specifically categorical verbal fluency) in apathetic compared to nonapathetic patients (−44.7 ± 58.1% versus +4.24 ± 67.6%, respectively; P = .040), even after adjusting for age and depressive symptoms. More severe apathy was correlated with better STV improvement while dual tasking (categorical verbal fluency) after CSF tapping (r = −0.412; P-value = 0.021), while it was not correlated with improvement on executive tests.

Conclusions: Our findings suggest that the presence of apathy is a predictor of better outcomes of gait disorders after CSF tapping in patients with iNPH.

KEYWORDS

apathy, dual tasking, executive functions, gait disorders, normal pressure hydrocephalus

1 | INTRODUCTION

Idiopathic normal pressure hydrocephalus (iNPH)—a neurological condition associating gait disorders, cognitive impairment, and urinary incontinence with ventricular enlargement at brain imaging—is a common condition in aging affecting around 6% of adults older than 80.[1] Apathy represents the most common behavioral disturbance in iNPH with a prevalence around 60%.[2,3] Apathy is defined by a state of decreased motivation affecting goal-directed behaviors and characterized by reduced interests or emotions independent of diminished level of consciousness, impaired cognition, or emotional disturbance.[4] Similar to cognition and gait, shunt surgery also improves apathy in iNPH.[2] The postsurgical reduction of apathy has been associated with cognitive improvement.[5]

The relationship between gait and apathy has recently been demonstrated in patients with suspected iNPH.[6] Deterioration of higher level of gait control measured by stride time variability (STV) during dual tasking correlated with severity of apathy, independently from the presence of executive deficits. Stride time variability that reflects the higher level of gait control[7] has been associated with executive functioning[8,9] that is specifically disturbed in iNPH[10]: increased STV (ie, worse gait control) being associated with poor executive function. However, the presence of apathy as a predictor of gait modification after cerebrospinal fluid (CSF) tapping has been never studied in iNPH patients.

Therefore, we conducted a prospective study in patients with iNPH, where we compared gait and cognitive changes before and 24 hours after CSF tapping in patients with and without apathy. Because increased STV during dual tasking has been associated with apathy[6] and that apathy is improved after CSF tapping,[2] we hypothesize that iNPH patients with apathy would have a better STV improvement than those without apathy after CSF tapping.

2 | METHODS

2.1 | Participants

All iNPH patients consecutively assessed at the Department of Neurology of the Geneva University Hospitals between October 2012 and November 2016 were included in this study. The study procedures were previously described in detail.[11] In summary, inclusion criteria for this analysis were patients with (i) a diagnosis of possible or probable iNPH that fulfilled the iNPH consensus guideline criteria[12] with (ii) a neurological examination, (iii) a comprehensive neuropsychological assessment and a spatio-temporal gait analysis performed before and 24 hours after CSF tapping, and (iv) a sufficient knowledge of French to perform the neuropsychological assessment. Exclusion criteria were presence of an acute medical illness in the past 3 months, a CSF tap test in the previous 2 months, a change in treatment between both evaluations, and a diagnosis of secondary NPH. One patient was excluded, because he could not speak French. After exclusion, a total of 33 patients with suspicion of iNPH were included in this study (78.4 ± 5.7 years; 36.4% women). Twelve patients accepted the shunt procedure (36.4%); among them, 11 patients (91.7%) improved their gait after the shunt. Cerebrospinal fluid tapping consisted on a lumbar puncture of 40 mL of CSF through a 20-gauge spinal needle. Comorbidities were documented by the global health status score (range: 0-10), based on the presence of diabetes, chronic heart failure, arthritis, hypertension, depression, stroke, Parkinson's disease, chronic obstructive pulmonary disease, angina, and myocardial infarction.[13] A vascular risk factor score (range: 0-5) was computed on the presence of diabetes, hypertension, hypercholesterolemia, body mass index >30 or smoking, and a cardiovascular risk factor score (range: 0-4) on the presence of myocardial infarction, angina, arrhythmia, or chronic heart failure.[14] White matter lesions were rated with a validated semiquantitative scale that demonstrated a moderate to good interrater reliability[15] on cerebral imaging (29 magnetic resonance imaging and 4 computed tomography) performed at the time of the clinical assessment. Total score (all brain; range: 0-30) and subscores (range: 0-6) were computed on the 5 regions combining the left and right hemispheres: frontal, temporal, parieto-occipital, basal ganglia, and infratentorial. This study protocol was approved by the ethical committee of Geneva University Hospitals.

2.2 | Apathy and neuropsychological assessment

A quantified measure of apathy was systematically assessed before CSF tapping with the Starkstein apathy scale (SAS)[16] that showed good psychometric properties in neurological patients.[17] Presence of apathy was defined by a SAS score ≥14, as recommended.[16] Depressive symptoms and anxiety were quantified with the Hospital Anxiety and Depression Scale (HADS) before CSF tapping.[18] A standardized neuropsychological assessment was conducted before and after CSF tapping.[19] The pre-CSF tapping assessment included an evaluation of executive functions (Color Trails Test,[20] Stroop test,[21] phonemic and categorical verbal fluencies,[22] attention [Wechsler Adult Intelligence Scale – III symbol digit test and digit span[23] and Wechsler Memory Scale – III spatial span[24]], and memory (the French version of the Free and Cued Selective Reminding Test[25]), while the post-CSF tapping assessment (conducted 24 hours after CSF tap test) focused on executive function and attention. Global cognitive functioning was assessed with the Mini-Mental State Examination.[26] We included a comparison of categorical and phonemic verbal fluencies before and after CSF tap test, because both tests have been considered as the most sensitive to change after CSF tapping in iNPH patients[19]; the difference between the pre-CSF and the post-CSF tapping was based on a delta value (in %): $100 \times (\text{Score}_{\text{postCSF tapping}} - \text{Score}_{\text{preCSFtapping}})/([\text{Score}_{\text{preCSF tapping}} + \text{Score}_{\text{postCSF tapping}}]/2)$.

Key points

- Apathy is frequent in patients with normal pressure hydrocephalus.
- Normal pressure hydrocephalus patients with apathy present better gait improvement after CSF tapping than those without apathy.
- Apathetic patients show a better gait improvement than nonapathetic patients in the condition of dual tasking.

2.3 | Gait evaluation and dual tasks

A quantitative spatio-temporal gait evaluation at comfortable walking speed was performed in ecological condition, while patients wearing their own shoes, as previously described.[27] The quantitative gait assessment included 5 individual randomized conditions: usual walking and walking while performing 4 different dual tasks: forward counting from 1, backward counting from 50, phonemic verbal fluency (enumerating words starting by letter p), and categorical verbal fluency (generating animal names). No prioritization was given for the walking or the cognitive task; the instruction of the dual tasks was to walk and to perform the cognitive task at the best of their capacity. Our main outcome—STV—was computed on the measurement of the heel marker trajectories on a distance of 8 m with an optoelectronic system including 12 cameras (VICON Mx3+; ViconPeak, Oxford, UK). Stride time variability (in %) was calculated following the formula: 100 × (standard deviation of stride time/mean value of stride time). The difference of STV between the pre-CSF and the post-CSF tapping was based on a delta value (in %): $100 \times (\text{STV}_{\text{postCSF tapping}} - \text{STV}_{\text{preCSF tapping}})/([\text{STV}_{\text{postCSF tapping}} + \text{STV}_{\text{preCSF tapping}}]/2)$.

2.4 | Statistics

Descriptive statistics of the iNPH patients with and without apathy were calculated. Data were represented graphically; model assumptions were tested with skewness and kurtosis. Baseline characteristics (before CSF tapping) were compared with sample *t*-test, Mann-Whitney *U*-test, or χ^2 as appropriate. We compared gait changes after CSF tapping (delta of STV) between those with (SAS ≥ 14) and those without apathy (SAS < 14) with sample *t*-test; we also

included an intragroup comparison—respectively in iNPH patients with and without apathy—between pre-CSF and post-CSF tapping based on the Wilcoxon signed-rank test or paired *t*-test as appropriate. Multivariable linear regression adjusted for age and depressive symptoms (HADS)—both variables have been linked with gait performances and/or apathy[28-32]—was performed to assess the association between baseline SAS (independent variable) and STV changes while categorical verbal fluency (dependent variable). Finally, to compare the correlation between changes in higher level of gait control and apathy, and changes in executive function and apathy, we performed a correlation (Pearson) between SAS and changes in STV while performing the cognitive task of categorical verbal fluency (ie, walking while generating animal names), as well between SAS and change in executive functions (categorical and phonemic verbal fluency). All analyses were conducted by using SPSS version 22 (SPSS Inc., Chicago, Ill., USA).

3 | RESULTS

Characteristics of iNPH patients with and without apathy are compared in Table 1. Both groups presented similar clinical characteristics, gait parameters, and white matter lesions, except for apathy (SAS) and depressive symptoms (HADS). The prevalence of apathy was 60.6%, and the intensity was mild (15.2 ± 4.1 for the entire group, range[1-19]).

Baseline cognitive performance was similar between patients with and without apathy (Table 2). Patients had mild global cognitive impairment with a mean value of MMSE of 24.2 (SD = 3.6) for the entire group.

Gait was more improved after CSF tapping in patients with apathy than those without apathy: patients with apathy showed better improvement in STV while enumerating animal names after CSF tapping, reducing significantly their STV of 44.7% (SD = 58.1) in comparison to patients without apathy (*P*-value = .040); Cohen's effect size value (*d* = 0.78) suggested a moderate to high impact (Table 3). The association between changes in STV while enumerating animal names after CSF tapping and presence of apathy remains significant after adjusting for age and HADS-depression score (β: −63.0, 95% CI: [−118.4; −7.6], *P*-value = .027). Gait speed was also more improved after CSF tapping in patients with apathy than those without apathy; however, we did not find any significant improvement for (mean values of) stride length and stride time, between patients with and without apathy (see tables in the Supporting Information).

TABLE 1 Clinical characteristics of patients with normal pressure hydrocephalus (*n* = 33)

	Apathy (n = 20)	No Apathy (n = 13)	*P*-value[a]
Age (years)	77.6 ± 5.4	79.7 ± 6.24	.313
Gender (%female)	35.0	38.5	.840
Disease duration (months)	32.5 ± 19.4	31.4 ± 23.1	.785
Comorbidities (GHS, 0-10)	1.85 ± 1.13	1.77 ± 0.73	.758
Vascular risk factor[b] (0-5)	1.60 ± 0.88	1.46 ± 0.78	.650
Cardiovascular risk factor[c] (0-4)	0.25 ± 0.44	0.31 ± 0.48	.785
Number of treatments, n	4.35 ± 2.47	3.54 ± 2.37	.357
Starkstein apathy scale (0-42)	17.8 ± 2.6	11.3 ± 2.5	**<.001**
HADS-depression (0-21)	7.70 ± 3.47	4.58 ± 2.78	**.013**
HADS-anxiety (0-21)	7.40 ± 3.42	5.17 ± 2.86	.068
Mini-Mental State Examination (/30)	24.4 ± 3.7	23.8 ± 3.7	.589
Gait speed (cm/s)			
Single task	69.6 ± 23.7	71.1 ± 20.5	.859
Dual task of forward counting	68.3 ± 23.0	61.3 ± 22.1	.417
Dual task of backward counting	60.2 ± 20.7	58.9 ± 20.8	.873
Dual task of categorical verbal fluency	48.4 ± 16.7	54.9 ± 19.4	.335
Dual task of phonemic verbal fluency	45.8 ± 16.3	50.6 ± 17.1	.447
White matter lesions[d]			
Total (0-30)	8.15 ± 5.01	6.33 ± 5.68	.352
Frontal (0-6)	3.20 ± 1.54	2.17 ± 1.53	.107
Temporal (0-6)	1.20 ± 1.64	0.92 ± 1.00	.954
Parieto-occipital (0-6)	2.90 ± 2.02	2.00 ± 1.91	.272
Basal ganglia (0-6)	0.65 ± 0.88	0.67 ± 0.98	.954
Infratentorial (0-6)	0.20 ± 0.62	0.58 ± 0.79	.182

Abbreviations: GHS: global health status score; HADS: Hospital Anxiety and Depression Scale.

[a]Comparisons are based on Mann-Whitney or Chi^2 as appropriate; significant differences (*P* values < 0.05) are in bold.

[b]Presence of diabetes, hypertension, hypercholesterolemia, body mass index >30 or smoking.

[c]Presence of myocardial infarction, angina, arrhythmia or chronic heart failure.

[d]Rated with the age-related white matter changes.

TABLE 2 Comparisons of cognitive performances between patients with normal pressure hydrocephalus with and without apathy (n = 33)

	Apathy (n = 20)	No Apathy (n = 13)	*P*-value[a]
Executive functions			
Color Trails Test, s			
Part 1	117.9 ± 66.7	131.1 ± 75.0	.605
Part 2	223.3 ± 84.5	226.1 ± 70.8	.865
Index[b]	1.54 ± 1.02	1.30 ± 0.63	.737
Verbal fluency, n			
Semantic	11.8 ± 4.8	13.9 ± 5.7	.275
Phonemic	12.1 ± 4.7	11.0 ± 5.5	.552
Stroop test, s			
Dot	18.3 ± 4.5	18.6 ± 5.9	.587
Word	28.3 ± 13.3	24.2 ± 5.8	.879
Color	48.5 ± 19.6	40.8 ± 17.7	.183
Index[c]	2.60 ± 0.63	2.26 ± 0.73	.503
Memory			
FCSRT free recall	18.1 ± 8.2	14.8 ± 5.9	.234
FCSRT total recall (free and cued recall)	38.7 ± 9.7	36.8 ± 8.4	.415
Attention			
WAIS-III symbol digit modalities test, n	30.7 ± 11.2	29.4 ± 9.5	.730
WAIS-III forward digit span, n	5.05 ± 0.94	5.23 ± 1.30	.758
WAIS-III backward digit span, n	3.75 ± 0.85	3.54 ± 1.05	.353
WMS -III forward visual span, n	4.32 ± 1.16	4.58 ± 1.00	.509
WMS -III backward visual span, n	3.68 ± 0.89	3.67 ± 1.07	.921

Abbreviations: iNPH: idiopathic normal pressure hydrocephalus; FCSRT: Free and Cued Selective Recall Reminding Test; WAIS-III: Wechsler Adult Intelligence Scale-III; WMS-III, Wechsler Memory Scale-III.

Values are presented with means (standard deviation).

Dot, name color of dot; Word, name color print of noncolored word; Color, name color print of colored word.

[a]Comparisons based on Mann-Whitney test.

[b]Color Trails Test index is calculated with the formula: (Part 2-Part 1/Part 1).

[c]Stroop index is calculated with the formula: (color part/dot part).

Figure 1 showed that when considering the entire group, iNPH patients improved their STV while enumerating animal names after CSF tapping. When considering individually patients with and without apathy, only patients with apathy improved their STV while enumerating animal names after CSF tapping with a $STV_{pre\text{-}CSF\ tapping}$ decreasing from 13.7% (SD = 18.7) to 6.19% (SD = 3.99); patients without apathy did not show any changes. When considering apathy as a continuous scale, the SAS were still associated with STV improvement while enumerating animal names after CSF tapping even after adjusting for age and HADS-depression score (β: −8.04, 95% CI: [−14.3; −1.8], *P*-value = .014).

The presence of apathy did not affect executive improvement after CSF tapping, as measured by neuropsychological tests: patients with and without apathy had similar changes after CSF tapping for the categorical verbal fluency (+11.9 ± 28.3% versus +9.31 ± 25.3%, respectively; *P*-value = .800) and for the phonemic verbal fluency (+3.68 ± 46.0% versus +15.2 ± 41.6%, respectively; *P*-value = .483). We reported in Figure 2a a nonsignificant correlation between SAS and changes in categorical verbal fluency (r = 0.076; *P*-value = .685), and in Figure 2b, a nonsignificant correlation between SAS and changes in phonemic verbal fluency (r = 0.108; *P*-value = .556), while we reported in Figure 2c a significant correlation between SAS and changes in STV while categorical verbal fluency (r = −0.412; *P*-value = .021).

4 | DISCUSSION

We tested the hypothesis that presence of apathy in iNPH patients may be considered as a predictor of improvement of higher level gait disorder after CSF tapping. Stride time variability while dual tasking (walking while enumerating animal names) showed greater improvement after CSF tapping in iNPH patients with apathy than in those without. This improvement was independent of depressive symptoms. Patients with and without apathy presented with similar baseline executive impairments. The presence of apathy was correlated with STV improvement during a dual task of walking and enumerating animal names after CSF tapping, whereas it was not correlated with improvement in executive function.

Idiopathic normal pressure hydrocephalus patients with apathy had a greater improvement of their higher level of gait control after CSF tapping than patients without apathy; the severity of apathy

TABLE 3 Gait parameters (stride time variability) and gait changes (delta of stride time variability) before and after CSF tapping in patients with definite normal pressure hydrocephalus (n = 33)

	Apathy (n = 20)		No Apathy (n = 13)		*P*-value[a]
	Pre-CSF tapping	Post-CSF tapping	Pre-CSF tapping	Post-CSF tapping	
Single task					
Stride time variability (%)	4.39 ± 1.96	2.83 ± 1.73**	3.45 ± 1.36	2.63 ± 1.21*	
Changes (%)	−51.8 ± 51.7		−31.1 ± 53.0		.286
Dual task					
Forward counting					
Stride time variability (%)	3.42 ± 1.40	3.32 ± 1.88	4.54 ± 2.20	3.45 ± 1.78	
Changes (%)	−9.50 ± 54.1		−30.3 ± 52.4		.300
Backward counting					
Stride time variability (%)	6.45 ± 3.42	6.30 ± 3.43	10.4 ± 23.6	12.1 ± 19.5	
Changes (%)	−6.72 ± 61.1		25.4 ± 55.0		.162
Categorical fluency					
Stride time variability (%)	13.7 ± 18.7	6.19 ± 3.99**	7.94 ± 6.19	9.12 ± 9.09	
Changes (%)	−44.7 ± 58.1		4.24 ± 67.6		**.040**
Phonemic fluency					
Stride time variability (%)	11.0 ± 8.6	10.3 ± 15.8	11.7 ± 10.4	14.8 ± 19.4	
Changes (%)	−22.5 ± 66.3		−0.57 ± 69.1		.397

Intragroup comparison are based on paired *t*-test or Wilcoxon signed-rank test (as appropriate) between stride time variability pre-CSF and post-CSF tapping.

Changes between pre-CSF and post-CSF tapping are calculated following the formula (in %): $100 \times (STV_{postCSF\ tapping} - STV_{preCSF\ tapping})/([STV_{preCSF\ tapping} + STV_{postCSF\ tapping}]/2)$.

[a]Comparisons are based on Mann-Whitney on changes in stride variability between apathetic and nonapathetic patients; significant differences (*P*-values < 0.05) are in bold.

*$P < .05$.

**$P < .01$.

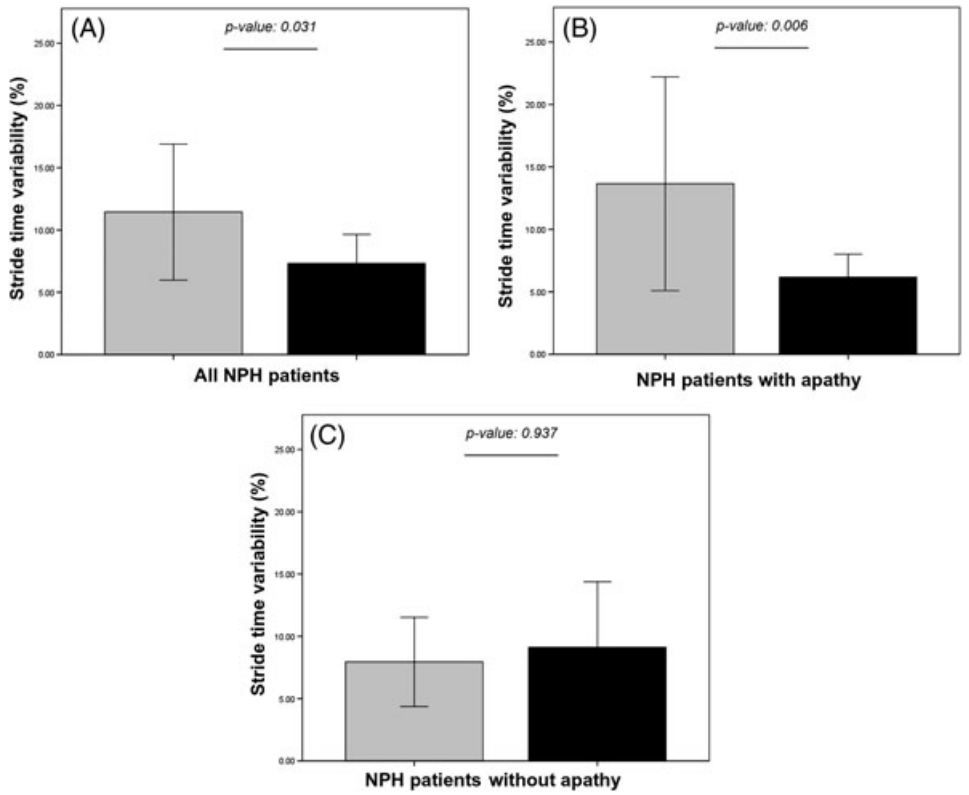

FIGURE 1 Comparison of stride time variability while categorical verbal fluency (in %) between pre-CSF (grey) and post-CSF (black) tapping in all iNPH patients A,; in patients with B, and without C, apathy

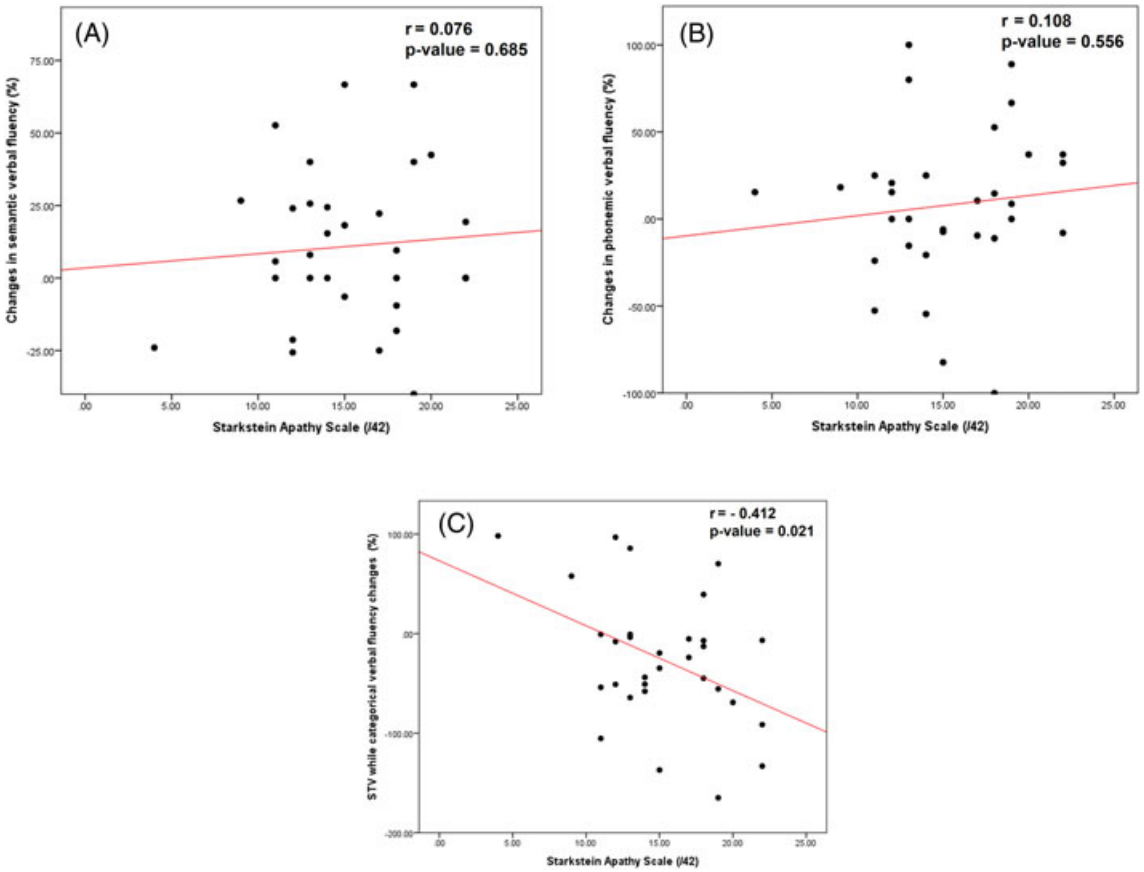

FIGURE 2 A, Correlation between changes in categorical verbal fluency and Starkstein apathy scale (r = 0.076; P-value = .685) showing no significant correlation between improvement of categorical verbal fluency after CSF tap test and Starkstein apathy scale. B, Correlation between changes in phonemic verbal fluency and Starkstein apathy scale (r = 0.108; P-value = .556) showing no significant correlation between improvement of phonemic verbal fluency after CSF tap test and Starkstein apathy scale. C, Correlation between changes in stride time variability (STV) while categorical verbal fluency (animal names) and Starkstein apathy scale (r = −0.412; P-value = .021) show that patients with the highest score at the Starkstein apathy scale (ie, increase level of apathy) had a better improvement of their STV (is, higher decrease in STV) after CSF tap test than patients with lower level of apathy [Colour figure can be viewed at wileyonlinelibrary.com]

was positively associated with the amplitude of gait improvement. The high prevalence of apathy (60.6%) is similar to those reported in previous studies in iNPH.[2,3,6] Apathy reflects a structural or a functional disruption of the corticosubcortical loops and differs from depression,[4,33] as confirmed by the present findings that are independent of depressive symptoms. Three individual subdomains of apathy have been identified: cognition, emotion, and autoactivation, related to dysfunction in different cortico-subcortical loops.[4] Here, we did not individualize these different subdomains of apathy, but we found that apathetic iNPH patients improved their STV after CSF tapping in a specific dual-task condition (walking and enumerating animal names) that is associated with executive functioning—this better improvement was also found for gait speed, but not for stride time and stride length. Interestingly, verbal fluency tasks—phonemic and categorical—are associated with separate frontal networks,[34,35] and unlike phonemic verbal fluency, bilateral hippocampi are selectively involved in the brain networks that supports categorical verbal fluency.[36] CSF tapping decreases the surrounding pressure around the ventricles not only in the frontal regions but also in the hippocampi, specifically involved in higher level of gait control—hippocampal volume has been associated with gait performance in older adults.[37,38] This hippocampal involvement in both categorical verbal fluency and higher level of gait control may explain why we reported a selective effect in the dual task of walking while enumerating animal names.

Apathy was associated with better gait improvement after CSF tapping, but it was not with better outcome of executive dysfunction. This dichotomy suggests that CSF tapping improves common networks associating apathy and gait, but not apathy and executive functioning in iNPH. When comparing apathy and executive changes after shunt surgery, Peterson et al. reported a nonsignificant correlation between improvement in apathy and improvement in executive function after iNPH surgery, while they found a significant correlation between improvement in apathy and postoperative improvement in global cognitive changes.[5] Apathy in iNPH may represent a phenotype that is relatively independent to dysfunction assessed by the classical executive tests, but more sensitive to motor initiation and the interference of motor and cognitive initiation in gait during dual tasking. Improvements in motor and cognitive initiation after CSF tapping in patients with iNPH may be explained by the decreased pressure on periventricular brain structures, but also on cortical structures in case of narrowing of cortical sulci translating cortical compression. The primary candidates for such functional deterioration related to increased CSF pressure in so-called iNPH are the basal ganglia and particularly the caudate nuclei given their tight topographical

relationship to the lateral ventricles, but also the prefrontal cortex given its size and fragility as a structure of late phylogenetical appearance. The cortico-basal ganglia-thalamo-cortical loops are involved in the pathophysiology of the different clinical phenotypes of apathy involving both basal ganglia and cortical prefrontal regions. These regions have been associated with both apathy[4,33] and gait control,[37,39] especially in dual-task conditions.[40] While bilateral caudate nucleus dysfunction is involved in all types of apathy, bilateral supplementary motor cortex and pallidal dysfunction are particularly involved in apathy with loss of autoinitiation.[4] Dysfunction in initiation, a key feature of apathy, is involved in initiation of steps and of evocation of names. Future studies should investigate whether autoactivation is specifically improved after CSF tapping or surgery in iNPH.

When comparing the performance of the dual task of forward counting between iNPH patients with and without apathy, the latter showed a dual-task gait increase in STV and decrease in gait speed, while the former presented similar performances between single walking task and walking while forward counting. Interestingly, when comparing Alzheimer's patients with and without extrapyramidal signs, Camiocioli et al. found a similar dual-task gait decrease in gait speed in both groups while performing the same dual task of forward counting.[41] This similar observation may suggest that iNPH patients without apathy could harbor another neurodegenerative condition in addition to iNPH. That should be confirmed in future investigations by using molecular neuroimaging and CSF biomarkers.

Quantifying apathy, while measuring gait in single and dual tasking before and after CSF tapping in a relatively large series of consecutive iNPH patients, constitutes the main strengths of this study. However, change in apathy was not measured after CSF tapping, because the SAS by definition evaluates a 4-week observational time.[16] The generalization of the study findings should be limited to iNPH patients that are still able to walk without assistance and cannot be applied to patients with severe gait disability.

5 | CONCLUSION

In conclusion, iNPH patients with apathy showed better gait improvement after CSF tapping than patients without apathy. This improvement was independent of depressive symptoms. These findings suggest a close relationship between apathy and reversible gait impairment in iNPH patients and should be taken into account in the clinical decision with candidates for shunt surgery.

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CONFLICT OF INTEREST

None declared.

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SUPPORTING INFORMATION

Additional Supporting Information may be found online in the supporting information tab for this article.

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