Motion sickness susceptibility and visually induced motion sickness as diagnostic signs in Parkinson's disease

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Abstract

Postural instability and loss of vestibular and somatosensory acuity are among the signs encountered in Parkinson's disease (PD). Visual dependency is described in PD. These modifications of sensory input hierarchy are predictors of motion sickness (MS). The aim of this study was to assess MS susceptibility and the effects of real induced MS in posture. Sixty-three PD patients, whose medication levels (levodopa) reflected the severity of the pathology were evaluated, and 27 healthy controls, filled a MS questionnaire; 11 PD patients and 41 healthy controls were assessed by posturography using virtual reality. The levels of levodopa predicted visual MS (p=0.01), but not real induced MS susceptibility. PD patients did not experience postural instability in virtual reality, contrary to healthy controls. Since PD patients do not seem to feel vestibular stimulated MS, they may not rely on vestibular and somatosensory inputs during the stimulation. However, they feel visually induced MS more with higher levels of levodopa. Levodopa amount can increase visual dependency for postural control. The strongest MS predictors must be studied in PD to better understand the effect of visual stimulation and its absence in vestibular stimulation.

Key Words: Parkinson's disease; motion sickness, motion sickness susceptibility; visual dependency.

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Parkinson's disease (PD) is a neurodegenerative disease characterized by destruction of dopamine neurons, involved in motor control. The cardinal symptoms are akinesia, rigidity, rest tremor and postural instability. Muscle rigidity affects the patient's motor performance, and plays an important part in their akinesia and postural instability.[1] Although postural instability is often considered as having its origin in motor neurology, non-motor signs as sensory disturbances are also important in PD. Postural instability in PD is not only based on muscle[2] and joint rigidity,[1,3] loss of muscle strength,[4] or failure to generate the right amount of postural force,[5] but also from a decrease in sensitivity and integration of the three senses necessary to maintain balance.

Difficulties in somatosensory integration, such as limb position information[6] and limb motion information[7,8] are described in PD patients. Therefore, this impaired kinesthesia disturbs postural control.[9,10] Although vestibular function is impaired in PD patients, it is unclear how it affect posture.[11] There were no differences in postural response evoked by galvanic stimulation, affecting Vestibulo Spinal Reflex (VSR), in PD patients compared to controls.[12] However, studies that assessed vestibular-evoked myogenic potential found abnormal responses in PD patients[13] (see Smith, 2018,[11] for a review). Moreover, head tilt perception is

inaccurate, highlighting a vestibular integration deficit.[14] PD patients also experience visuospatial deficits,[15] deteriorating self-motion perception, which are required for optimal postural control.[16] In addition to these sensorial integration dysfunctions, PD patients have difficulties integrating and organizing multisensory information.[17,18] This sensory organization impairment causes them to be overly reliant on visual input,[19] despite visual deficit, as well for visual subjective vertical,[20] for self-motion perception,[21] and for postural control.[22,23]

Inadequate integration of different movement stimuli can provoke motion sickness (MS).[24] Symptoms of MS such as discomfort, nausea, vomiting, dizziness, vertigo, loss of concentration, headache and increased fatigue are well known.[25] MS pathophysiology is two pronged:

- Vestibular stimulation, experienced in passive traveling by motored means of transport as car, train, boat, etc. and worsened by an absence of visual cues, which we call Real Induced Motion Sickness (RIMS).
- Visual stimulation without vestibular stimulation or physical motion.[26] Here the individual is motionless but the visual scenario is vivid as in daily life looking at traffic, or when exposed to virtual reality (VR) with head mounted display (Figure 1), provoking Visual Induced Motion Sickness (VIMS).[27]

Among the different theories explaining MS, the first is a theory of sensory conflict, which argues a neural or a sensory mismatch, especially between visual and vestibular input.[26,28] Visual dependency can more easily generate MS, when relying on incongruent visual input. Another theory postulates that some situations provoke a prolonged postural instability, thereby inducing MS. For example, in vehicles where people frequently experience changes in gravito-inertial forces, in amplitude and direction, which can provoke postural instability.[29] Both theories can provide arguments favorable to a hypothesis that PD patients could be susceptible to MS. Indeed PD patients are known to be visually dependent and to have an unstable posture.[21,22] Some individual predictors of MS susceptibility can stimulate debate. Mittelstaedt's review[30] highlighted the role of vestibular sensitivity in RIMS susceptibility when PD patients had unclear vestibular problems, other predictors such as anxiety[31,32] or difficulties regulating posture with vestibular input[16] which supports the above hypothesis. However, to our knowledge, no study had been published on this subject. We note that normal aging decreases sensory input acuity also,[33] and that aging could worsen problems already existing in PD, as PD can worsen problems previously existing in the elderly.

Dopaminergic drugs, that are used to reduce the cardinal bradykinetic symptoms of PD, may have side effects that worsen other motor and even non-motor symptoms such as sensorimotor neuropathy and anxiety.
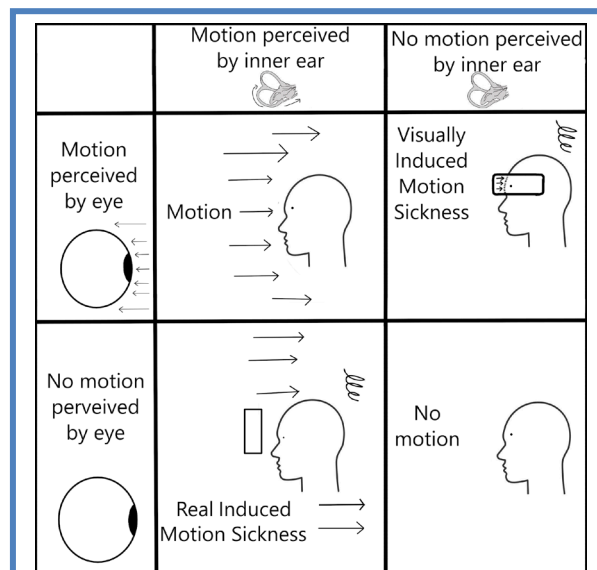

Fig 1. *Conditions of Real Induced and Visually Induced Motion Sickness apparition according to sensory mode that perceive motion.*
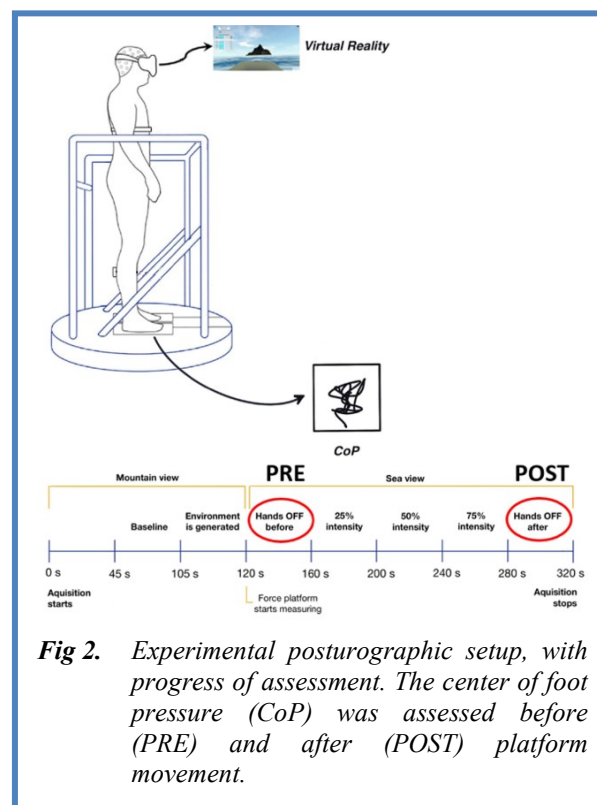

Fig 2. *Experimental posturographic setup, with progress of assessment. The center of foot pressure (CoP) was assessed before (PRE) and after (POST) platform movement.*

Medication deteriorates postural control by reducing postural reflex response,[5,34] can degrade proprioceptive input,[35] and the score of condition 5 in the Sensory Organization Test where vestibular input is the most important.[36] Therefore, we can hypothesize that the more PD patients take dopaminergic drugs, the more they should be susceptible to MS. This study's aim will

be on the one hand to evaluate subjective RIMS and visual discomfort in patients with PD and evaluate if age and the amount of medication can predict susceptibility to visual dependency and RIMS susceptibility. On the other hand, the second aim will be to assess if PD patients have worse postural control than HC in a simulated situation that can provoke RIMS. A VR-based experiment called BioVRSea is used in this case, having previously been shown useful in the assessment of neurophysiological signals of postural control/motion sickness in healthy,[37] and concussion subjects.[38]

Materials and Methods

The study was conducted in accordance with the Declaration of Helsinki, and approved by the Icelandic Bioethical Committee, nr. VSN20-101-V, date of approval: February 2022.

All eligible participants were informed about the study protocol and were free to refuse to be included.

Written informed consent to participate in this study was provided by the participants.

Participants

Sixty-three patients with idiopathic PD and 27 Healthy Control (HC), aged between 50 and 90, filled out a questionnaire. Eleven patients with idiopathic PD and 41 HC had a posturographic assessment. HC had to be at least 50 years old, and to have no neurological, ocular disease nor any balance disorder.

Methods

Questionnaire

The MS susceptibility questionnaire was partitioned in two sections. The first part was based on a French version of short Motion Sickness Susceptibility Questionnaire (MSSQ)[39,40] to assess RIMS susceptibility. This susceptibility was asked for before disease's onset and at the present time for PD patients. Mean score was calculated for both periods. Each item was evaluated with a four point Likert-scale. HC were asked for 10 years ago (called MSSQ 10 years (M10) for both groups) and for current time (called Current MSSQ (CM)). The second part assessed visual dependence susceptibility, based on Mallinson's questionnaire on Visual-Vestibular Mismatch,[41] including 13 items with a four point Likert-scale about visual situations that can lead to sensations of discomfort for the current time only. The situations were varied; some rely on visual motion, some on vestibular sensation motion, and some present an open space visual scene. As described above for the first part, a mean score was calculated for this section (called VD). A neurologist assessed the patient when they came in consultation in University Hospital Nancy. After being assessed with a questionnaire, the neurologist assessed age, amount of daily medication with Levodopa Equivalent Dose (LED), disease severity with Hoehn and Yahr Stage,[42] and if the participant had

vision conditions such as glaucoma, cataract or macular degeneration, and if patient had an eye operation in his life.

Posturographic assessment

Posturographic evaluation was the same as the one used by Jacob and colleagues, evaluating individuals suffering from concussion.[38] After removing their shoes, participants were instructed to stand on a forceplate (sampling frequency 90 Hz, Virtualis, Clapiers, France), mounted on a moveable platform (Virtualis, Clapiers, France). The forceplate had four sensors under each foot platform and computed the Center of Pressure (CoP) in antero-posterior (AP) and medio-lateral (ML) axis.

Participants wore VR goggles showing a stationary mountain view during the first 120 sec., meanwhile they were instructed to stand still on the forceplates. Then the display changed to a sea simulation and participants saw a small boat at sea with waves and a small island in the distance. Participants have still to stand quietly during the first 40 sec (Pre). Then the platform moved synchronized with the displayed waves for 120 sec, with an increase of amplitude every 40 sec (respectively 25, 50 and 75% of maximal amplitude of platform). Patients were asked to remain as upright as possible and to hold security bars on the front of them while the platform moved. Then, the platform stopped moving, while the VR display continued to show the sea scene, and the patient stood quietly during 40 last sec without their hands on the bar (Post) as phase Pre (Figure 2). The platform movement synchronised to the visual stimulation added somatosensory and vestibular stimulation. The subjects can experience a boat simulator with all senses, which can provoke RIMS as if she/he were really on a boat.

For Pre and Post phases, the equivalent of 95% confidence ellipse of area covered and the length travelled by the CoP (Total Excursion, TotEx) were extracted from platform data. As visual stimulation could be more efficient in frontal plane, because of a potential for less efficiency of depth perception,[43] the effects on TotEx in the AP axis and ML axis were assessed. Before and after the VR experiment, a questionnaire allowed the assessment of MS symptoms that participants felt.

Statistical analysis

Data analysis was performed using the Statistica Software. To compare MS susceptibility of patients and healthy controls, independent t-tests were performed for each questionnaire part. A 2-way ANOVA (group x time) with repeated measure was performed to assess if PD became more susceptible to RIMS than HC (time being the comparison between M10 and CM). Univariate linear regressions were performed between the mean scores for each questionnaire part and parameters such as age, disease duration and LED for PD patients, which can reflect loss of and need for dopamine.

Table 1. *Participants' demographic data for questionnaire (on left) and for postural assessment (on right). Mean (±SD) or n (%).*

		Parkinson Disease (questionnaire)	Healthy Control (questionnaire)	Parkinson Disease (postural control)	Healthy Control (postural control)
n		63	27	11	41
Age		67.1 (± 9.2)	62.2 (± 8.5)	62.3 (±12.4)	58.9 (±6.4)
Gender	Men	42 (67%)	12 (44%)	9 (82%)	19 (46%)
	Women	20 (32%)	15 (56%)	2 (18%)	22 (54%)
Hoehn and Yahr stage	Stage 1	4 (6%)			
	Stage 2	32 (51%)			
	Stage 2.5	2 (3%)			
	Stage 3	21 (33%)			
Ocular disease	Glaucoma	1 (2%)	0 (0%)		
	Cataract	13 (21%)	1 (4%)		
	Macular degeneration	3 (5%)	0 (0%)		
	Eye surgery	16 (25%)	3 (11%)		

For posturographic assessment, a 2-way ANOVA (group x situation) with repeated measure was performed for each parameter (the comparison between Pre and Post conditions). Then, a post-hoc analysis was performed with a Tukey-HSD. As sample sizes are unequal and lose statistical power using ANOVA, a paired T-test was performed for each group between Pre and Post, and a T-test for unequal variance (Welch test) was performed in each situation to compare groups. Bonferroni correction was applied. As each set of data has two comparisons, the significant threshold will be 0.025 instead 0.05.

Fig 3. *Mean score of the questionnaire parts, compared between Parkinson Disease (PD) patients and Healthy Controls (HC). Mean score of motion sickness susceptibility ten years ago or before disease onset (M10) (a), mean score of current sickness susceptibility (CM) (b), mean score of visual dependency (VD) (c).*

Results

Population description

Sixty-three participants with PD (42 men, 20 women, one information missing) and 27 HC (12 men and 15 women) filled out the MS questionnaire. Mean age of PD patients was 67.1 (SD 9.2) y and mean age of HC was 62.2 (SD 8.5) y. Age difference between groups was significant (t = 2.3, $p = 0.02$). Mean Hoehn and Yahr stage was 2.3 (SD 0.6), with four patients where stage was not supplied. Complete demographic data are presented in Table 1 in the left-hand columns.

Postural control was evaluated in 11 PD patients and 41 HC. Mean age of PD patients was 62.3 (SD 12.4) and mean age of HC was 58.9 (SD 6.4). There were no age differences between groups. Patients were all classified as early stage. Demographic data are presented in Table 1 in the right-hand columns.

Motion sickness susceptibility

No difference was demonstrated between PD and HC participants for M10 (M = 0.62, SD = 0.71 vs M = 0.49, SD = 0.55, $p = 0.40$), for MA (M = 0.71, SD = 0.86 vs M = 0.44, SD = 0.60, $p = 0.14$) and for VD (M = 0.73, SD = 0.68 vs M = 0.52, SD = 0.59, $p = 0.16$) (Figure 3). ANOVA reveals no group effect ($F_{(1,88)} = 1.38$, $p = 0.24$), nor time effect ($F_{(1,88)} = 0.20$, $p = 0.66$), nor interaction effect ($F_{(1,88)} = 1.64$, $p = 0.20$). In M10, no difference between groups was highlighted, in the period in which both groups were healthy (before onset of the disease in PD patients).

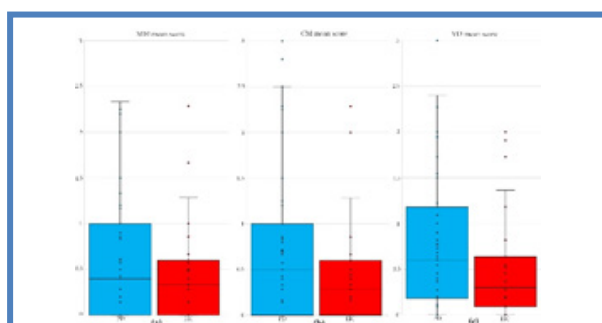Figure 4 shows the correlation between questionnaire scores and age and between questionnaire scores and
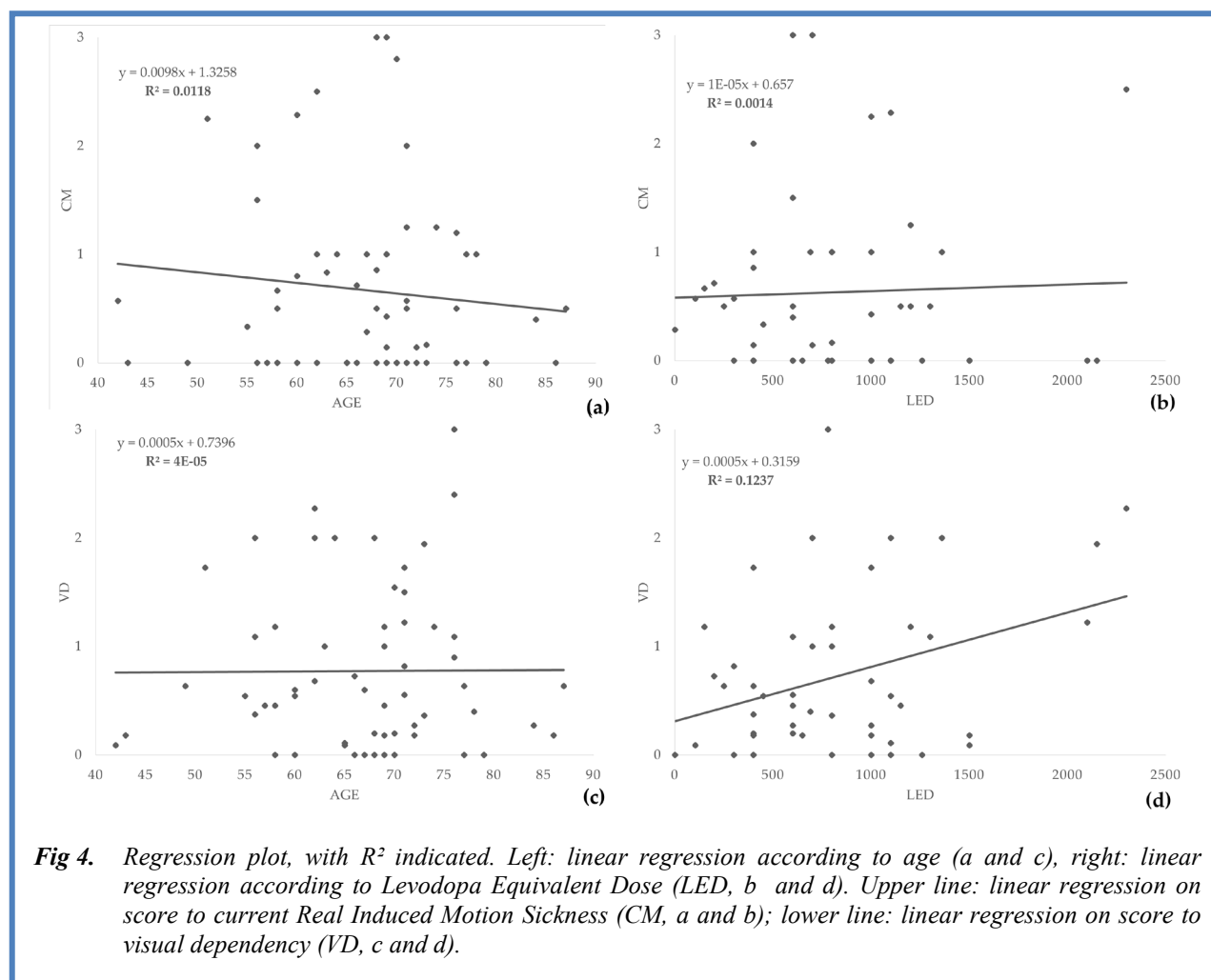
Fig 4. *Regression plot, with R² indicated. Left: linear regression according to age (a and c), right: linear regression according to Levodopa Equivalent Dose (LED, b and d). Upper line: linear regression on score to current Real Induced Motion Sickness (CM, a and b); lower line: linear regression on score to visual dependency (VD, c and d).*

LED. Age cannot predict M10 ($R^2 = 0.01$, $p = 0.33$), nor MC ($R^2 < 0.01$, $p = 0.40$) nor VD ($R^2 < 0.01$, $p = 0.96$) scores. LED cannot predict either M10 ($R^2 < 0.01$, $p = 0.74$) or MA ($R^2 < 0.01$, $p = 0.95$) scores. Nonetheless, higher LED is predictive of a higher score in VD ($R^2 = 0.12$, $p = 0.013$).

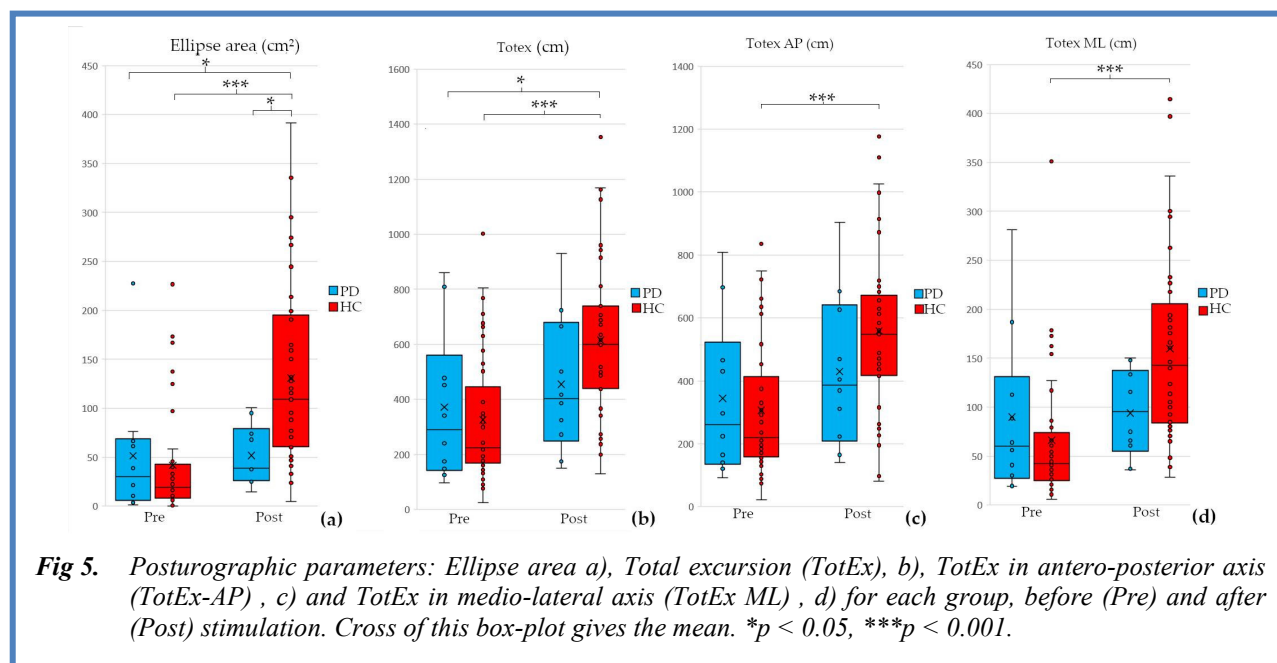*Posturography and Motion Sickness*

Postural parameters are presented in Figure 5. ANOVA on 95% confidence of ellipse area revealed no group effect, ($F(1,49) = 2.6$, $p = 0.11$) but a condition effect ($F(1,49) = 9.26$, $p = 0.004$) and an interaction effect ($F(1,49) = 9.1$, $p = 0.004$). Post-hoc analysis showed HC had less precise postural control after stimulation (M = 55.6, SD = 70.73) than before (M = 46.6, SD = 25.6, $p < 0.001$), than PD before stimulation (M = 49.8, SD = 23.9, $p = 0.014$) and after stimulation (M = 49.4, SD = 57.2, $p = 0.015$).

ANOVA on TotEx shows no group effect ($F(1,49) = 0.50$, $p = 0.48$) but a condition effect ($F(1,49) = 17.3$, $p < 0.001$) and an interaction effect ($F(1,49) = 5.35$, $p = 0.02$). Post-hoc analysis shows that HC after stimulation (M = 487.0, SD = 504.0) are less stable than before stimulation (M=326.3, SD=233.1, $p<0.001$) than PD

before stimulation (M = 363.9, SD = 277.0, $p = 0.05$) but not than PD after stimulation (M = 449.7, SD = 469.7, $p = 0.31$).

At uni-axial TotEx, ANOVA revealed only an effect of condition for the AP axis ($F(1,49) = 15.7$, $p < 0.001$), and a condition effect ($F(1,49) = 15.2$, $p < 0.001$) and an interaction effect ($F(1,49) = 12.8$, $p < 0.001$). However, for both axes, post-hoc analysis revealed a difference only for HC before (AP-axis: M = 307.1, SD = 211.4, ML axis: M = 66.2, SD = 66.37) and after simulation (AP-axis: M = 455.9, SD = 470.0, $p < 0.001$, ML-axis: M = 107.8, SD = 113.1, $p < 0.001$). PD patients had a similar stability in both axes before stimulation (AP-axis: M = 334.7, SD = 250.8, ML axis: M = 90.1, SD = 84.5) and after (AP-axis: M = 425.4, SD = 2441.0, ML axis: M = 91.6, SD = 100.5).

Figure 6 presents the score of the symptom questionnaire. A paired t-test revealed a difference in the symptom questionnaire score in the HC group (Pre: M = 0.8, SD = 1.20, Post: M = 2.8, SD = 4.50, t(53) = 3.42, $p = 0.001$), but not in the PD group (Pre: M = 2.1, SD = 2.3, Post: M = 1.9, SD = 3.8, t(10) = 0.12, $p =$

Fig 5. *Posturographic parameters: Ellipse area a), Total excursion (TotEx), b), TotEx in antero-posterior axis (TotEx-AP) , c) and TotEx in medio-lateral axis (TotEx ML) , d) for each group, before (Pre) and after (Post) stimulation. Cross of this box-plot gives the mean. *p < 0.05, ***p < 0.001.*

0.9). A Welch test revealed a score significantly higher in PD than HC ($t(63) = 2.7$, $p = 0.008$) before posturographic assessment, but no significant difference after ($t(63) = 0.62$, $p = 0.53$).

Discussion

This study had two main aims. The first was to assess if PD patients were more susceptible to MS and/or visual dependency, and if age and LED can be a predictor of these susceptibilities. Contrary to our hypothesis, PD patients did not feel subjectively more susceptible than HC in RIMS, or visual dependency. Despite the absence of a difference between PD and HC, LED, which is a specific PD parameter, seems to be a predictor of visual dependency. Age is not a predictor of either susceptibility, and LED is not a predictor of RIMS susceptibility.

The second aim was to assess if a RIMS provoking situation worsened postural control in PD patients more than in HC. Contrary to our hypothesis, the stimulation

Fig 6. *Total score of feeling questionnaire, before (Pre) and after (Post) stimulation for Parkinson's Disease patients (PD) and Healthy Control (HC), box plot. **: p < 0.01.*

seems to provoke RIMS in HC, but not in PD patients. Age seems to have no relationship with RIMS nor visual dependency susceptibility. The influence of age in MS is not clear. On the one hand, some authors think that older people have higher RIMS susceptibility, despite lack of literature, due to avoiding provocative situation behavior.[44] This hypothesis can be supported by the involvement of sensory deficits, which can worsen RIMS susceptibility,[30] while higher proprioceptive weighting allows the decrease of RIMS susceptibility.[45] Yet, getting older decreases sensory acuity, such as visual acuity, with loss of processing speed, motion discrimination,[46] vestibular sensory[47] and somatosensory input,[48] which could lead to visual dependency. On the other hand, for another authors, aging has not shown the same effect on vestibular stimulation in RIMS and on visual stimulation in VIMS.[49] RIMS susceptibility begins around age of 5, increases up to around twenty and decreases during adulthood.[50] This decrease could be due to vestibular acuity decrease that desensitizes sensory conflict. However, this hypothesis cannot explain why, contrary to RIMS, VIMS increases with age.[30] An another explanation can be habituation, which is quite specific factor of MS,[51] which can explain VIMS or cybersickness where older people have not often, if ever, experienced VR simulations. PD patients do not seem be more RIMS susceptible with increasing age, similarly to healthy subjects. Here again, the sensory acuity loss or the habituation can explain these results.

PD patients do not seem to be more susceptible to RIMS than the healthy elderly. All our results point in this direction. PD patients scores of current RIMS susceptibility did not differ to PD patient scores of RIMS susceptibility before disease onset, nor to HC current score. Furthermore, sensory stimulation in the
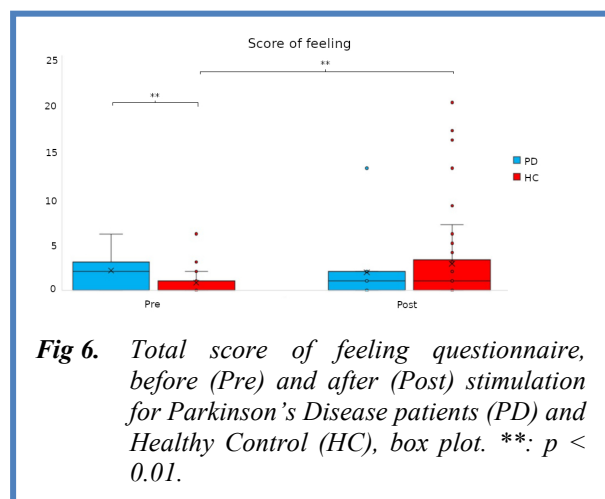
postural task did not provoke postural instability nor increased feelings of discomfort in PD patients. On the contrary, HC worsened their overall postural performance and precision after stimulation, which reflects postural instability provoked by MS. Furthermore, they felt worse after the postural test. We can note that the effect of stimulation was similar on both axes, and not present only on the ML axis as we hypothesized. HC had no significantly different postural performance after stimulation than PD patients, but their postural control was less precise than PD patients. PD patients are not more motion sick than healthy subjects and did not feel RIMS. We could hypothesize that vestibular and somatosensory stimulation did not perturb PD, as they more rely on visual input to control their posture.[23] Their somatosensory and vestibular inputs are impaired, and PD patients might not feel these stimulations accurately enough to experience completely the boat simulation. Therefore, PD patients did not feel worse after postural task than before. They do not seem to feel VIMS when they have only a visual stimulation during postural recordings. As HC felt worse after rather than before stimulation, the stimulation is MS provocative for susceptible subjects. This absence of MS can be explained because PD vestibular dysfunctions may not be the same dysfunctions that are a RIMS predictive. For example, PD patients have a higher vestibular-ocular reflex gain than healthy subjects,[52] but this parameter is not a clear predictor of RIMS whereas time constant seems to be,[53] but was not studied in PD to our knowledge. Vestibular evoked myogenic potential threshold and asymmetry are also predictors of RIMS, but not amplitude.[54] However, vestibular evoked myogenic potentials in PD patients showed amplitude abnormalities.[13] Further studies on these specific parameters need to be conducted. Further studies on MS with BioVRSea on electroencephalographic responses can be interesting to, as in healthy subject, for example to investigate if HC or PD can adapt to this perturbation, as HC know a cortical adaptation during a proprioceptive perturbation,[55,56] and investigate the effect of vision on this adaptation.[57]

Concerning the relation between MS and medication, LED did not predict RIMS susceptibility. As discussed above, RIMS susceptibility does not seem to be more frequent in PD patients than in HC. Nevertheless, if PD patient scores on the VD questionnaire were not significantly different from the HC score, LED, a PD specific parameter, seems to predict VD score. These results are contradictory. The more intriguing result is the absence of difference in RIMS susceptibility between groups, because PD have a more important visual dependency than healthy subjects.[16,21,22] These studies highlighted visual dependency in a self-motion perception task or in a task where subjects needed to separate/discriminate target and field to perceive and analyze target, as in a rod and frame test to perceive vertical. However, to our knowledge, no experiments

studied visual dependency as factor of MS in PD. We can hypothesize that visual dependency decreases performance on tasks that need multisensory-integration in the PD group, but is not enough severe to induce a feeling of discomfort in a provocative stimulation. Given this absence of group difference, the relation between LED and the visual dependency score is harder to interpret. If PD patients are globally not more susceptible to VIMS, this susceptibility seems depend on medication. Dopaminergic drugs deteriorate postural stability, especially proprioceptive acuity[35] and seem to increase visual dependency. Azulay et al. remark visual dependency does not depend on medication,[22] but this conclusion is made because he did not see differences in performance before and after taking the drug. Nonetheless, this statement concerns a short-term effect of medication, but did not consider long-term effect of medication. Furthermore, our results are in agreement with Hawkins et al. findings, which indicate that LED has an inverse relationship with postural performance in tasks on firm and foam surfaces, with VR-induced visual perturbation,[23] even if this task, as in another studies, assessed visual dependency concerning postural stability, and not directly MS susceptibility.

This study has some limitations. First, our samples have unequal size, especially for postural task. As well as for questionnaire rather than for posturographic assessments, more men than women in PD group are included in our study. Nonetheless, this is representative of gender ratio in PD: men/women with PD is around 2/1.[58,59] We also note that our visual dependence questionnaire assessed a quite large spectrum of situations that could provoke MS. However, factors which provoke VIMS or RIMS,[30] or visual vertigo with large open spaces, are not the same. Our results remain quite broad. Follow-up studies with this questionnaire can specialize this questionnaire to VIMS, or partition their questionnaires to have specific scores. Lastly, in our study, Levodopa is used to reflect disease severity, but our results can be explained by side effects of Levodopa too. Side effects that could affect postural control may include orthostatic hypotension and abnormal movements at the start of treatment (gradual increase in dosage may limit these effects) and alternating involuntary movements and disabling stiffness with prolonged treatment. Rarely, gait disturbance, blurred or double vision and disorientation may occur. Wright et al. showed that kinesthetic sensitivity of axial musculature is impaired in PD, especially when using levodopa medication, that contributes to impairment of posture.[60] In any case, levodopa is the effective therapeutic strategy to overcome the worsening of PD.

In conclusion, this study suggests PD patients have not higher real induced motion sickness susceptibility, but are susceptible to some visual-induced motion sickness provocative situations. This difference can be explained by a high reliance on visual input and a low

performance of vestibular and somatosensory inputs, as well as a potential habituation of provocative stimulations. If stimulation to habituate to a specific disturbing situation can help to desensitize to visual-induced motion sickness, strategies such as practicing physical activities that modify the sensory input hierarchy, increasing somatosensory weighting, could be efficient to decrease visual overreliance and limit this sickness effect that can occur in daily life, as the increased risk of falling.

List of acronyms

AP - antero-posterior
CM - Current MSSQ
CoP - Center of Pressure
HC - Healthy Control
LED - Levodopa Equivalent Dose
M10 - MSSQ 10 years
ML - medio-lateral
MS - motion sickness
MSSQ - Motion Sickness Susceptibility Questionnaire
PD - Parkinson's Disease
RIMS - Real Induced Motion Sickness
TotEx - Total Excursion,
VD - Visual dependence mean score
VIMS - Visual Induced Motion Sickness
VR - virtual reality
VSR - Vestibulo Spinal Reflex

Contributions of Authors

Conceptualization: HP, PG, PP; methodology: HP, PG, PP; software: AP, DJ, RA; validation, AP, DJ, HP, PG, PP; formal analysis: AP, DJ, RA, HP, PG, PP; investigation: SF; resources: AP, DJ, RA, SF; data curation: HP, PG, PP; writing—original draft preparation: AP, DJ, PG, PP; writing—review and editing: AP, DJ, HP, PG, PP; visualization: AP, DJ, RA; supervision: HP, PG, PP; project administration: PG, PP; funding acquisition: PG, PP; All authors have read and agreed to the published version of the manuscript.

Data Availability Statement

Requests may be directed to the following clinicians (MD): SF, HP and PP.

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Conflict of Interest

The authors declare no conflict of interest. The funders had no role in the design of the study; in the collection, analyses, or interpretation of data; in the writing of the manuscript, or in the decision to publish the results.

Ethical Publication Statement

We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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