Movement Disorders
CLINICAL PRACTICE

RESEARCH ARTICLE

Phantom Boarder Relates to Experimentally-Induced Presence Hallucinations in Parkinson's Disease

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Abstract: Background: Phantom boarder (PB) is the sensation that someone uninvited is in the patient's home despite evidence to the contrary. It is mostly reported by patients with neurodegenerative disorders such as Alzheimer's disease, dementia with Lewy bodies or Parkinson's disease (PD). Presence hallucination (PH) is frequent in neurodegenerative disease, shares several aspects with PB, and is the sensation that someone is nearby, behind or next to the patient (when nobody is actually there). Recent work developed a sensorimotor method to robotically induce PH (robot-induced PH, riPH) and demonstrated that a subgroup of PD patients showed abnormal sensitivity for riPH.

Objective: We investigated if PD patients with PB (PD-PB) would (1) show elevated sensitivity for riPH that (2) is comparable to that of patients reporting PH, but not PB (PD-PH).

Methods: We studied the sensitivity of non-demented PD patients in a sensorimotor stimulation paradigm, during which three groups of patients (PD-PB; PD-PH; PD patients without hallucinations, PD-nPH) were exposed to different conditions of conflicting sensorimotor stimulation.

Results: We show that PD-PB and PD-PH groups had a higher sensitivity to riPH (compared to PD-nPH). PD-PB and PD-PH groups did not differ in riPH sensitivity. Together with interview data, these behavioral data on riPH show that PB is associated with PH, suggesting that both share some underlying brain mechanisms, although interview data also revealed phenomenological differences.

Conclusions: Because PD-PB patients did not suffer from dementia nor delusions, we argue that these shared mechanisms are of perceptual-hallucinatory nature, involving sensorimotor signals and their integration.

Patients with phantom boarder phenomenon (PB) report that someone uninvited has entered or lives in the patient's home, despite of evidence to the contrary.[1] The unsolicited visitor is mostly experienced by patients as an unfamiliar intruder with malevolent intentions (e.g., harm or rob the patient) or hassling behaviors (e.g., make noise), although the visitor may also be experienced as a friend or family member.[1–4] PB is clinically relevant as it occurs repeatedly, and is a compelling experience, which is often destabilizing for patients, caregivers, and their relationship. Furthermore, PB has been associated with earlier home placement and delirium.[5] PB is prevalent in several neurodegenerative diseases associated with dementia, such as dementia with Lewy bodies (DLB),[6,7] Alzheimer's disease (AD),[8,9] vascular dementia[10] and Parkinson's disease (PD).[6] PB is often described as the most common "delusion" (together with paranoid ideation) in such diseases, with Aarsland et al.[6] reporting PB in 41% of DLB and 17% of PD dementia (PDD) patients.

Despite this clinical relevance, understanding of the involved brain mechanisms of PB remains limited and its "exact nosology (…) debatable".[11] In his initial clinical description, Rowan[1] described PB as a delusional symptom and subsequent authors classified PB among delusional misidentification syndromes such

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[Corrections added on 11 April 2023, after online publication. PD-nH has been revised to PD-nPH throghout the article.]

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as Capgras syndrome, mirror or television misidentification.[9,10,12] Others have considered PB as a complex hallucination, based on the observation that in some cases the PB is also heard or seen.[8,10,13] Either classification (hallucination, delusional misidentification) has received some support from the results of symptom cluster analysis.[7,8,14] These studies and findings are sparse, however, and further complicated by the fact that research was carried out in patients with different neurodegenerative diseases. Thus, Ballard[8] tested patients with dementia of different etiologies and linked PB with complex visual hallucinations (their Factor 1), whereas Nagahama et al.[7,14] studied DLB patients and associated PB with delusional misidentification (their Factor 1). PB is further complicated by the importance of additional psychological and social factors. Thus, fear and anxiety,[1,8,10] social isolation, lack of interpersonal relationships and personal loss[15] have been reported in patients with PB. Although most patients with PB suffer from dementia, past work showed that PB is independent of the degree of severity of the associated cognitive deficits objectified by neuropsychological evaluation[8,16] and can even be observed in patients without any clinical signs of dementia.[1,3] Finally, regarding the neural correlates of PB, a SPECT (single photon emission computed tomography) study of a large group DLB patients[14] showed involvement of a network consisting of insula, inferior frontal gyrus, hippocampus, and the striatum.

Presence hallucination (PH) defined as the vivid sensation that somebody is nearby when no one is actually there and can neither be seen or heard.[17] PH shares several clinical aspects with PB and yet, PH has not been considered in relation to PB and their potential relationship has not been investigated experimentally. PH is grouped among minor hallucinations (together with passage hallucinations and visual illusions)[18] and is frequent in PD patients.[17,19] PH may occur in recurrent fashion, affecting about half of patients,[20,21] and often precedes the onset of structured visual hallucinations.[22,23] PH also occurs in elderly healthy individuals,[24] in extreme conditions (sailors, explorers or extreme mountain climbers),[25,26] psychiatric illnesses,[27,28] and neurological cases and illnesses other than PD (epilepsy, DLB).[29–31] These *presences* observed in those different populations share phenomenological aspects with the PH in PD, as subjects describe feeling someone close to the body, behind, above their shoulder or to one side. PB and PH share other characteristics: in PB the *uninvited intruder* or *person* is often an unfamiliar person as is most often the case for the *presence* in PH.[17,32,33] In PH, the sensed *presence* often appears at home, which is a defining feature of PB. Typically, the sensed *presence* in PH cannot be seen or heard, again as the *phantom boarder*, and both are frequently reported by patients with DLB and PD.[29,34] Although at first sight, PH and PB may share features with extracampine hallucinations (EH) and include the hallucination of a person in one's home, PH and PB should be distinguished from EH. EH were first described by Eugen Bleuler[35] and defined as hallucinations outside a given sensory modality's perceptual field: thus outside the visual fields in the case of a visual EH (see also Sato & Berrios).[36] Concerning PH and PB, one important difference with EH is that they are *seen* by the patient in the case of visual EH (i.e., case 1, case 5 from Bleuler[35]; heard in the case of an auditory EH). This is not the case for PH[37] or PB,[1] which are neither seen, heard nor felt (if, in rare instances, they are heard or felt, they still differ, because such cases of PH are not extracampine, but intracampine hallucinations). Therefore, EH that are seen or heard or felt, are a broader class of hallucinations (visual, tactile, auditory) and, as already described by Bleuler,[35] are not specific for people, beings, or animals, as for PH and PB. Based on these clinical similarities between PH and PB and the previous link of PB to hallucinations,[8,10,13] we here studied whether PB is associated with PH by exposing a group of PD patients with PB to a sensorimotor stimulation protocol that has been shown to induce experimentally-controlled or robot-induced PH (riPH)[38] in healthy participants[37] and in PD patients.[32] The sensitivity of PD patients with PB (PD-PB) to riPH was tested behaviorally and compared with those of two other groups of PD patients (patients with PH but no PB: PD-PH; patients without PB nor PH: PD-nPH). Based on the assumption that PB and PH share common mechanisms, we predicted that PD-PB patients would show similar sensitivity to riPH as described previously in PD-PH patients[32] and elevated sensitivity compared to PD patients without hallucinations (PD-nPH).

Materials and Methods

Participants

In a group of PD patients that was recruited for a previous study,[32] we retrospectively searched for patients with PB (PD-PB, N = 4; 16.7%) and compared them with two groups of PD patients without PB: patients with PH, but without PB (PD-PH, N = 7; 29.2%) and patients with neither PB nor PH (PD-nPH, N = 13; 54.2%). Three of the PD-PB patients had both PB and PH and only one patient had PB without PH.

All patients gave written informed consent prior to participating in the study approved by the local ethics committee. Diagnosis of PD was made by trained neurologists. Patients were not suffering from other neurological disorders, had neither psychiatric illnesses nor substance abuse disorders. The study procedures were administered in the following order: cognitive examination, experimental robotic paradigm, neuropsychiatric assessment and debriefing session. All patients were tested on their regular pharmacological treatment and at similar time of the day.

Clinical Assessment

Global cognitive functioning and screening for dementia were carried out by means of the Montreal Cognitive Assessment (MoCA).[39] Motor symptoms severity of PD were assessed by trained neurologists using the Part III of the Unified Parkinson's Disease Rating Scale (MDS-UPDRS).[40] The frequency and phenomenology of PH and other hallucinations, PB, and delusions were obtained through a semi-structured interview conducted by a trained neuropsychologist. We used the Prodromal Questionnaire (PQ-16)[41] to assess the occurrence of psychotic

prodromal signs (Part 1) including hallucinations and delusional ideas, and the level of distress associated to such signs (Part 2).

Robot-Induced PH Paradigm

The patients were seated and equipped with a blindfold and headphones delivering constant white noise to isolate them from external stimulations and distractions. As described in Bernasconi et al.[38] participants were instructed to perform back and forth movements with their most affected hand by means of a robotic device (Geomagic Touch®) placed in front of them. The hand movement performed by patients was reproduced by another robotic device located behind them, delivering tactile feedback on the participants' back (see Fig. 1A). Participants were asked to perform the sensorimotor task, while a randomized delay from 0 to 500 ms (6 delays with steps of 100 ms) was introduced between the hand movement and the tactile feedback on the back. On each trial, each patient performed 10 successive poking movements (automatically counted) and immediately afterwards gave a Yes or No answer to the following question assessing riPH (robot-induced PH): "Did you feel as if someone was standing close by—behind or next to you?". In total, each patient underwent three sessions of 18 trials (three repetitions per delay). Breaks between sessions allowed patients to avoid physical discomfort and fatigue. This experimental procedure does not only involve tactile stimulation on the back but also proprioceptive and motor cues from the upper limb (and additional robotically controlled spatiotemporal cues related to the incongruency between these proprioceptive-tactile-motor signals). Therefore, tactile cues alone are not sufficient to induce PH, as they are present in the same way in all experimental conditions experimental condition. What differs is the spatiotemporal congruency between the involved sensorimotor (proprioceptive, tactile, and motor) signals.

Data Analysis

For each participant (except for one patient from the PD-PB group who was not able to perform the task due to severe dyskinesia), responses to the Yes/No riPH were averaged for each delay (leading to one value per delay, per participant). Responses were then analyzed with linear mixed-effects models (lme4 and lmerTest packages).[42,43] The model included delay (6 delays) and group of patients (PD-PB, PD-PH, PD-nPH) as fixed effect (interaction between the two), and included a random intercept for patients. Significance of fixed effects was estimated using Satterthwaite's approximation for degrees of freedom of F statistics.

Data Sharing

Data and scripts used to generate the analyses presented in the paper are available here: https://gitlab.epfl.ch/fbernasc/phantom_boarder_pd.git.

Results

We assessed whether riPH depends on the degree of conflict applied during sensorimotor stimulation (i.e., different delays inserted between the movements of the front robot and the back robot) and here, especially, whether this differs between the three groups of patients (PD-PB; PD-PH; PD-nPH). Behavioral results show[1] that the intensity of riPH increased with increasing spatiotemporal conflict for all three groups (delay dependency) (main effect of Delay; $F_{(1,110)} = 12$, p-value < 0.01). Critically,

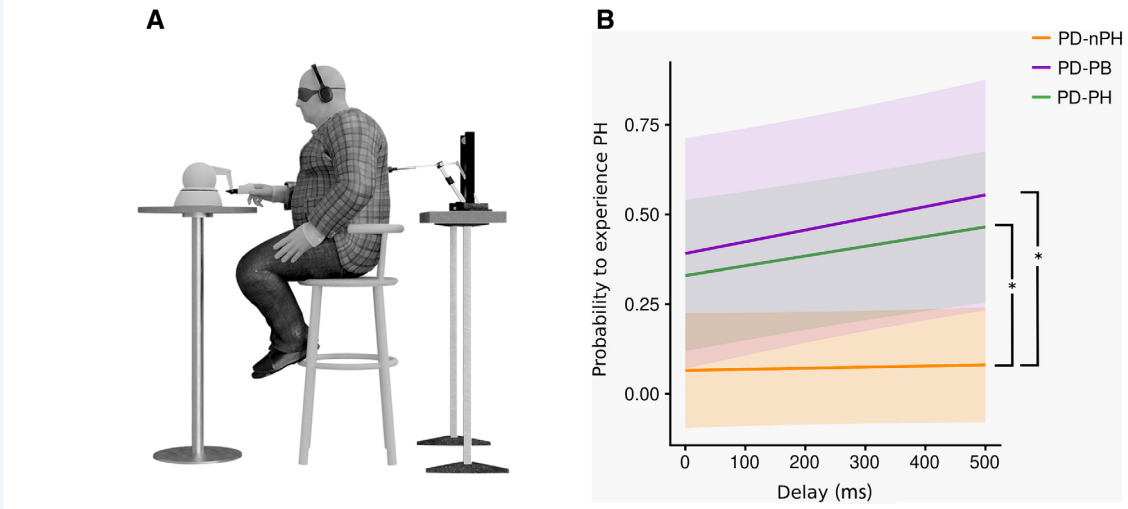

Fig 1. riPH. (**A**) Robotic setup. (**B**) Higher proneness to riPH and higher delay sensitivity in PD-PB (purple) vs. PD-nPH (orange). Thick line: mean of fitted model; shaded area: 95% confidence interval.

TABLE 1 *Demographic and Clinical Data for Each Group of Patients with PD. Statistics Were Calculated Comparing the Three Groups of Patients*

	PD-PB (N = 4)[a]	PD-PH (N = 7)[a]	PD-nPH (N = 13)[a]	*p*-value[b]
Age (years)	63 (14)	62 (15)	66 (8)	>0.9
Sex at birth				0.20
F	2 (50%)	2 (29%)	9 (69%)	
M	2 (50%)	5 (71%)	4 (31%)	
Clinical characteristics of PD				
Duration of PD (years)	13 (3)	7 (4)	9 (6)	0.12
UPDRS III	24 (16)	19 (12)	19 (18)	0.80
PQ16 (Part 1)	3 (2)	5 (2)	1 (1)	<0.001
PQ16 (Part 2)	3 (3)	4 (6)	1 (3)	0.13
Levodopa equivalent daily dose	559 (166)	646 (437)	786 (653)	>0.9
(LEDD including dopamine agonists) (mg/day)				
Cognitive examination				
MoCA	26 (2)	27 (2)	25 (2)	0.30

[a]Mean (SD); n (%).
[b]Kruskal–Wallis rank sum test; Fisher's exact test.

this delay dependency differed between the three patient groups (interaction: $_{F(2,110)} = 3.11$, *p*-value = 0.048; Figure 1B). Post-hoc analysis revealed that both PD-PB and PD-PH had a higher delay sensitivity compared to PD-nPH (PD-PB vs. PD-nPH: interaction: $F_{(1,75)} = 4.3$; *p*-value = 0.04; PD-PH vs. PD-nPH: $F_{(1,95)} = 6.4$, *p*-value = 0.01). There was no delay sensitivity difference between PD-PB and PD-PH (interaction: $F_{(1,\ 50)} = 0.06$, *p*-value = 0.8). Critically, there was also a main effect of group and PD-PB had a higher intercept (bias) compared to PD-nPH (PD-PB vs. PD-nPH: main effect of Group: $F_{(1,\ 17)} = 4.2$, *p*-value = 0.04) (Fig. 1B), whereas the intercept did not differ between PD-PH vs. PD-nPH (main effect of Group: $F_{(1,\ 20)} = 3.8$, *p*-value = 0.06) or between PD-PB and PD-PH (main effect of Group: $F_{(1,\ 11)} = 0.07$, *p*-value = 0.8).

Antiparkinsonian medication, motor impairment, age, and gender did not differ between the three groups. All patients presented normal cognition to mild cognitive impairment, and MoCA scores did not differ between the three groups (see Table 1). There was only one difference in a questionnaire screening for risk of developing psychosis (PQ-16 score, Part 1), which was elevated for both, PD-PB and for PD-PH groups, versus the PD-nPH group (PD-PB and PD-PH did not differ in PQ-16).

Detailed interviews with all patients revealed the following additional findings. For three PD-PB, the *phantom boarder* was experienced as an unfamiliar person, as was the felt presence experienced during PH. The remaining PD-PB patient experienced her own children (or other family members) as *phantom boarders* and her sister (or mother) as the *presence* during PH. Neither the PB nor the PH was seen or heard by any of the four patients. All PB occurred in places familiar to the patients, either at home or at secondary well-known vacation home, while PH occurred either at home (two patients) or outside the patients' home (one patient). However, all three patients having both PH and PB clearly distinguished one from each other. Thus, for all three patients who experienced both PH and PB, the two phenomena always occurred at different times. Moreover, whereas these patients felt the presence (PH) to be in the same room as the patient, to be located very close to them (0–3 m), and at a fixed location either next to or behind them, this was different for the PB. The PB was always experienced at larger and more variable distances from the patient's body and never in the same room as the one occupied by the patient (Fig. 2).

In addition to PH, PD-PB patients also reported other hallucinations (Table 2). Thus, among the four PD-PB patients, three reported complex visual hallucinations, two reported visual illusions, two simple auditory hallucinations (sounds, noise), and one had passage hallucinations. These hallucinations were never reported at the same time as the PB. One of the PD-PB patients reported mild theft delusions, always during PB instances. Visual and auditory hallucinations did not differ between PD-PB and PD-PH ($p > 0.9$).

Discussion

The present study investigates PB and PH in patients with PD that share several phenomenological characteristics concerning sensed presences, but also differ in other aspects. To the best of our knowledge, our study is the first to provide behavioral

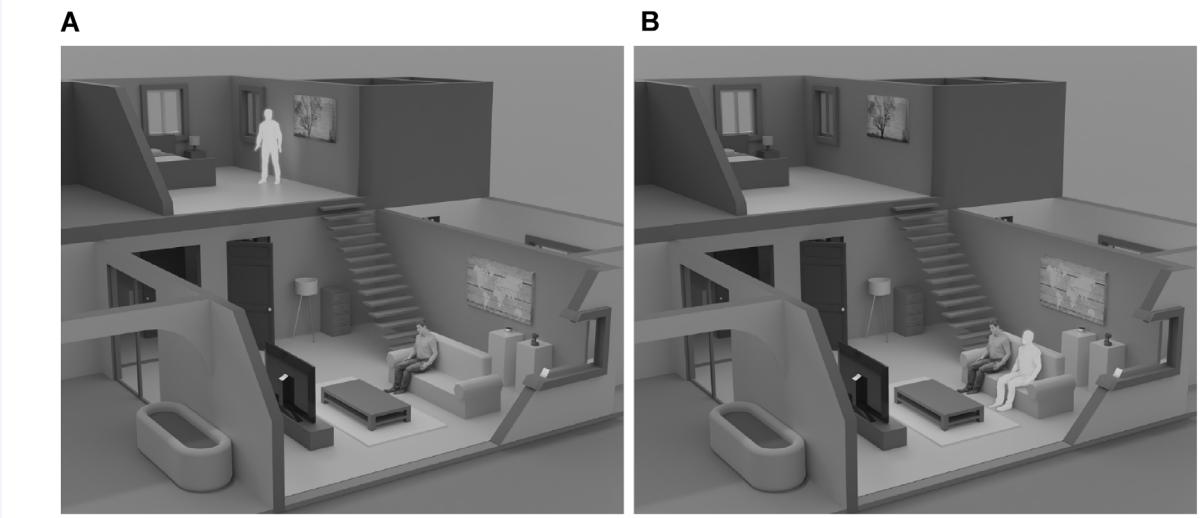

Fig 2. Illustration of PB and PH in an ecological setting. PB (**A**) is located on the first floor and PH (**B**) next to the patient (indicated as sitting on couch). Hallucinated PB and PH are indicated by the white silhouette.

indications that PD patients with PB show[1] elevated proneness to riPH, similar to patients with PD-PH, and[2] a higher delay sensitivity compared to PD-nPH patients when exposed to a robotic procedure that has been shown to experimentally induce PH in different patient populations.[32,37,44] Moreover, elevated proneness and higher delay sensitivity for riPH in PD-PB[3] did not differ from PD-PH patients. Our robotic procedure exposed participants to different levels of conflicting sensorimotor stimulation and allowed us to measure riPH in real-time and across several controlled delay conditions. First, the finding of a higher riPH proneness (stronger bias) in reporting PH shows that PD-PB patients behave abnormally, compared to PD-nPH, when exposed to the robotic procedure, linking the symptom of PB that PD-PB patients experienced in daily life to experimentally-induced and real-time PH in the laboratory. Second, evidence of higher delay sensitivity during sensorimotor stimulation indicates that PD-PB, compared to PD-nPH, is compatible with an alteration of sensorimotor brain processes in fronto-temporal regions[32] and altered sensorimotor processes in hallucinations in the present PD-PB patients.[45–47] Third, the present data linked PB to PH by showing that riPH proneness and delay sensitivity did not differ between PD-PB and PD-PH. These findings are not related to a general response bias that PD patients have when exposed to the procedure because riPHs were much weaker in PD-nPH and because all patient groups showed delay sensitivity. Moreover, there were no significant demographic and clinical differences between the three groups. Antiparkinsonian medication, motor impairment, age, illness duration or gender cannot account for the reported differences in experimentally riPH (Table 1). Moreover, the present PD patients were all without

Table 2 *Other Hallucinations Experienced by Patients of the PD-PB Group*

Types of hallucinations/delusions in the PD-PB group	Patient 1	Patient 2	Patient 3	Patient 4
• Phantom boarder (PB)	✓	✓	✓	✓
• Presence hallucinations (PH)	✓	×	✓	✓
• Passage hallucinations	×	×	×	✓
• Visual illusions (e.g., pareidolia)	×	✓	×	✓
• Visual hallucinations	×	✓	✓	✓
• Auditory hallucinations	×	✓	×	✓
• Somatosensory hallucinations	×	×	✓	✓
• Olfactory/gustatory hallucinations	✓	×	✓	×

✓: presence of hallucination.
×: absence of hallucination.

dementia and only showed mild cognitive impairments (MoCA > 22), not differing between the three tested groups.

We argue that PB share underlying brain mechanisms with PH and that these mechanisms are of perceptual nature involving sensorimotor processes.[32,48] Thus, an altered sensorimotor integration of bodily signals would not only favor the occurrence of PH as shown previously,[32,49] but also of PB (present study). Involvement of perceptual mechanisms in PB is compatible with previous clinical work on PB, showing that in some cases the *phantom boarder* is heard or seen[8,10] and because some authors have used cluster analysis (of symptoms) and associated PB with complex visual hallucinations[8,10] and auditory hallucinations.[10] While, these previous data are compatible with a hallucinatory and perceptual origin of PB, the present data from the robotic paradigm suggest that the perceptual mechanisms of PB may involve primarily the sensorimotor system, including tactile, proprioceptive and motor signals and their integration. We note that these sensorimotor signals have been shown to be among the first and most prominent to be altered in PD.[45,50,51] The present robotic procedure[38] was motivated by earlier neurological data showing that invasive electrical stimulation in temporo-parietal cortex can reliably induce the PH and that disturbed processing of sensorimotor own body signals (tactile, proprioceptive, and motor cues) modulates PH.[31] A limitation of the present study is that we could not test riPH in a group of PD patients who had pure PB (without PH). It could thus be argued that not PB, but the occurrence of PH (and PB) in the present PD-PB patients, led to the elevated riPH proneness and delay sensitivity. We did report a patient with isolated PB, but were not able to test him fully with the riPH procedure. However, we report a higher bias (intercept) for riPH in PD-PB compared to PD-nPH, whereas this was not the case for PD-PH, suggesting that the occurrence of PB and not PH is related to this difference in riPH bias. None of our PD-PB patients had a history of symptoms related to delusional misidentifications and only one reported a mild transitory theft delusion directly related to the experience of PB, providing no support for the involvement of delusional-cognitive mechanisms in the present cases of PB.

Interview data revealed similarities, but also phenomenological differences between PB and PH. Concerning similarities, all PD-PB patients reported sensing the presence of another person during PB and PH, always without seeing or hearing the person. The sensed person was most often an unknown person, in PB and PH (except in one patient), and in all PD-PB patients the PH and PB always appeared and disappeared suddenly. However, all patients who reported PB as well PH clearly distinguished between the two phenomena, considering them not to be the same presences, and not associated with the same emotional valence, and context of occurrence. First, the presence (PH) sensed by all PD-PB patients (and PD-PH patients), was experienced very close (0–3 m) to their body either behind or to the side of the patient, and within the current room at a very specific location.[17,32] Patients very often report PH "as if someone was bending over" their shoulders. This differed for the phantom boarder, who was experienced by all PD-PB patients at much larger distances, outside the room (where the patient would be located at the moment) or even on a different floor or rooms, and also at more various and less defined locations inside the patients' home (Fig. 2). As indicated by a patient from the PD-PB group, "while the PH stays close to me, the other presences (PB) seem to wander in my apartment". Another marked difference between PB and PH is that they never occurred at the same time in the present PD-PB patients. Other differences between PB and PH are that the PB can represent multiple presences in all of our four PD-PB patients, whereas this was not reported by PD-PH. Moreover, past work[32,48] showed that PH may occur at different places and also outside one's home, whereas PB are limited to very familiar places, such as the patients' home (or secondary home). The present PD-PB patients' reports are not compatible with EH, as the sensed presence was neither seen nor heard as required for EH.[35,36] The PD-PB patients also did not report to be touched by the hallucinated presences. EH require an extracampine tactile, auditory, or visual hallucinatory component, which is not the case for PH or PB. However, borderline cases may exist for whom it is difficult to draw a clear line between the three phenomena, for example when patients indicate to feel being touched by a hallucinated presence who is nearby and in their home, but is perceived as remaining outside their tactile field of perception (i.e., a presence experienced at a distance, yet felt as touching the patient; a presence in the immediate proximity felt to be touching the patient; the former being rather a tactile EH and the latter case a PH associated with a tactile intracampine component). More work is needed on this topic, by acquiring detailed data about patients' experience, through the use of specialized questionnaires, and by searching for behavioral and neural differences and similarities (e.g., testing EH patients with riPH or other procedures), between these three fascinating feats of the human mind. Overall, these interview data on the phenomenological characteristics of PB and PH suggest that additional mechanisms, beyond the described sensorimotor mechanisms of PH, are likely to also be involved in PB. These may especially concern brain regions involving familiarity processing (compatible with prior work linking PB to misidentification or persons and places),[52] but also anxiety,[1,8,10] lack of interpersonal relationships, personal loss and living alone.[15] Nagahama et al.[7] included "the feeling of presence"—PH—in their factor analysis for psychotic symptoms in DLB, but did not relate PH to PB in their study. Future work should investigate how these additional mechanisms of PB interact with sensorimotor mechanisms of PH. The present robotic procedure has the advantage that it can be combined with tasks examining such additional processes: for example, by performing behavioral tasks during robotic stimulation.[44,53]

The present study suffers from several limitations. More experimental data in larger groups of patients, including a group with PB without PH (pure PD-PB), are needed to further investigate the brain mechanisms of *presences* and *phantom boarders* in PD, and to determine the clinical, behavioral and neural similarities and differences between both symptoms. Future studies should therefore acquire imaging data, investigating whether patients with PB recruit networks linked to PH[32] and/or whether brain regions involved in PD patients with PB rather

overlap with networks of visual hallucinations.[54–56] Extended neuropsychological examination would be relevant in understanding the relationship between cognitive dysfunctions (or decline) and PB, and the cognitive mechanisms involved.[52,57] Additionally, a clearly defined scale for assessing PB and PH would allow to refine their phenomenological differences and similarities. Finally, delusional misidentification syndrome (e.g., Capgras, reduplicative paramnesia) was not evaluated in depth in our interviews nor tested behaviorally and should be included in future studies.

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Author Roles

(1) Research project: A. Conception, B. Organization, C. Execution; (2) Statistical Analysis: A. Design, B. Execution, C. Review and Critique; (3) Manuscript Preparation: A. Writing of the first draft, B. Review and Critique.

O.B.: 1A, 1B, 2A, 2C, 3A, 3B.

F.B.: 1C, 2A, 2B, 2C, 3B.

J.P.: 1A, 1B, 1C, 2C, 3A, 3B.

Disclosures

Ethical Compliance Statement: The Ethics Committee of the Canton of Vaud (CER-VD) and the Ethics Committee of the Canton of Geneva (CCER-GE) in Switzerland evaluated and approved the study protocol. All participants were presented to the study orally and by means of a written information sheet. All participants gave their written informed consent prior enrollment in the study. We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this work is consistent with those guidelines.

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