RESEARCH ARTICLE

Investigating Visual Misperceptions in Parkinson's Disease: A Novel Behavioral Paradigm

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ABSTRACT: Visual misperception and hallucinations represent a major problem in advanced PD. The pathophysiological mechanisms underlying these symptoms remain poorly understood, with limited tests for their assessment. A recent hypothesis has suggested that visual misperception and hallucinations may arise from disrupted processing in the attentional networks. To assess and quantify visual misperceptions, we developed the novel bistable percept paradigm (BPP), which consists of a battery of "single" and "hidden" monochromatic images that subjects are required to study until they are satisfied that they have recognized everything that the image may represent. In this experiment, 45 patients and 18 age-matched controls performed the BPP. Using an error score value derived from the control group, 23 patients were identified as having significant deficits on the task. Compared to patients who were unimpaired on the task, this group of patients had significantly higher levels of self-reported hallucinations on the SCales for Outcomes in PArkinson's Disease–Psychiatric Complications and also symptoms of rapid eye movement sleep behavior disorder (RBD). Furthermore, impairment on the BPP was associated with significantly reduced performance on an attentional set-shifting task. Patients with impaired performance on the BPP had higher rates of hallucinations, increased symptoms of RBD, and poorer performance on set shifting, suggesting disrupted processing within the attentional control networks. We propose that the BPP may offer a novel approach for exploring the neural correlates underlying visual hallucinations and misperceptions in PD. © 2012 *Movement* Disorder Society

Key Words: visual hallucinations; misperception; bistable percept paradigm; Parkinson's disease; attentional control networks

The development of visual misperception and hallucinations occurs in over half of all PD patients with advanced disease[1] and represents a key predictor for the transition to institutional care.[2] Visual misperception represents the failure to successfully integrate stimuli that have been physically presented, whereas hallucinations occur where there is perception in the absence of a clear stimulus. The pathophysiological mechanisms underlying these neuropsychiatric symptoms in PD remain poorly understood, and current therapies offer only limited benefits.

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Previous work has suggested that hallucinations are likely to arise from a range of pathologies,[3] implicating widespread regions, including the retina,[4] cortical and subcortical regions,[5–7] as well as dopaminergic and cholinergic neurotransmitter systems.[8,9] Visual hallucinations in PD are often comorbid with rapid eye movement (REM) sleep behavior disorder (RBD), where patients act out their dreams during sleep, and this association has led to the proposal that symptoms may be caused by the intrusion of REM-like sleep imagery into waking consciousness.[10]

A number of researchers have suggested a key role for perceptual and attentional deficits[11–15] in the development of visual hallucinations. Extending this work, a recent hypothesis has proposed that visual misperception and hallucinations in PD arise from dysfunction within the attentional control networks.[16] Specifically, this model suggests that there is a relative inability to recruit activation in the dorsal attention network (DAN) in the presence of an ambiguous percept. This network is comprised of widespread regions

TABLE 1. Demographics, Questionnaires and Neuropsychological Assessment of BPP Impaired and BPP Normal Patients

	BPP impaired	BPP normal	*t* Value	*P* Value
Descriptives				
Number	23	22		
Age, yr	67.3 ± 8.3	60.1 ± 9.1	1.64	0.108
Disease duration, yr	9.0 ± 5.8	5.6 ± 7.8	1.63	0.111
H & Y, stage	2.5 ± 0.9	2.0 ± 0.8	1.96	0.056
UPDRS-III	32.3 ± 16.8	27.1 ± 16.1	1.06	0.296
Dopa dose equivalent (mg/day)	644.3 ± 354.3	542.0 ± 430.1	0.87	0.391
MMSE	26.7 ± 3.2	27.8 ± 2.9	−1.23	0.225
MoCA	22.9 ± 5.4	25.0 ± 3.9	−1.52	0.135
BDI-II	9.6 ± 6.5	11.5 ± 9.7	−0.76	0.450
Outcome measures				
Hidden (%)	64.5 ± 14.4	76.8 ± 11.7	−6.10	0.000***
Single (%)	68.7 ± 19.7	85.8 ± 12.8	−5.12	0.000***
Misperceptions (%)	23.6 ± 19.2	7.3 ± 6.2	4.87	0.000***
Missed images (%)	14.3 ± 7.6	10.6 ± 6.5	4.14	0.000***
Predicted impairments				
SCOPA-$PC_{1–4}$	1.8 ± 2.4	0.6 ± 1.2	2.23	0.033*
RBDQ	6.8 ± 3.8	3.6 ± 2.5	3.42	0.002**
TMT_{B-A}	86.8 ± 38.3	36.7 ± 21.5	−3.64	0.002**

H & Y - Hoehn and Yahr stage; UPDRS-III - Unified Parkinson's DIsease Rating Scale, Motor Subsection; MMSE - Mini Mental State Examination; MoCA - Montreal Cognitive Assessment; BDI-II - Beck Depression Inventory II; SCOPA-$PC_{1–4}$ - Scales for Outcomes in Parkinson's Disease - Psychiatric Complications, Subsection 1–4; RBDQ - Rapid Eye Movement Sleep Behaviour Disorder Questionnaire; TMT_{B-A} - Trail Making Test, Part B minus Part A. Results and statistics are from independent samples t-tests with assumption of unequal variance where appropriate.
Significance levels: *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.

in the dorsolateral prefrontal cortex, the posterior parietal cortices, and the head of the caudate nucleus and is thought to be critical for directing attention and encoding neural signals related to the behavioral significance of a stimulus.[17] This failure to engage the DAN would then lead to an over-reliance on the default mode network (DMN), which consists of regions normally involved in the retrieval and manipulation of episodic memories and semantic knowledge[18,19] and the ventral attention network (VAN), which normally assists in the rapid reorienting of attention toward salient stimuli.[20]

The attentional network hypothesis of visual misperception and hallucinations is clearly rudimentary, but does allow for some empirical testing by the use of targeted cognitive paradigms. Previous studies have utilized selected cognitive testing batteries and novel behavioral approaches to investigate visual hallucinations across a range of clinical conditions.[21–23] Though currently available paradigms are able to identify some neuropsychiatric symptoms in PD, the detailed examination of the specific cognitive deficits underlying such symptoms is still at an early stage. In this study, we assessed the utility of a novel bistable percept paradigm (BPP) to objectively evaluate the attentional network hypothesis. The BPP is a computer-based task that requires participants to process visual stimuli and records whether information is being correctly or incorrectly interpreted. We predicted that impaired performance on the BPP would be associated with an increased prevalence of self-reported hallucinations and symptoms of RBD. Furthermore, we anticipated that patients with poor performance on the BPP would have more difficulty performing tasks that require rapid attentional set shifting resulting from a relative inability to activate the DAN.

Patients and Methods

Participants

The 45 patients with PD and 18 age-matched controls included in this study were all recruited from the Brain and Mind Research Institute PD Research Clinic. All patients satisfied the United Kingdom Parkinson's Disease Society Brain Bank criteria and were assessed on their regular medication. Demographic details are presented in Table 1. Permission for the study was obtained from the local research ethical committee, and all patients gave written informed consent.

All patients underwent assessment in their ''on'' state. Patients were all rated as between H & Y stages I to IV and were assessed on section III of the UPDRS (UPDRS-III). Four patients were untreated, whereas 31 were taking levodopa, including 15 who were on L-dopa alone, 9 who were taking L-dopa combined with entacapone, 10 who were taking dopamine agonist monotherapy, and 7 who were taking a combination of L-dopa and dopamine agonist therapy. Six of the patients had also undergone previous bilateral

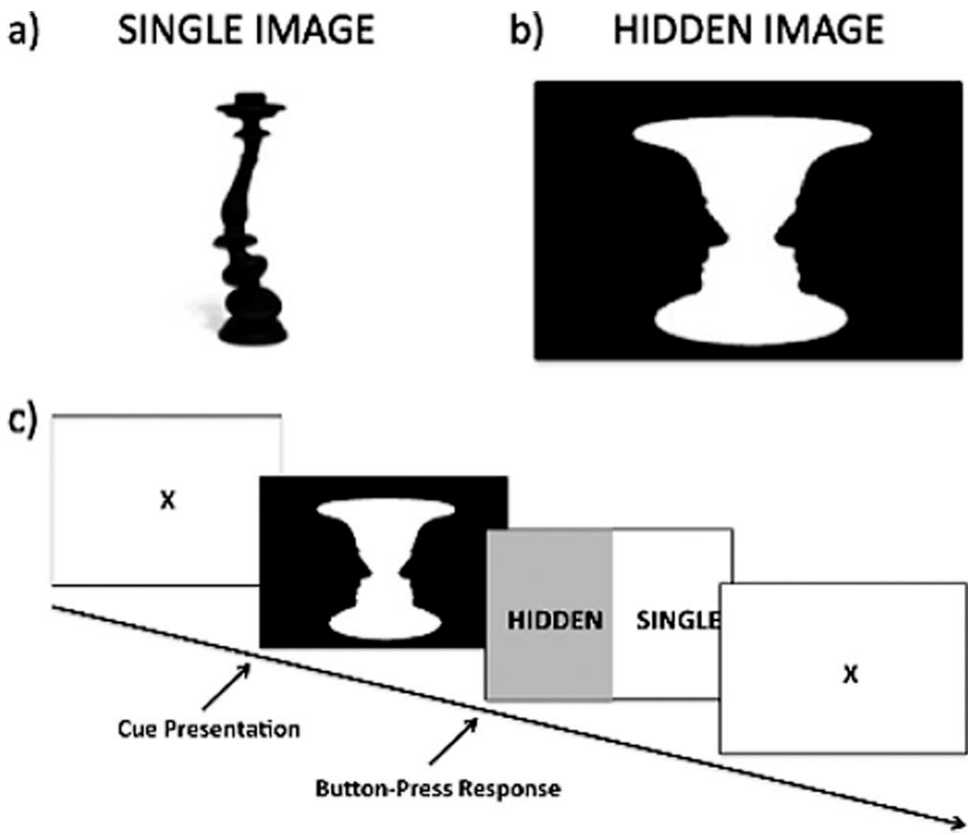

FIG. 1. An example of the images presented to the patients in the BPP. (**A**) Single image of a warped candlestick (in black). (**B**) Hidden image containing a vase (in white) and the sillhouette of two faces (in black). (**C**) Graphic depiction of the experimental paradigm.

STN-DBS. Eleven patients were taking a selective serotonin reuptake inhibitor, and 4 patients were taking a tricyclic antidepressant for mood. Two patients were taking melatonin, and another 6 were taking a nocturnal benzodiazepine to aid sleep.

BPP

The paradigm was constructed using *EPrime* software (Psychology Software Tools, Sharpsburg, PA) and was conducted with the patient sitting in front of the screen with left and right hands positioned over corresponding response buttons that controlled both the initial response to the cue as well as the answers to subsequent questions. Participants were allowed to wear spectacles, if required, during behavioral testing. All trials were conducted indoors in the same laboratory room under standard lighting conditions to control for luminance.

All participants were trained on the paradigm before commencing the study. Images from the training period were not presented again during the task. Each trial was signaled by the appearance of a black fixation crosshair in the middle of a white screen. After a variable delay of between 50 and 100 msec, the crosshair disappeared and participants were presented with a monochromatic black-and-white image. Images represented either ''single'' images or ''bistable percepts'' (i.e., ''hidden'' images as shown in Fig. 1A,B, respectively). Single and bistable images were presented in a randomized order. Participants were required to study the image until they were satisfied that they had recognized everything that the image may represent (i.e., decide whether they were looking at either a single or hidden image) before pressing a response button. This response triggered a screen where participants indicated by button press whether they had identified a single or hidden image. After this button press, the fixation crosshair reappeared, signaling the start of the next trial (see Fig. 1C for a graphical depiction of the task).

After demonstrating familiarity with the BPP, participants performed two separate trials of the paradigm, each including a sample of 20 single images and 20 hidden images. Button responses were logged, and participants also described the specific objects that they saw in each image aloud to the examiner; however, they were not given any feedback during the testing phase.

Primary outcome measures included the following: (1) correct response on ''hidden'' images, recorded as instances when the subject correctly identified a bistable percept; (2) correct response on ''single'' images, recorded as instances when the subject correctly identified a single percept; (3) ''misperceptions'', recorded as instances when the subject incorrectly identified a single percept as a bistable precept or incorrectly reported an image that was not present in either a single or bistable

percept; and (4) ''missed'' images, recorded as instances when the subject incorrectly identified a bistable percept as a single percept.

BPP Error Score

An error score was calculated by averaging the percentage of missed images and misperceptions. The average and standard deviation (SD) of this error score was calculated for the cohort of age-matched controls. In keeping with widespread practice in neuropsychological testing, a cut score was then defined as the average value for the control cohort plus 1.5 SDs. Using this cut score, the group of 45 subjects with PD was split into those with an error score below the cut value (i.e., ''BPP normal'') and those with an error score above the cut value (i.e., ''BPP impaired'').

Neuropsychological Assessments

Performance data are included in Table 1. None of the patients showed evidence of clinical dementia,[24] and the Montreal Cognitive Assessment (MoCA) and the Mini Mental State Examination (MMSE) were used as general measures of cognition.[25] Depressive symptoms were recorded using the Beck Depression Inventory Second Edition (BDI-II).[26] To assess for the presence of visual hallucinations and misperceptions, patients were assessed by a semistructured interview, utilizing the SCales for Outcome in PArkinson's Disease–Psychiatric Complications (SCOPA-PC). This questionnaire contains a subsection of four questions (SCOPA-$PC_{1–4}$) that specifically query the presence of visual misperception and hallucinations, as well as probing for the presence of delusional thinking.[27] Sleep quality was assessed using the Rapid Eye Movement Sleep Behavior Disorder Questionnaire (RBDQ),[28] because these symptoms have been shown to be associated with the development of hallucinations.[29]

To explore the role of attentional set shifting, all patients performed the Trail Making Test (TMT) Parts A and B.[30] In keeping with previous studies exploring the specific component of attentional set shifting, we calculated a difference score (TMT_{B-A}).[31] This measure is a well-described neuropsychological measure of attentional set-shifting impairment and has also been shown to correlate with resting-state functional MRI activity within important hubs of the DAN, such as the posterior parietal cortices.[32]

Statistical Analysis

All data analysis was performed using Statistical Package for the Social Sciences software (*version 16*; SPSS, Inc., Chicago, IL). A one-way analysis of variance was used to test for differences between the three groups in age and depressive symptoms (using scores on the BDI-II). After separating the PD patients into two groups based on their BPP error score, independent-sample *t* tests were used to test for differences between BPP impaired and BPP normal PD patients.

Results

BPP Error Score

The control group had an average BPP error score of 7% (SD 2.67). As such, the cut score was defined at 11% (1.5 SDs above the mean BPP error score for controls). There were 23 PD patients with a BPP error score above and 22 PD patients below this cut score.

Demographic Information

The three groups did not differ significantly, in terms of age EG – (F 1.538; *P* 0.223) or depressive symptoms, as measured by the BDI-II (F 1.774; *P* 0.178). As shown in Table 1, the patient groups (i.e., BPP impaired and BPP normal) did not differ on their disease duration, MMSE, MoCA, dopamine dose equivalence, motor severity (UPDRS-III score), or H & Y stage.

Between-Group Differences on the BPP

Overall analysis of the BPP paradigm showed that there was a significant effect of group on all BPP measures (Table 1), as follows:

1. Patients classified as BPP impaired by their BPP error score were significantly less accurate at correctly identifying bistable percepts than the BPP normal patients (*t* 6.10; *P* 0.001).
2. BPP impaired patients were significantly less accurate at correctly identifying single images than BPP normal patients (*t* 5.12; *P* 0.001).
3. BPP impaired patients were significantly more likely to misperceive a stimulus than BPP normal patients (*t* 4.87; *P* 0.001).
4. BPP impaired patients were significantly worse than BPP normal patients when comparing the rates of missing images (*t* 4.14; *P* 0.001).

Neuropsychological Assessments

As shown in Table 1, compared to BPP normal patients, BPP impaired patients had significantly higher rates of neuropsychiatric symptoms on the SCOPA-$PC_{1–4}$ (*t* 2.23; *P* 0.033) and significantly more self-reported RBD (*t* 3.42; *P* 0.002). In addition, BPP impaired patients had significantly poorer performance than BPP normal patients on the TMT_{B-A}, a reliable measure of attentional set shifting (*t* –3.41; *P* 0.002).

Discussion

The results presented here suggest that the novel BPP is capable of accurately distinguishing the

behavioral performance of those patients who have developed visual misperception and hallucinations in PD. Despite being matched on a number of key measures, including disease duration, motor severity (UPDRS-III), dopamine dose equivalence, depressive symptoms (BDI-II), and two broad measures of cognition (e.g., the Mini–Mental State Examination [MMSE] and MoCA), performance on the BPP clearly delineated two patient groups that were phenotypically characterized by their level of hallucinations, symptoms of RBD, and impaired attentional set shifting.

There is a current paucity of tests that can accurately probe for the presence of visual hallucinations in PD. Recently, the *Movement Disorders Society Task Force on Parkinson's Disease Rating Scales* found that current popular scales were not optimal to identify and track patients with hallucinations.[33–35] This suggests that novel paradigms are warranted for the ongoing study of these symptoms in PD, and currently, there are very few practical tests that have been developed to accomplish this task.[36,37] Attempting to resolve this problem, one study compared the performance of PD patients with and without visual hallucinations, along with age-matched controls, on a number of tests probing visual imagery, visual perception, and memory.[34] The results of the study revealed that PD patients with visual hallucinations had intact visual imagery processes and spatial perception; however, they did demonstrate impairments in object perception and recognition memory. These results suggest that PD patients with visual hallucinations suffer from the faulty perceptual processing of environmental stimuli, although the study explored performance on a number of tests, making extrapolation to the underlying neural deficiencies more difficult.

Performance on the BPP was strongly correlated with self-reported visual hallucinations, as evaluated by the SCOPA-PC questionnaire, which has been shown to accurately assess for the presence of psychotic features in patients with PD.[38,39] Performance on the BPP was also significantly associated with the self-reported presence of RBD, a symptom that has previously been shown to correlate strongly with the presence of hallucinations in PD.[37]

The group of PD patients who displayed worse performance on the BPP was also significantly more likely to suffer from deficiencies in rapid attentional set shifting. This disruption in attentional processing is in keeping with a number of the previously reported studies that highlighted the role of attention and perceptual impairments in parkinsonian hallucinations. One such study showed that impairment of object and space perception in PD patients with visual hallucinations, possibly in association with a decreased sustained visual attention, might play a role in pathogenesis.[38] In addition, though the recognition of objects appears intact in PD patients with visual hallucinations, they are significantly slower in image recognition than in patients without these symptoms, a finding that is not simply explained by executive dysfunction.[22] These findings suggest that both image-recognition speed and sustained attention decline in PD and are exacerbated by the onset of visual hallucinations.

The deficiency in rapid attentional set shifting was also a specific prediction of the recently proposed attentional network model of visual hallucinations in PD.[16] This model suggests that a failure to properly recruit the DAN in the presence of an ambiguous percept may trigger visual misperception and hallucinations. The DAN specifically subserves executive functions, and a recent functional MRI study has reported that resting-state activity within important hubs of the DAN is correlated with attentional set-shifting performance, as assessed by the TMT_{B-A}.[32] Therefore, the impaired performance recorded on the TMT_{B-A} by patients classified as BPP impaired in this study suggests a critical role for the DAN in the pathophysiology underlying hallucinations.

Participants did not undergo formal assessment of contrast discrimination in this study, so it is not clear whether this factor influenced behavioral performance on the task. Reduced visual information processing and retinal pathology has been associated with the occurrence of hallucinations in PD[13,14] and would be in keeping with previous pathophysiological models that have highlighted the key role of perceptual difficulties.[6,12,39] Thus, future studies utilizing the BPP would benefit from a more detailed assessment of visual perception to help account for this potential contribution.

Conclusion

The combination of this novel paradigm, in conjunction with functional neuroimaging, may allow for empiric testing to help understand the pathophysiology underlying visual misperceptions and hallucinations in PD. ■

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