Multi-Modal Hallucinations and Cognitive Function in Parkinson's Disease

Heather Katzen^a Connie Myerson^b Spiridon Papapetropoulos^a Fatta Nahab^a
Bruno Gallo^a Bonnie Levin^{a, b}

^aDepartment of Neurology, University of Miami, Miller School of Medicine, Miami, Fla., and ^bDepartment of Psychology, University of Miami, Coral Gables, Fla., USA

Key Words

Cognition • Hallucinations • Neuropsychiatry • Neuropsychology • Parkinson's disease

Abstract

Background/Aims: Hallucinations have been linked to a constellation of cognitive deficits in Parkinson's disease (PD), but it is not known whether multi-modal hallucinations are associated with greater neuropsychological dysfunction. **Methods:** 152 idiopathic PD patients were categorized based on the presence or absence of hallucinations and then were further subdivided into visual-only (VHonly; n = 35) or multi-modal (VHplus; n = 12) hallucination groups. All participants underwent detailed neuropsychological assessment. **Results:** Participants with hallucinations performed more poorly on select neuropsychological measures and exhibited more mood symptoms. There were no differences between VHonly and VHplus groups. **Conclusions:** PD patients with multi-modal hallucinations are not at greater risk for neuropsychological impairment than those with single-modal hallucinations.

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Introduction

Hallucinations are a common nonmotor symptom of Parkinson's disease (PD), occurring in as many as 44% of patients [1]. Hallucinations in PD may be attributable to multiple factors including pharmacotherapy, disease pathology, depression, cognitive impairment, and sleep-wake cycle disturbances [1–5]. Research has shown that the presence of hallucinations is a risk factor for poor clinical outcomes such as morbidity, nursing home placement and cognitive decline [6].

Visual images are the most common subtype of hallucination reported in PD [7]. However, other hallucinatory experiences may also occur. While a significant portion of PD patients report their experiences within a single sensory modality, approximately 30% of patients have multi-modal hallucinations. The majority of these patients describe primary visual hallucinations accompanied by experiences within a second modality including auditory, olfactory, somatic or gustatory phenomenon [7, 8]. Efforts to provide a unifying pathophysiology of hallucinations in PD have described a 3-factor model of aberrant gating which includes varying degrees of: (1) impairment in internal levels of activation and alert-

ness; (2) disturbances in visual processing, and (3) disruption in the interaction between activation and sensory input [9].

The majority of studies have shown that PD patients with hallucinations exhibit greater cognitive deficits than those patients without hallucinations [2]. What is not known is whether PD patients who experience hallucinations in more than one modality (visual-plus) are more cognitively impaired than individuals whose hallucinations are limited to a single type (visual-only). One study utilized a brief cognitive screening instrument and found that PD patients with visual and auditory hallucinations did not have greater cognitive impairment than patients with only visual disturbances; however, no studies have employed comprehensive neuropsychological evaluation to examine this question [8]. Even less is known regarding the effects of multi-modal hallucinations on emotion. Understanding the differences between these groups may have important clinical implications in terms of treatment planning and clinical management.

The purpose of this study was to first examine differences in neuropsychological and emotional functioning between patients with and without visual hallucinations and then to determine whether patients with multi-modal hallucinations are at greater risk for cognitive and emotional dysfunction.

Methods

Patients

Patients were recruited from the University of Miami Movement Disorders Clinic. All patients were undergoing a pre-surgical evaluation for possible deep brain stimulation treatment and were screened by a specialist in movement disorders. Each patient underwent a comprehensive neurologic evaluation including a modified Hoehn and Yahr rating for staging of illness [10].

Inclusion criteria included a diagnosis of idiopathic PD based on UK PD Brain Bank criteria [11], an age range between 40 and 79 years and a minimum of at least an 8th grade education (the last grade in the US education system before high school, usually contains 13–14-year-olds). All patients also had to have completed the neuropsychological test battery and hallucinations questionnaire to be included in the present study. Exclusion criteria included a history of drug or alcohol abuse, major psychiatric disorder, major medical illnesses (e.g. MI, CABG, CHF, type I diabetes, transplant), neurological illness other than PD (e.g. AD, epilepsy) or prior neurosurgical intervention. One hundred and fifty-two consecutive patients met inclusion/exclusion criteria.

Participants were classified into groups based on the presence or absence of visual hallucinations. One hundred and five participants (69%) were identified as non-hallucinators, as defined by no history of visual or any other type of hallucination (noH). Forty-seven (31%) patients were classified as visual hallucinators

(VH), as defined by a history of one or more episodes of visual hallucinations currently or in the past. The VH group was then sub-divided into those with a history of only visual hallucinations (VHonly; $n = 35$) and those with visual hallucinations plus another modality (VHplus; $n = 12$). Of the VHplus group, 6 had VH plus auditory, 3 had VH plus somatic/cutaneous, 1 had VH plus olfactory and 2 had VH plus auditory and somatic hallucinations. Demographic and disease characteristics of these groups are shown in table 1.

Procedures and Data Collection

The protocol was approved by the University of Miami, Miller School of Medicine institutional review board. Written informed consent was obtained and each patient underwent a comprehensive interview and neuropsychological evaluation. Subjects were evaluated in the Division of Neuropsychology at the University of Miami and were assessed in their primary language [English ($n = 102$), Spanish ($n = 48$) or Creole ($n = 2$)].

Neuropsychological Examination

The neuropsychological battery consisted of tests shown to be clinically and empirically sensitive to the spectrum of cognitive functions known to be compromised in PD [12]. We assessed the domains detailed below.

Premorbid Intellectual Functioning. National Adult Reading Test [13] (NART-eVIQ).

Language. Boston Naming Test [14] (BNT), Controlled Oral Word-Association Test [15] (FAS/Animals).

Memory. California Verbal Learning Test, Second Edition [16]: total words recalled (CVLT-II-total), short-delay free recall (CVLT-II-sf), and long-delay free recall (CVLT-II-lf); Benton Visual Retention Test [17] (BVRT).

Visuospatial. Ghent Embedded Figures Test [18] (Ghent); Hooper Visual Orientation Test [19] (HVOT); Judgment of Line Orientation Test [20] (JLO, 15-item version).

Attention and Executive. Wechsler Adult Intelligence Scale-Third Edition [21]: Digit Span (WAIS-III-DS) and Similarities (WAIS-III-Sim); Symbol Digit Modalities Test [22] (SDMT); Oral Trail-Making Test [23, 24]: trail A time (OTMT-Time A) and trail B time (OTMT-Time B); Paced Auditory Serial Addition Task [25]: 3-second delay condition (PASAT-3 s) and 5-second delay condition (PASAT-5 s); Wisconsin Card Sorting Test – modified version [26]: total categories (mWCST-cat), and perseverative errors (mWCST-persev).

Mood and Affect. Beck Depression Inventory, Second Edition [27] (BDI-II); Beck Anxiety Inventory [28] (BAI); Apathy Evaluation Scale [29] (AES).

Hallucinations Questionnaire. The University of Miami Parkinson's Disease Hallucinations Questionnaire (UM-PDHQ) was used to assess the presence and quality of hallucinations. This instrument has been used in prior studies to assess and characterize hallucinations in PD and is described in detail elsewhere [7]. Type and number of hallucination modalities (e.g. visual, auditory, cutaneous), the number of images, the frequency of experiences, and level of distress were quantified using this examiner administered scale.

Medication Data. Medication data was collected from participants' medical records. The percentage of patients in each group that were prescribed agents in the following categories were recorded and are shown in table 2: levodopa, dopamine agonists,

Table 1. Demographic/disease characteristics and neuropsychological test data for each of the groups

	Group comparisons							
	no history (n = 105)	vs.	visual hallucina- tions (n = 47)	p	visual only (n = 35)	vs.	visual plus (n = 12)	p
<i>Patient demographics</i>								
Age at exam, years	64.6 ± 9.0		65.6 ± 8.9	0.54	66.3 ± 8.7		63.5 ± 9.4	0.35
Education, years	14.1 ± 3.8		14.0 ± 3.7	0.95	14.1 ± 3.8		14.0 ± 3.3	0.93
Male/female, n	73/32		29/18	0.34	22/13		7/5	0.78
Handedness (R/L/ambi), n	98/6/1		40/5/2	0.21	30/3/2		10/2/0	0.54
Age at onset, years	54.8 ± 9.8		53.9 ± 9.8	0.64	54.8 ± 8.8		51.3 ± 12.5	0.37
Disease duration, years	9.9 ± 4.9		11.7 ± 5.8	0.05*	11.5 ± 6.0		12.3 ± 5.3	0.69
Disease severity, H&Y	2.3 ± 1.2		2.3 ± 0.7	0.97	2.2 ± 0.7		2.8 ± 0.5	0.20
MMSE	27.2 ± 2.5		26.5 ± 3.1	0.15	26.9 ± 2.5		25.4 ± 4.2	0.28
<i>Neuropsychological measures</i>								
NART-eVIQ	105.2 ± 8.5		109.9 ± 9.5	0.13	112.9 ± 7.6		101.7 ± 10.4	0.08
WAIS-III								
Digit span	15.0 ± 4.2		14.2 ± 5.0	0.27	14.3 ± 5.0		13.8 ± 5.2	0.76
Similarities	20.9 ± 6.0		20.0 ± 6.6	0.61	19.8 ± 6.9		20.5 ± 6.0	0.80
Boston Naming Test	50.1 ± 8.3		49.6 ± 7.8	0.74	50.0 ± 7.5		48.5 ± 9.1	0.65
Symbol Digit Modalities Test	28.4 ± 12.4		24.2 ± 11.9	0.09	23.3 ± 11.2		27.6 ± 14.2	0.37
FAS	34.0 ± 12.6		32.7 ± 13.8	0.73	34.7 ± 10.4		28.6 ± 19.3	0.46
Animals	15.8 ± 4.9		14.2 ± 6.4	0.13	14.4 ± 4.6		13.5 ± 10.7	0.71
Judgment of Line Orientation	10.3 ± 2.8		8.4 ± 4.4	0.02*	7.8 ± 4.3		9.8 ± 5.2	0.37
Benton Visual Retention Test	9.9 ± 2.8		8.8 ± 3.2	0.28	8.9 ± 3.3		6.6 ± 3.4	0.86
Hooper Visual Orientation Test	20.8 ± 4.8		20.7 ± 5.1	0.89	20.4 ± 5.2		21.9 ± 5.1	0.42
Ghent Embedded Figures Test	34.0 ± 1.8		33.8 ± 2.9	0.92	33.5 ± 2.9		34.4 ± 3.0	0.60
CVLT-II								
Total for trials 1–5	37.4 ± 11.5		33.8 ± 11.2	0.18	35.0 ± 10.6		30.3 ± 12.8	0.29
Short delay free recall	6.9 ± 3.4		5.9 ± 2.8	0.18	6.4 ± 2.6		4.4 ± 3.1	0.08
Long delay free recall	7.2 ± 3.5		6.6 ± 3.1	0.48	7.1 ± 2.7		5.1 ± 4.0	0.18
OTMT								
Time for trial A	10.3 ± 5.8		11.0 ± 3.3	0.56	12.2 ± 3.4		8.9 ± 2.2	0.08
Time for trial B	38.0 ± 17.1		36.2 ± 14.77	0.77	33.8 ± 12.7		41.0 ± 19.4	0.46
PASAT								
3-second trial	32.9 ± 9.5		28.8 ± 13.3	0.35	30.0 ± 12.1		27.4 ± 16.0	0.77
5-second trial	36.2 ± 9.5		28.1 ± 13.3	0.04*	27.7 ± 11.6		28.6 ± 16.6	0.92
mWCST								
Categories	4.1 ± 2.4		2.2 ± 2.3	0.01**	2.0 ± 2.0		2.5 ± 3.0	0.74
Perseverative errors	4.6 ± 5.8		12.1 ± 9.7	0.005**	12.6 ± 8.5		11.0 ± 13.3	0.80
Beck Depression Inventory II	11.5 ± 7.5		15.8 ± 10.4	0.005**	14.9 ± 10.6		18.9 ± 9.5	0.29
Beck Anxiety Inventory	15.3 ± 9.9		19.3 ± 12.7	0.05*	18.0 ± 12.8		23.1 ± 12.2	0.30
Apathy Evaluation Scale	29.7 ± 10.4		35.5 ± 10.5	0.14	38.0 ± 11.2		28.7 ± 4.5	0.21

Data are means ± SD or frequencies, as appropriate. Categorical variables were analyzed by χ^2 . n varies depending on the measure, the number provided is group maximum.

MMSE = Mini-Mental State Examination; NART-eVIQ = National Adult Reading Test-estimated Verbal Intelligence Quotient; WAIS-III = Wechsler Adult Intelligence Scale, Third

Edition; FAS = Controlled Oral Word Association Test-phonemic; Animals = Controlled Oral Word Association Test-semantic; CVLT-II = California Verbal Learning Test, Second Edition; OTMT = Oral Trail Making Test; PASAT = Paced Auditory Serial Addition Task; mWCST = Modified Wisconsin Card Sort Test. * p ≤ 0.05; ** p ≤ 0.01.

Table 2. Medication data for each of the groups

	No history vs. visual hallucinations		Pearson χ^2	p	Visual only vs. visual plus		Pearson χ^2	p
	noH (n = 105)	VH (n = 47)			VHonly (n = 35)	VHplus (n = 12)		
Levodopa	86 (81.9)	40 (85.1)	0.235	0.63	31 (88.6)	9 (75.0)	1.298	0.25
Dopamine agonist	54 (51.4)	27 (57.4)	0.472	0.49	22 (62.9)	5 (41.7)	1.641	0.20
NMDA rec. agonist	31 (29.5)	10 (21.3)	1.121	0.29	8 (22.9)	2 (16.7)	0.204	0.65
Anticholinergics	8 (7.6)	3 (6.4)	0.074	0.79	3 (8.6)	0	1.099	0.30
COMT inhibitor	45 (42.9)	13 (27.7)	3.178	0.08	9 (25.7)	4 (33.3)	0.259	0.61
MAO-B inhibitor	13 (12.4)	8 (17.0)	0.587	0.44	6 (17.1)	2 (16.7)	0.001	0.97
Cholinesterase inhibitor	2 (1.9)	1 (2.1)	0.008	0.93	0	1 (8.3)	2.980	0.08
Antipsychotics	3 (2.9)	4 (8.5)	2.362	0.12	3 (8.6)	1 (8.3)	0.001	0.98
Antidepressants	25 (23.8)	15 (31.9)	1.100	0.29	9 (25.7)	6 (50.0)	2.426	0.12
Statins	12 (11.4)	1 (2.1)	3.591	0.06 [†]	1 (2.9)	0	0.350	0.55

Values are frequency on medication (percentage of group).

NMDA = N-methyl-D-aspartate; COMT = catechol-O-methyl transferase; MAO = monoamine oxidase. [†] $p \leq 0.06$ (statistical trend).

N-methyl-D-aspartate receptor antagonists, anticholinergics, catechol-O-methyltransferase inhibitors, monoamine oxidase type B inhibitors, cholinesterase inhibitors, antipsychotics, antidepressants, statins.

Statistics

Groups (VH vs. noH; VHonly vs. VHplus) were compared on demographic characteristics, disease variables and medication usage. Independent sample *t* tests and Pearson χ^2 analyses were employed for continuous variables and categorical variables, respectively. Analysis of covariance (ANCOVA) and analysis of variance (ANOVA) were employed to compare groups on the neuropsychological and affective measures.

Results

VH versus noH

Comparison of the VH and noH groups revealed that the VH group had a longer disease duration [$t(150) = -1.962$, $p = 0.05$]; no other differences were found on any demographic or disease variable. No differences were observed between groups with regard to medication usage; however, there was a trend toward greater statin usage among the noH group ($p = 0.06$). Given the difference observed between groups in disease duration, this variable was used as a covariate in further analyses between the VH and noH groups. The VH group performed significantly worse than the noH group on select visuospatial measures [JLO: $F(1, 91) = 5.551$, $p = 0.02$] and executive tasks including working memory [PASAT-5 s:

$F(1, 32)$, $p = 0.04$] and set shifting and maintenance [mWCST categories: $F(1, 26) = 7.801$, $p = 0.01$; mWCST perseverative errors: $F(1, 26) = 9.581$, $p = 0.005$]. The VH group also endorsed greater depressive and anxious symptomatology [BDI: $F(1, 138) = 8.277$, $p = 0.005$; BAI: $F(1, 122) = 3.859$, $p = 0.05$]. All group comparisons are shown in table 1.

VHonly versus VHplus

No differences were observed between the VHonly and VHplus groups on any demographic or disease variables or in medication usage. No group differences were observed on any neuropsychological or affective measure. All group comparisons are shown in table 1.

Discussion

Our findings are consistent with previous work demonstrating that patients with hallucinations exhibit greater cognitive deficits compared to patients without a history of hallucinations. Prior studies, which have largely focused on visual hallucinations, have yielded mixed results; some showing widespread cognitive changes [4], and others demonstrating impairments in specific domains such as memory, executive and visuospatial skills [30–34]. This variability may in part be attributed to differences in test selection, educational levels, and medication usage.

We addressed many of these potential confounds by employing a comprehensive neuropsychological battery in well-educated non-demented PD patients with comparable medication usage. Patients with visual hallucinations exhibited difficulty formulating spatial judgments and demonstrated executive dysfunction characterized by working memory and set shifting difficulties. They also reported more depression and anxiety. We did not observe reduced performance in verbal fluency or verbal memory as reported by others [31, 32]. The presence of greater cognitive impairment among hallucinators is consistent with prior work [31, 33] and may reflect structural and functional changes in brain regions involved in higher visual processing [33, 35].

To our knowledge, this is the first study to utilize a comprehensive neuropsychological battery to examine whether patients with multi-modal sensory experiences are at greater risk for cognitive and emotional deficits compared to those with single-modal visual hallucinations. We found no differences in cognitive performance or emotional well-being. Therefore, it appears that the presence of hallucinations in more than one modality is not necessarily associated with a greater risk of either cognitive or affective impairment among PD patients with comparable disease severity and duration [36]. While this study was not designed to examine the neural circuitry underlying single versus multi-modal hallucinators, our findings suggest that if in fact there is more widespread neuroanatomical dysfunction in PD patients

with multi-modal hallucinations, this does not appear to be associated with a picture of greater cognitive impairment.

One limitation of this study is that our sample of multi-modal hallucinators was small, and therefore the power to detect a difference between groups was limited. Additional studies with larger numbers should be conducted to further examine this question. Another shortcoming of this investigation is that while we documented that our PD groups were not prescribed different medications, we did not have sufficiently detailed dose information to calculate LDOPA equivalent dosages.

An important implication of these findings is that clinicians should not expect additional cognitive impairment or emotional disruption in PD patients who present with multi-modal hallucinations above and beyond the deficits observed in patients with single-modal (visual) hallucinations. Furthermore, with regard to clinical management of cognitive symptoms, these findings suggest that PD patients with multi-modal hallucinations do not necessarily require higher dosages of medication.

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