ping, ischemia must be suspected, even without the immediate appearance of clinical symptoms.

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Neuropsychological Deficits in Parkinson's Disease Patients With Visual Hallucinations

Blanca Ramírez-Ruiz, MSc,[1,2]* Carme Junqué, PhD,[1,2] María-José Martí, MD, PhD,[2,3] Francesc Valldeoriola, MD,[2,3] and Eduardo Tolosa, MD[2,3]

[1]Department of Psychiatry and Clinical Psychobiology, University of Barcelona, Barcelona, Spain; [2]Institut d'Investigacions Biomèdiques August Pi i Sunyer (IDIBAPS), Barcelona, Spain; [3]Parkinson's Disease and Movement Disorders Unit, Neurology Service, Institut Clinic de Neurociències, Hospital Clínic Universitari, University of Barcelona, Barcelona, Spain

Abstract: **Recent neuropathological and neuroimaging studies suggest the involvement of several temporal regions in Parkinson's disease (PD) patients with visual hallucinations (VH). We examined 24 nondemented PD patients with VH, 21 PD patients without VH, and 21 healthy controls using a battery of tests assessing different aspects of temporal lobe function. PD patients with VH showed poorer performance in language, verbal learning, semantic fluency, and visuoperceptive functions compared to controls and PD patients without VH. Differences in verbal learning and visuoperceptive functions were independent of general cognitive status, disease severity, and depression. We suggest that a wide range of neuropsychological deficits can contribute to the emergence of VH in PD.** © 2006 Movement Disorder Society

Key words: Parkinson's disease; visual hallucinations; language; memory; visuoperceptive

Visual hallucinations (VH) belong to the most frequent neuropsychiatric symptoms of Parkinson's disease

*Correspondence to: Dr. Carme Junqué, Departament de Psiquiatria i Psicobiologia Clínica, Universitat de Barcelona, IDIBAPS, Casanova 143, 08036 Barcelona, Spain. E-mail: cjunque@ub.edu

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(PD), affecting 22% to 50% of patients.[1,2] Although they have been associated with the presence of cognitive impairment or frank dementia,[1,2] only few studies have addressed specific neuropsychological deficits in nondemented PD patients with VH. Barnes and colleagues[3,4] found that hallucinating PD patients showed deficits in object perception and visual recognition memory in comparison with both nonhallucinating PD patients and healthy controls. Cognitive deficits related to executive functions have also been reported in hallucinating PD patients.[5] In terms of the underlying brain mechanisms, neuroimaging and neuropathological studies found an association between temporal and frontal cerebral abnormalities and VH in PD patients.[6–8] Our study investigates the potential relationship between VH in PD and deficits in different neuropsychological domains, with a particular emphasis on testing of temporal lobe function.

PATIENTS AND METHODS

Subjects

Participants were recruited from the Parkinson Disease and Movement Disorders Unit, Hospital Clinic Universitari, Barcelona, Spain. The sample comprised 24 PD patients who had experienced VH in the previous year, 21 PD patients who never suffered from VH, and 21 healthy elderly controls. The controls comprised spouses of PD patients and community volunteers without any history of psychiatric or neurological disorders who were matched with patients for age and education. All subjects gave informed consent to participate in this study, which was approved by the local Ethics Committee.

Diagnostic Criteria and Clinical Evaluation

The details of the diagnostic criteria and clinical assessment are described elsewhere.[9] Patients who fulfilled Diagnostic and Statistical Manual of Mental Disorders, Revised Fourth Edition, criteria for dementia or presented clinical depression (Hamilton score ≥ 15) were excluded. All patients passed a visual acuity test consisting of reading from a distance of approximately 40 cm.

The demographic and clinical characteristic of the sample (PD+VH; PD; Controls) are, respectively, as follows: age (74.7 $\pm$ 5.4; 73.3 $\pm$ 6.1; 73 $\pm$ 6.7), sex distribution ([males/females] 10/14; 9/12; 9/12), years of education (7.3 $\pm$ 3.4; 7.7 $\pm$ 3.4; 7.9 $\pm$ 4.6), and visual acuity (0.5 $\pm$ 0.2; 0.6 $\pm$ 0.2; 0.7 $\pm$ 0.2). None of these variables differed significantly among the samples. PD patients with VH obtained a lower Mini-Mental State Examination (MMSE; 26.7 $\pm$ 2.1) and a higher Hamilton scores (6.5 $\pm$ 4.6) than both non-VH PD patients (MMSE = 29.2 $\pm$ 1.4; Hamilton = 3.6 $\pm$ 2.7) and healthy controls (MMSE = 29.9 $\pm$ 1.6; Hamilton = 2.7 $\pm$ 2.6) ($P < 0.0005$; $P < 0.05$, respectively). Concerning clinical differences between both PD groups, hallucinating PD patients presented similar motor severity as assessed by the Unified Parkinson's Disease Rating Scale (UPDRS) Motor scale (30.6 $\pm$ 14.5) but a more advanced Hoehn & Yahr stage (3.3 $\pm$ 1.1) compared to nonhallucinating PD patients (UPDRS = 24.9 $\pm$ 13.7; H&Y = 2.5 $\pm$ 0.7) ($P = 0.21$; $P = 0.015$, respectively). No differences in levodopa daily dose were found between PD groups with and without VH ($P = 0.071$).

The VH in our PD sample consisted of well-formed images of people, faces, or animals. Insight into the hallucinatory nature of the phenomenon was maintained in 63% of the patients. Associated delusions were present in 33% of the patients. These delusions were primarily paranoid in type and involved elementary misbelieves concerning infidelity or theft. All hallucinating PD patients but 1 were on dopaminergic treatment at the time of evaluation. A modification of the antiparkinsonian treatment preceding the onset of VH was recorded in 33% of the patients. The VH disappeared after dopaminergic drug dosage reduction in 11 cases (45.8%), whereas 5 patients (20.8%) needed antipsychotic medication and 5 (20.8%) required both interventions. In the remaining patients, medication was not modified because the hallucinations were well tolerated.

Neuropsychological Assessment

General intellectual ability was assessed by the Information and Similarities subtests from Wechsler Adult Intelligence Scale-Third Edition (WAIS-III). Language was evaluated by a verbal comprehension test (Token Test) and a picture naming test (Boston Naming Test) and frontal functions by phonological and semantic fluency tests. A modified version of the Rey Auditory–Verbal Learning Test (RAVLT) was used to assess verbal learning, delayed recall, and recognition. To assess visuoperceptive functions, we used the Benton Facial Recognition Test (BFRT) and the standard drawing and multiple-choice version of the Benton Visual Form Discrimination Test (VFDT).[10] Finally, we evaluated visual memory functions using the subtest Memory for Faces from Warrington's Recognition Memory Test. All tests were administered and scored according to conventional procedures.[11]

Data Analysis

Statistical analysis was carried out using SPSS 11.0. Sex distribution was compared with χ^2 tests. For normally distributed quantitative variables with homogene-

TABLE 1. *Neuropsychological test scores*

	PD with VH (n = 24)	PD without VH (n = 21)	Controls (n = 21)	*P* value[a]	*P* value[b]
General intellectual ability					
Similarities (WAIS-III)	11.7 ± 4.8	12.7 ± 6.0	14.0 ± 5.1	NS	—
Information (WAIS-III)	11.5 ± 6.9	12.4 ± 6.1	12.9 ± 7.3	NS	—
Language					
Token Test	27.8 ± 4.4	30.6 ± 2.8	31.9 ± 3.0	<0.05**	NS
Boston Naming Test	47.3 ± 7.0	51.8 ± 4.3	52.5 ± 5.4	<0.05**	NS
Verbal memory					
RAVLT-learning	24.8 ± 7.2	38.8 ± 7.0	41.9 ± 7.2	<0.0005**	<0.0005
RAVLT-memory loss	8.4 ± 10.8	3.7 ± 4.4	3.0 ± 2.7	0.043*	—
RAVLT-correct recognition	12.5 ± 2.1	12.8 ± 2.1	13.7 ± 1.4	NS	—
RAVLT-false recognition	2.4 ± 2.3	1.3 ± 1.3	0.9 ± 0.8	0.007*	—
Executive functions					
Phonological fluency (P)	7.4 ± 4.5	9.7 ± 3.7	12.7 ± 6.1	0.002*	—
Semantic fluency (animals)	9.4 ± 4.5	13.3 ± 4.7	17.1 ± 5.1	<0.05**	NS
Visuoperceptive functions					
Benton Facial Recognition Test	43.7 ± 4.5	49.0 ± 4.3	49.4 ± 2.9	<0.0005**	<0.013
Visual Form Discrimination Test	26.4 ± 3.8	28.8 ± 3.1	30.4 ± 2.0	<0.05**	NS
Visual Memory					
Warrington's Recognition Test	31.1 ± 5.6	33.5 ± 6.5	40.4 ± 5.6	<0.005*	—

[a]Analysis of variance.
[b]Analysis of covariance between PD groups using Mini-Mental State Evaluation, Hoehn & Yahr, and Hamilton scores as covariates.
*Significant difference between PD patients with VH and controls.
**Significant difference between PD patients with VH and controls and between PD patients with VH and PD patients without VH.
PD, Parkinson's disease; VH, visual hallucinations; WAIS-III, Wechsler Adult Intelligence Scale, Third Edition; RAVLT-learning, sum of words correctly recalled from Trials 1 to 5; RAVLT-memory loss, percentage of memory loss: percentage of words loss after 20 min of interference; RAVLT-correct recognition, number of words correctly recognized from the original list; RAVLT-false recognition, number of words falsely recognized from the original list.

ity of variance, we used an analysis of variance (ANOVA test) and the post hoc Bonferroni test. For non-normally distributed variables, and/or in the case of no equality of variance between the groups, we used the nonparametric Mann–Whitney *U* test. Finally, we performed an analysis of covariance (ANCOVA) using MMSE, Hoehn & Yahr stage, and Hamilton scores to determine whether differences between the two PD groups on cognitive tests persisted after controlling for these variables.

RESULTS

The results of the neuropsychological assessment are shown in Table 1. The groups did not differ in general intellectual ability as evaluated with the Similarities ($P = 0.343$) and Information ($P = 0.777$) subtests from WAIS-III. However, the ANOVA showed group differences in the other domains assessed: language, memory, frontal, and visuoperceptive functions. The post hoc test revealed poorer performance in PD patients with VH than in healthy controls in all neuropsychological measures except RAVLT-recognition. Patients with VH were more impaired than those without VH on language assessed by Token and Boston Naming tests ($P = 0.034$ and $P = 0.032$, respectively), verbal learning ($P < 0.0005$), semantic fluency ($P = 0.022$), and visuoperceptive functions evaluated by Benton Facial Recognition ($P < 0.0005$) and Visual Form Discrimination ($P = 0.042$). The only significant difference between PD patients without VH and healthy controls was observed in visual memory ($P = 0.001$).

Given that the specific neuropsychological deficits may be related to the global cognitive performance (MMSE score) and to the differences in illness stage (Hoehn & Yahr scale) and mood (Hamilton scale), we performed an ANCOVA between the two PD groups using these clinical variables as covariates. After removing the effect of these variables, differences in RAVLT-verbal learning ($P < 0.0005$) and in Benton Facial Recognition Test ($P = 0.013$) remained significant.

DISCUSSION

PD patients with VH showed several cognitive deficits affecting not only visuoperceptive performance, as previously reported, but also language and verbal memory domains. This finding suggests that cerebral dysfunction in PD with VH extends beyond the subcortical–frontal circuits typically described in PD.

On tests of verbal memory, PD patients with VH demonstrate the classic PD pattern, consisting of deficits in free recall but normal recognition.[12] However, in addition, our patients with VH showed deficits in long-term retention (RAVLT-memory loss) and the presence of false intrusions. These findings could reflect an involvement of limbic and temporal cortex, as suggested by neuropathological studies,[7] in addition to a disruption of nigro-striato-thalamo-cortical circuitry.

The differences on Benton's Facial recognition test in the hallucinating patients are in agreement with the neuropsychological[4] as well as neuroimaging findings suggesting involvement of visual associative areas in patients with VH.[6,8] These differences are not likely to be caused by a lower visual acuity, although we cannot exclude a faulty visual input due to primary visual deficits commonly seen in PD, such as reduced color and contrast discrimination.[13] Differences in BFRT remained significant after controlling for MMSE. In contrast, the differences between hallucinating and nonhallucinating PD patients on the VFDT lost significance after such covariation. The performance on VFDT, in addition to visuoperceptive skills involved in BFRT, requires additional visuospatial functions. This finding could explain why this test depends to a higher degree on the general cognitive status.

Regarding the face recognition memory, our results are partially in agreement with those reported by Barnes and associates (2003) that found a significant impairment in PD patients with and without VH vs. healthy controls. Nevertheless, we did not observe significant statistical differences on the Warrington Facial Recognition test between hallucinating and nonhallucinating groups, although PD patients with VH scored lower than those without this symptom. Longitudinal studies can clarify the relevance of visual memory impairment on cognitive decline associated with the presence of hallucinations.

In addition to complex visual dysfunction, we observed deficits on language comprehension. The poor performance on the Token Test could be related to an impairment in sentence processing,[14] which has been associated with a reduced activation of anteromedial prefrontal cortex and posterolateral temporal regions.[15] Additionally, the reduced performance on the Token Test could also be related to a visual attention impairment, as alterations of parieto-frontal connections associated primarily with the attentional modulation of visual perception have been implicated in the pathophysiology of VH.[8]

The recruitment of inferior frontal and ventral temporal regions have been observed during confrontation naming and semantic fluency tasks in healthy subjects.[16,17] The deficits found in these cognitive domains in our hallucinating patients, therefore, could reflect dysfunction in these brain areas. From the clinical point of view, naming impairment has been related to the level of general cognitive dysfunction in PD[12] and prospective studies have identified deficits in verbal fluency as predictors of the development of dementia.[18] This finding is consistent with our finding that the differences between the PD groups in naming and category fluency lost significance once the effects of general cognitive status were removed from the analysis. The reduced efficiency in language and semantic fluency in our PD patients with VH could reflect the starting point of the more general cognitive decline which might later lead to dementia.

In conclusion, hallucinating PD patients were characterized by a wide range of impairments affecting several cognitive domains. The presence of such extensive deficits may escape the compensatory processes and, thus, favor the perception of VH. Given the well-documented relationship between dementia and hallucinations, it remains to be shown in longitudinal studies whether visuoperceptive and memory impairments found in our hallucinating PD sample are the earliest symptoms of a progressive cognitive decline.

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OFF–*off* Rebound Dyskinesia in Subthalamic Nucleus Deep Brain Stimulation of Parkinson's Disease

Matthew A. Brodsky, MD,* Penelope Hogarth, MD, and John G. Nutt, MD

Department of Neurology, Oregon Health & Science University, and Parkinson's Disease Research, Education & Clinical Care Center, Portland VA Medical Center, Portland, Oregon, USA

Video 

Abstract: **A 61-year-old man with Parkinson's disease (PD), motor fluctuations, and dyskinesias underwent bilateral implantation of deep brain stimulation (DBS) electrodes in the subthalamic nucleus (STN). One month after surgery, DBS was optimized to bilateral monopolar settings at the most proximal electrode just superior to the STN, which improved motor fluctuations and dyskinesias. At several postoperative evaluations off medications overnight, both stimulators were turned off and within 60 seconds he developed severe dyskinesias. When the stimulators were turned back on, the dyskinesias soon resolved. This article is a first report of a unique pattern of rebound-type dyskinesia that occurred in the *off* medication state produced by stopping STN DBS.** © 2006 Movement Disorder Society

Key words: Parkinson's disease; dyskinesia; deep brain stimulation

Deep brain stimulation (DBS) for Parkinson's disease (PD) has become a widely accepted treatment for patients who suffer from disabling dyskinesia or motor fluctuations and have reached an endpoint of further improvement with medical therapy. DBS improves all of the cardinal signs of PD, including muscle rigidity, bradykinesia, and tremor.[1] Furthermore, an improvement in levodopa-induced dyskinesias is often seen after DBS surgery of the subthalamic nucleus (STN).[2] It is not certain whether this improvement is due to a decrease in L-dopa dose permitted by the improvement in parkinsonian symptoms[2] or a direct effect of the stimulation itself.[3] Direct stimulation of the STN has also been demonstrated to *induce* dyskinesia.[4] Although DBS of the STN has been reported to both increase or decrease dyskinesia while a patient is *on* medications, we report a case in which dyskinesias were induced by turning off STN stimulation when the subject was pharmacologically *off*.

CASE REPORT

A 61-year-old right-handed man with PD developed symptoms of left-sided muscle rigidity and bradykinesia 15 years before DBS surgery. He initially was treated with selegiline, with modest benefit. He subsequently was started on L-dopa/carbidopa 12 years before surgery and responded with an improvement in his PD symptoms. Ten years before surgery, a dopamine agonist was added to his drug regimen. Seven years before surgery, he began to develop motor fluctuations and dyskinesias. Over the subsequent 7 years, these motor complications progressively became more disabling. In the year before surgery, he was spending approximately 10% of his waking hours in the *on* state without disabling dyskinesias. During the month before surgery, his medication regimen was as follows: carbidopa/L-dopa 25/100, 2.5 tablets every 3 hours 8 times per day (around the clock); entacapone 200 mg with each dose of carbidopa/L-dopa; pramipexole 1.5 mg three times per day (t.i.d.); and

This article includes Supplementary Video, available online at http://www.interscience.wiley.com/jpages/0885-3185/suppmat

*Correspondence to: Dr. Matthew A. Brodsky, Parkinson Center of Oregon, Oregon Health & Science University, 3181 SW Sam Jackson Park Road, Mailcode OP-32, Portland, OR 97239.
E-mail: brodskym@ohsu.edu

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