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Neuropsychological Impairment, Depression, and Parkinson's Disease

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Abstract: *Depression, a frequently cited concomitant of Parkinson's disease (PD), can result in the overestimation of the frequency and severity of PD-related neuropsychological deficits. In this study, 89 nondemented and 19 demented PD patients, as determined by Mini-Mental State Examination (MMSE) scores, were significantly more depressed than 64 controls on two depression scales. There were significant, negative associations between scores on the Geriatric Depression Scale and performance on 8 neuropsychological test variables. Analysis of covariance was used to statistically control for level of depression, as well as age and education. Both PD groups were significantly impaired on 7 neuropsychological test variables, including measures of visuomotor, memory, and executive functions. The demented PD group was more impaired than the nondemented PD and control groups on 9 neuropsychological test variables. Cognitive impairments in the nondemented PD group were relatively subtle and not apparent on the MMSE. Depression remains a potential confound, but it is unlikely to account for all of the neuropsychological deficits associated with PD. A model for the neuropsychological examination of PD patients is discussed.*

Keywords: Neuropsychological impairment, depression, Parkinson's disease.

James Parkinson's original description of the movement disorder that bears his name stated that the "senses and the intellect remain uninjured" (Parkinson, 1817). Although controversy remains as to the exact nature of cognitive decline in Parkinson's disease (PD; Boller, 1980), there is an emerging consensus that it can occur in a substantial number of PD patients (Cummings & Benson, 1988; Lishman, 1987; Mayeux, 1989; Pillon, Dubois, Lhermitte, & Agid, 1986; Raskin, Borod, & Tweedy, 1990; Taylor, Saint-Cyr, & Lang, 1988).

Opinion has diverged regarding the form of cognitive deterioration in PD. Many investigators describe a slowing of cognition, or *bradyphrenia* (Rogers, Lees, Smith, Trimble, & Stern, 1987), with accuracy of cognitive function remaining relatively undisturbed. Some investigators have suggested that the cognitive slowing is directly related to the motor

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slowing commonly reported in PD (Rogers, 1986), whereas others have found cognitive and motor slowing in PD to be unrelated in terms of their responsivity to L-dopa therapy (Pillon et al., 1989).

Cognitive abilities associated with the frontal lobes are frequently reported to be specifically disrupted in PD. These functions have been variously defined as including mental flexibility or the ability to shift cognitive set (Sandson & Albert, 1987; Raskin et al., 1990), word fluency (Gotham et al., 1988), or the ability to generate self-directed, efficient strategies in problem solving (Taylor, Saint-Cyr, & Lang, 1986).

Visuospatial cognitive impairments are occasionally found in PD (Hovestadt, de Jong, & Meerwaldt, 1987). Some investigators have suggested that difficulties in this area are actually manifestations of frontal lobe deficits (e.g., organization, planning, and problem solving; Raskin, Tweedy, & Borod, 1990). Others have indicated that visuospatial deficits are artifacts of impaired motor functioning (i.e., many measures of visuospatial skills currently in use are both timed and require manipulation of test materials; Girotti, Soliveri, Carella, Geminiani, Aiello, & Caraceni, 1988).

Memory dysfunction is widely reported in the PD literature. The memory impairment in PD has been described as a deficit in retrieval of information already consolidated in memory rather than in acquisition of new information (Brown & Marsden, 1988; Cummings & Benson, 1988). Conversely, other researchers report acquisition memory deficits in PD, which they attribute to slowed cognitive processes that are unable to keep pace with stimulus presentation (Huber, Shulman, Paulson, & Shuttleworth, 1989; Riklan, Reynolds, & Stellar, 1989; Sagar, Sullivan, Gabrieli, Corkin, & Growdon, 1988).

Some investigators have suggested that memory decline occurs only in a subgroup of PD patients. A study by Huber, Shuttleworth, and Paulson (1986) demonstrated that PD patients who scored above a dementia cut-off on the Mini-Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975) failed to demonstrate the selective memory impairments seen in demented PD patients.

Depression, a frequent concomitant of PD, is a potential source of error variance in cognitive testing and could result in an overestimation of cognitive deterioration (Raskin, Borod, & Tweedy, 1990). The co-occurrence of depression and PD is quite high, suggesting an association between the two disorders. Estimates of the prevalence of depression in PD patients range from 39% (Mayeux, Stern, Cote, & Williams, 1984) to 90% (Mindham, 1970).

A number of investigators believe that depression in PD is a direct effect of the disease, resulting from various neurochemical and neuroanatomical changes (Agid et al., 1986; Mayeux, 1989; Rogers et al., 1987; Sano et al., 1989; Santamaria, Tolosa, & Valles, 1986). Others report that depression in PD is an understandable emotional reaction to the disabling nature of the disease and is unrelated to organic processes (Bieliauskas & Glantz, 1989; Bieliauskas, Klawans, & Glantz, 1986; Taylor, Saint-Cyr, Lang, & Kenny, 1986).

One of the most common referral questions encountered in clinical practice is the differentiation of cognitive dysfunction caused by neurological disease from a "pseudodementia" attributable to depression (Salzman & Gutfreund, 1986). Depression in otherwise healthy individuals is frequently associated with impaired performance on neuropsychological tests, particularly among the elderly (Raskin, 1986; Savard, Rey, & Post, 1980). Weingartner (1986) proposed that the neuropsychological impairments seen on a wide array of measures in depressed individuals are caused by a failure to sustain effortful, as opposed to automatic, cognitive processing. Niederehe (1986) suggested that depression entails a reduction in the self-initiation of behaviors, rather than an inability to complete them. Thus, the increased frequency of depression in PD might result in further psychomotor slowing, or decreased initiation, or both, with consequent artifactual overestimation of the

level of cognitive impairment.

In the present investigation, we attempted to clarify the nature and extent of cognitive impairment in PD and to assess the degree to which this impairment may derive from depressed mood. We divided the PD group into those who obtained MMSE scores greater than or equal to 26 (nondemented PD) and those whose scores were less than 26 (demented PD). We compared both PD and the normal control groups on two self-report measures of depression, and we examined the association between depression level and neuropsychological test performance in all patients. We then compared both PD groups' and the normal control group's performances on a battery of neuropsychological tests, statistically controlling for level of depression, as well as age and education, through analysis of covariance (ANCOVA).

The neuropsychological test battery contained measures of global cognitive functioning, visuomotor speed, speed of cognitive processing, concept formation and novel problem solving, set shifting, immediate and delayed verbal recall, immediate visual recall, verbal learning with cued retrieval, and visuospatial abilities. We were able to factor out the motor components from one of the measures to obtain a relatively uncontaminated estimate of set shifting and speed of cognitive processing.

We hypothesized that both PD groups would be more depressed than the control group and that there would be a positive relationship between level of depression and level of neuropsychological impairment. We hypothesized that both PD groups would manifest significant cognitive impairments, relative to controls, in spite of being covaried on level of depression. Furthermore, we hypothesized that the demented PD group would be more impaired than the nondemented PD group. Finally, we hypothesized that the impairments exhibited by both PD groups would be particularly evident with respect to frontal lobe functions and speed of processing.

Method

Participants

A total of 108 patients enrolled in a PD treatment and rehabilitation program participated in the study. Sixty-four (15 men, 49 women) of their spouses and caregivers served as the control group. The mean age of participants in the control group was 66.44 years ($SD = 9.44$), and the mean years of education were 14.14 ($SD = 2.0$). Participants in the nondemented PD group (51 men, 38 women) had a mean age of 69.35 years ($SD = 8.48$) and an average of 14.05 years of education ($SD = 2.81$). The mean age of the demented group (16 men, 3 women) was 74.94 years ($SD = 3.51$); they averaged 11.91 years of education ($SD = 0.83$).

Participants' age when symptoms first appeared and when PD was first diagnosed was determined from medical records and the admission interview by the medical director (Gerald Jogerst). The mean age of first appearance of symptoms was 61.39 years ($SD = 10.10$) for the nondemented PD group and 68.12 years ($SD = 9.31$) for the demented PD group. Mean duration of symptoms was 6.81 years ($SD = 5.18$) for the nondemented PD group and 8.31 years ($SD = 6.24$) for the demented PD group. Mean age of first diagnosis was 64.53 years ($SD = 8.68$) for the nondemented PD group and 68.12 years ($SD = 10.16$) for the demented PD group. Mean duration since diagnosis was 4.70 years ($SD = 4.36$) for the nondemented PD group and 8.20 years ($SD = 6.71$) for the demented PD group.

All members of both PD groups were receiving therapeutic levels of antiparkinsonian medication. With respect to disease severity, the two PD groups ranged across all levels of Hoehn and Yahr's (1967) scale. The modal Hoehn–Yahr score for both PD groups was Stage 3 (i.e., mild to moderate bilateral disease; some postural instability; physically independent).

Procedure

The patients and controls were examined during the first week of their participation in a 4-week therapeutic program. All tests were administered in the standard format. The test battery included two self-report depression scales to measure depression—the Geriatric Depression Scale (GDS; Yesavage et al., 1983) and the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961).

The MMSE (Folstein et al., 1975) was administered as a measure of general cognitive integrity. Attentional processes were assessed with the Mental Control subscale from the Wechsler Memory Scale–Revised (WMS-R; Wechsler, 1987), in which subjects are instructed to recite the alphabet and perform serial addition and subtraction tasks. Also administered was the WMS-R Visual Memory Span subscale, in which subjects are required to tap out sequences of blocks immediately after presentation, both forward and in reverse order.

Two measures of verbal memory were administered, the Logical Memory and Verbal Paired Associates subscales from the WMS-R (Wechsler, 1987). The Logical Memory subscale requires subjects to repeat two paragraphs from memory both immediately after a single presentation and after a 30-min delay. The Verbal Paired Associates subscale consists of three learning trials, in which subjects are presented with four related and four unrelated word pairs. Subjects are then required to give the second word after being cued with the first.

Visuospatial constructional and sequencing abilities were evaluated with the Block Design and Picture Arrangement subscales, respectively, from the Wechsler Adult Intelligence Scale–Revised (WAIS-R; Wechsler, 1981). Visual scanning and psychomotor speed were measured with the Trail Making Test (Parts A and B; United States War Department, 1944). Part A requires individuals to use a pencil to connect numbered dots on a piece of paper as quickly as they can. Part B is similar, with the exception that subjects are required to alternate back and forth between numbers and letters, thus adding a set shifting or divided attention component to the task.

Two motor-free measures of frontal lobe function (i.e., set shifting and novel problem solving) were included in the battery. The set-shifting component of the Trail Making Test was isolated from the visual scanning and motor components by subtracting the time (number of seconds) required to complete Part A from the time required to complete Part B. The resulting variable is a measure of mental-set-shifting speed, relatively uncontaminated by visuomotor task requirements. The Booklet Category Test (DeFilippis & McCampbell, 1979) was also included in the battery. It requires subjects to generate and test hypotheses and to make cognitive shifts when the conceptual nature of the task changes.

Results

To determine whether or not there actually was a negative association between depression and neuropsychological test performance, we calculated Pearson correlation coefficients between each of the two depression scales and each of the neuropsychological test variables (see Table 1). For the GDS, there were significant ($p < .01$) negative associations between test performance and depression level on 8 of the 12 neuropsychological test variables, but for the BDI, only one correlation was significant.

We then performed two independent ANCOVAs examining the level of depression on both depression scales for the three experimental groups, (normal controls, nondemented PD patients, and demented patients), covarying for age and years of education (see Table 2).

Table 1
Pearson Correlations Between Depression Scales and Neuropsychological Test Performance

Test	*N*	Geriatric Depression Scale	Beck Depression Inventory
Mini-Mental State Examination	141	−.20*	−.05
Booklet Category Test	118	.29**	.08
Trail Making Test			
Part A	117	.32**	.11
Part B	116	.37**	.11
Part B − Part A	115	.23*	.09
Wechsler Memory Scale–Revised			
Mental Control	95	−.12	.01
Logical Memory			
Immediate recall	101	−.30*	−.04
Delayed recall	102	−.13	−.01
Verbal Paired Associates	62	−.15	−.07
Visual Memory Span	68	−.04	.08
Wechsler Adult Intelligence Scale–Revised			
Picture Arrangement	113	−.35**	−.15
Block Design	113	−.37**	−.22*
Geriatric Depression Scale	142	—	.52**
Beck Depression Inventory	142	.52**	—

*$p < .01$. **$p < .001$.

There were significant effects for both the GDS and the BDI, with the GDS accounting for more of the variance between groups. Univariate Tukey B post hoc analyses indicated that the nondemented and the demented PD groups were both more depressed than the control group on the GDS, but not the BDI.

We also calculated correlations between each depression scale and duration since onset of symptoms, duration since first diagnosis, and the Hoehn–Yahr (1967) stage-of-illness score. There was a significant association between GDS and Hoehn–Yahr scores ($r = .32, p < .01$), indicating that increased depression was associated with the more advanced stages of illness. Correlations between GDS and durations since onset of symptoms and first diagnosis were not significant. The BDI did not correlate significantly with any of these measures.

In the second phase of our study, we conducted a multivariate analysis of covariance (MANCOVA) examining cognitive test performances in the three experimental groups. Of the two depression scales, the GDS score accounted for the greatest portion of intergroup variance, and therefore it was chosen as the covariate for level of depression. Age and education were also covaried. We did not include the MMSE in this analysis because we used it as a criterion for group membership. We were unable to include WMS-R Verbal Paired Associates score in the MANCOVA because of empty cells in the design. Furthermore, only 50 subjects were included in the MANCOVA because not all subjects were tested on all variables. In spite of the reduced power of our design, the overall effect for group was significant (Hotelling's $T = 1.511$), approximate $F(18, 70) = 2.94, p < .001$.

Table 2
Summary of Analyses of Covariance

Test	*F*	*df*	α	η^2
Beck Depression Inventory[a]	4.88	2, 105	.009	.08
Geriatric Depression Scale[a]	5.94	2, 105	.004	.14
Booklet Category Test[b]	6.34	2, 88	.003	.25
Trail Making Test[b]				
Part A	24.42	2, 87	.001	.44
Part B	8.71	2, 86	.001	.31
Part B – Part A	1.27	2, 85	.286	.10
Wechsler Memory Scale–Revised[b]				
Mental Control	3.97	2, 72	.023	.19
Logical Memory				
Immediate recall	2.48	2, 76	.091	.17
Delayed recall	3.46	2, 77	.037	.16
Verbal Paired Associates	1.42	2, 45	.252	.12
Visual Memory Span	5.21	2, 50	.009	.23
Wechsler Adult Intelligence Scale–Revised[b]				
Picture Arrangement	4.81	2, 82	.011	.23
Block Design	8.56	2, 82	.001	.28

[a] Age and years of education are covariates.
[b] Age, years of education, and Geriatric Depression Scale score are covariates.

Table 2 presents the summary statistics for the ANCOVAs examining performance on each of the neuropsychological test variables in the three experimental groups, with age, education, and depression level covaried. Eight of 11 neuropsychological test variables were significantly different across the three groups ($p < .05$). The 3 measures that were not significantly different were the set-shifting component of the Trail Making Test (Part B – Part A) and the Verbal Paired Associates and Logical Memory (immediate recall portion) subscales of the WMS-R. The proportions of variance accounted for (η^2) by group membership for the 8 significantly different variables ranged from a high of .44 for Part A of the Trail Making Test to a low of .16 for the delayed recall portion of the Logical Memory subscale of the WMS-R.

Table 3 presents means, standard deviations, and subsample sizes across the three experimental groups for each of the neuropsychological test variables. Also presented in Table 3 are the results of the univariate post hoc Tukey B analyses. On seven of the eight neuropsychological test variables that were significantly different, the nondemented PD group was more impaired than the normal control group, and the demented PD group was more impaired than both the control and the nondemented PD groups ($p < .05$). The only significantly different test variable that did not show this pattern was the WMS-R Mental Control subscale, on which only the demented PD group was more impaired than the control group ($p < .05$).

Discussion

Our results suggest the following: (a) Depression in PD patients and normal controls was negatively related to performance on neuropsychological tests; (b) both demented and

Table 3
Mean Scores and Standard Deviations on Depression Scales and Neuropsychological Tests

Test	Normal controls			Nondemented PD group			Demented PD group		
	n	*M*	*SD*	*n*	*M*	*SD*	*n*	*M*	*SD*
Geriatric Depression Scale[ab]	59	9.41	6.31	70	12.31	5.70	13	15.38	9.46
Beck Depression Inventory	59	8.10	5.85	70	8.97	6.64	13	8.62	8.01
Mini-Mental State Examination[ac]	61	28.87	1.40	89	28.91	1.27	19	21.53	3.52
Booklet Category Test (no. of errors)[abc]	41	64.00	31.00	84	85.81	32.02	18	131.72	21.71
Trail Making Test (seconds for completion)									
Part A[abc]	41	43.05	33.45	83	71.71	45.30	18	241.67	128.12
Part B[abc]	41	108.85	78.24	82	210.62	134.50	18	401.67	53.61
Part B – Part A	41	65.80	64.08	81	135.90	117.86	18	160.00	115.84
Wechsler Memory Scale–Revised									
Mental Control[ac]	40	4.80	1.40	64	4.67	1.59	14	1.86	1.41
Logical Memory									
Immediate recall	39	23.26	9.19	71	17.63	9.98	16	3.00	4.66
Delayed recall[abc]	39	14.33	9.22	71	9.61	8.60	16	3.00	4.66
Verbal Paired Associates	35	16.46	3.65	38	14.92	3.82	1	8.00	0.00
Visual Memory Span[abc]	27	12.63	3.91	47	10.11	5.14	12	2.50	4.60
Wechsler Adult Intelligence Scale–Revised									
Picture Arrangement									
(age-corrected scaled score)[abc]	35	9.63	2.35	84	6.56	3.41	18	3.56	2.87
Block Design									
(age-corrected scaled score)[abc]	35	9.97	3.06	84	6.96	3.55	18	3.11	1.78

Note. PD = Parkinson's disease.
[a] Demented PD group is more impaired than normal controls ($p < .05$).
[b] Nondemented PD group is more impaired than normal controls ($p < .05$).
[c] Demented PD group is more impaired than the nondemented PD group ($p < .05$).

nondemented PD patients reported more depressive symptoms than do normal controls; (c) level of depression and stage of illness in PD were modestly and positively associated; (d) PD patients manifested a number of neuropsychological impairments, with both PD groups being more impaired than normal controls and with demented PD patients being more impaired than nondemented PD patients; and (e) these cognitive impairments cannot be accounted for entirely by the PD patients' greater depression.

With respect to the specific neuropsychological impairments noted in this study, PD patients differed from controls in terms of (a) scanning and visuomotor speed (Trail Making Test, Parts A and B, and WMS-R Visual Memory Span), which appeared to account for the greatest degree of intergroup variance; (b) visuospatial organization and constructional ability (WAIS-R Block Design); (c) novel problem solving (Booklet Category Test); (d) logical sequencing (WAIS-R Picture Arrangement); and (e) delayed verbal recall (delayed recall portion of the WMS-R Logical Memory subscale). The demented PD group differed from normal controls and the nondemented PD group on a measure of concentration (WMS-R Mental Control). Neither PD group significantly differed from the controls in terms of verbal acquisition (immediate recall score on WMS-R Logical Memory and Verbal Paired Associates) or on the motor-free measure of set-shifting speed (Trail Making Test, Part B – Part A).

Our results suggest that although the two depression scales were significantly correlated with each other, the GDS is a more sensitive measure of depression in this population than the BDI. The greater sensitivity of the GDS probably stems from the fact that it was originally developed specifically for use with geriatric populations (Yesavage et al., 1983), unlike the BDI.

The nondemented PD group was more impaired than the control group on seven of the neuropsychological test variables. This occurred despite the fact both groups performed equivalently on the MMSE. This finding argues against the position that only a relatively small subset of PD patients display cognitive deterioration (Huber et al., 1986). It also suggests that the MMSE is too gross a measure to detect the relatively subtle cognitive deficits associated with PD. The high-level, subtle nature of these cognitive impairments might account for their lack of description in the medical literature until relatively recently, when more sophisticated neuropsychometric measurement paradigms have been used.

Our finding of impairments on measures relatively resistant to motor functioning artifact (e.g., the Booklet Category Test, WAIS-R Picture Arrangement, and the delayed recall portion of the WMS-R Logical Memory subscale) indicates that cognitive abilities do decline as PD progresses. Furthermore, the memory impairments of our PD groups do not appear to be the result of acquisition deficits, as suggested by some investigators (Huber et al., 1989; Riklan et al., 1989; Sagar et al., 1988), but instead seem to reflect patients' inability to retrieve information already consolidated (Brown & Marsden, 1988; Cummings & Benson, 1988). With respect to the neuropathological mechanism responsible for the cognitive declines seen in PD, our findings are consistent with Taylor, Saint-Cyr, and Lang's (1986) hypothesis that functional impairments of the frontal lobes resulting from inhibited outflow from the neostriatal-prefrontal loop are to blame.

Raskin, Borod, and Tweedy (1990) proposed a systematic approach to the assessment of neuropsychological impairment in PD patients. They recommended first assessing global cognitive integrity with a dementia rating scale, then evaluating mood status and depression level and, if these are grossly preserved, administering a neuropsychological test battery that includes measures of attention, memory, visuospatial skills, set shifting, language, and motor functions. Our results suggest that depression may not account for all of the neuropsychological deficits characteristic of PD. Obtaining neuropsychological test data when working with

individuals in this population might therefore be helpful, even if they are depressed. However, as our results also demonstrate, clinicians should recognize that depression remains a potential confound when determining the cognitive impairment of sufferers of PD.

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