

## Cognitive Functioning in Parkinson's Disease: In Relation to Prevalence of Dementia and Psychiatric Diagnosis

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Forty-three neurologically and psychiatrically assessed patients with idiopathic Parkinson's disease (PD) underwent detailed cognitive assessment. Cognitive deficits typical of senile dementia of Alzheimer's type (SDAT) were found in 7% but the majority showed definite impairments not typical of SDAT. Cognitive impairment was significantly more likely in those with more severe PD symptoms. There was substantial agreement between psychiatric diagnosis and psychological picture of SDAT and some links were found between other diagnostic categories and nature of cognitive functioning. However, cognitive deficits were also found in two-thirds of patients with no psychiatric diagnosis.

The prevalence of dementia in Parkinson's disease has been a longstanding subject of speculation and investigation. It is recognised that it is important to have factual information on the issue for both theoretical and practical reasons, but it is also acknowledged that accurate information is extremely difficult to obtain. Recent reviews (Boller, 1980; Mayeux, 1982; Brown & Marsden, 1984) highlight the wide variation in estimates of prevalence from 20% to 81%.

There are a number of factors which may contribute to such a degree of variation, including the definition of dementia adopted, the means of assessing presence of dementia and the composition of the PD sample.

Definitions of dementia adopted in studies of PD patients have varied from an inclusion of all patients with evidence of cognitive impairment to a narrower definition more consistent with the DSM-III criteria for diagnosis of dementia (American Psychiatric Association, 1980). A useful definition is one whose operational criteria are clearly defined and which is narrow enough for useful theoretical conclusions to be drawn, e.g. for the purposes of the present study our interest is in the presence of a defined constellation of deficits seen as being typical of senile dementia of Alzheimer's type (SDAT).

The method of assessment needs to be able to distinguish changes due to dementia from those caused by other factors, including normal ageing, depression, acute confusional states and physical disability, all of which have an increased risk of being present in patients with PD.

Finally, aetiology of PD in the sample assessed needs to be clearly stated. The most useful group to study from a theoretical point of view are patients with idiopathic PD.

### Aim

The central aim of the study reported here was to clarify whether there is an increased risk of patients with idiopathic PD having cognitive deficits typical of senile dementia of Alzheimer's type (SDAT). Secondary aims were to ascertain the proportion of patients with idiopathic PD having cognitive impairments not typical of SDAT; and to examine how the nature and severity of these deficits and impairments related to severity of PD, psychiatric diagnosis and types of treatment being received by subjects. Deficits typical of SDAT were defined as evidence of impairment in secondary memory (shown through impaired performance on a test of secondary memory or on an information/orientation scale); and of a significant decline in general intelligence (shown by a discrepancy of more than one standard deviation between current and pre-morbid intelligence).

### Subjects

All patients with a diagnosis of idiopathic PD attending the Regional Neurological Centre, Newcastle General Hospital, between September 1981 and March 1985 were invited to participate in a research study on complications of PD. Two patients declined but a cohort of 81 was established

and all were assessed by neurologists and psychiatrists over a 3-year period to establish the severity of their PD as well as the presence of any degree of dementia or depression, or other significant psychiatric disorder.

Appointments were sent to 63 patients drawn from the cohort of 67 patients still living, for psychological assessment between July 1984 and March 1985. Twenty did not attend (two refused, five moved away, five had temporary ill health, four because of miscellaneous reasons, four for reasons unknown) and four were not sent appointments for practical reasons e.g. they were living a great distance from the hospital. The attenders comprised 21 women (mean age 63.6 years, range 59–77) and 22 men (mean age 66 years range 53–71 years). Their mean duration of disease was 14 years ( $\pm 13$ ) and ratings on the Webster Parkinson Disability Scale (Webster, 1968) showed that at the time of psychological assessment 18 had mild impairment (Webster 0–9), 20 showed moderate impairment (Webster 10–19) and five showed severe impairment (Webster 20+). Of those with predominantly unilateral symptoms nine had left-sided disabilities and eight had right-sided disabilities.

All patients except one were receiving medication; (31 levodopa preparations; one bromocriptine, two anticholinergic medication and eight a

combined drug therapy). Three had had stereotaxic surgery.

Age-matched control subjects included spouses of patients and attenders at Newcastle Age Concern Pop-In. None had any severe physical disability or history of psychiatric illness.

### Method

All control subjects and 35 patients were seen in a quiet, well lit room. Seven patients were seen at their place of residence and one during a short-stay hospital admission.

Administration of tests took between 45 and 90 minutes and subjects were given time to rest between tests as appropriate. An attempt was made to assess all patients in the areas listed below, with the exception of the visual-spatial imagery test which was administered to a subgroup of 13 patients.

Full descriptions of the tests are not given but brief notes give cogent information on why the tests were chosen for use with this population.

1. *Pre-morbid intelligence: New Adult Reading Test (NART)* (Nelson & O'Connell, 1978). This test has been standardised on British population samples up to age 70 and has proved to give valid results with patients diagnosed as demented.

2. *Current intelligence: Raven's Progressive Matrices (PM)* Standard or Coloured Version, as appropriate (Raven *et al* 1977). This was chosen in preference to the more

TABLE I  
Number of patients with idiopathic PD showing normal and impaired performance on each of the series of tests

Area of cognitive functioning	Test	Criteria for impairment	% (n) results in normal range	% (n) results showing impairment
Intelligence	NART PM	Discrepancy of one s.d. or more between PM and NART	82% (28)	18% (6)
Information/orientation	CAPE I/Q scale	Score $\leq 7$ shown in validation studies to be strong indicator of the presence of dementia	93% (37)	7% (3)
Primary memory	Digit-Span forwards	Below age-matched norms (i.e. $< 5$ )	86% (33)	14% (5)
	Digit-Span backwards	Below age-matched norms (i.e. $< 4$ )	72% (26)	28% (10)
Secondary memory	Two-choice verbal recognition	Below range scores of 12 age-matched control subjects	71% (24)	29% (10)
Retrieval from long-term memory	Benton Word Fluency Test (Total)	a) Below range scores b) Below 25%ile scores c) Above 25%ile scores of 12 age-matched controls	c) 22% (8)	a) 33% (12) b) 78% (28)
	Visuospatial imagery	Block counting test	Below range scores	31% (4)
			12 age-matched controls	69% (9)

TABLE II  
Cognitive performance of 43 patients with idiopathic PD

Cognitive Performance	Number	% of sample
Cognitive deficits typical of SDAT	3	7
Definite evidence of deficits not typical of SDAT	28	65
Question raised of deficits (at least one mildly impaired score)	8	19
No cognitive deficits	1	2
Insufficient data	3	7

commonly used Wechsler Adult Intelligence Scale since movements are not required to complete the items and it is untimed. In addition, British norms are available up to age 89 years.

3. *Knowledge of current information/orientation: Clifton Assessment Procedures for the Elderly (CAPE) Information/Orientation Scale* (Pattie and Gilleard, 1979). This test has been validated against diagnosis of dementia and standardised on British samples over age 60 years.

4. *Primary Memory: Digit span* forwards and backwards. The standard subtest from Wechsler Adult Intelligence Scale (Wechsler, 1958) was used.

5. *Secondary Memory: 2 choice verbal recognition memory* The test used was an extended version of that described by Morris *et al* (1983) and found to be sensitive to memory deficits of patients with mild to moderate SDAT. The recognition format appears to be equally informative, yet it is less threatening to patients than a recall test.

6. *Retrieval from long-term memory: Benton Word Fluency Test* (Benton, 1968).

7. *Visuospatial imagery: modification of block counting test* from Stanford-Binet Intelligence Scale (Terman & Merrill, 1973). Each item requires mental reconstruction of a three-dimensional object from a line drawing and it thus tests an aspect of visuospatial functioning without requiring a motor response.

## Results

All, or the majority of tests, were completed by 36 out of 43 patients with the remainder carrying out such tests as their disabilities and circumstances would allow. Table I shows results with regard to performance on each test and Table II gives the results in a manner related to the aims of the study.

A majority of patients showed deficits on the tests of retrieval from long-term memory (Benton Word Fluency Test) and visuospatial imagery (Block Counting Test).

On the word fluency test significant differences were found between patients and control subjects in the number of words produced on each letter and in total (see Table III). A number of perseverative intrusions (i.e. production

of a word from a previous letter category) were given by 10 (32%) of patients but no control subjects. There was no significant difference in the number of repetitions (i.e. of a word already given) the majority of subjects giving at least one repetitive response.

TABLE III  
Average number of words recalled on each letter and in total on Benton Word Fluency Test

	Letter 1	Letter 2	Letter 3	Total
Age-matched Control subjects (n = 12)	16.4	15.6	15.1	47.1
Idiopathic PD patients (n = 36)	10.4	9.9	9.9	30.3
Significance of difference	$P < 0.01$	$P < 0.02$	$P < 0.01$	$P < 0.001$

On the test of visuospatial imagery control subjects scored significantly higher than the patient group (controls: average 12.5/14; patients: average 7.45/14;  $t = 3.75$ ,  $P < 0.01$ ).

In all other areas assessed, a minority of patients showed impairment in comparison with age-matched norms drawn from British standardisation studies, or control data.

## Relationship of cognitive dysfunction to other measures

A significant, though fairly low, positive correlation was found between length of illness and severity of PD as measured by Webster scale ratings ( $r = +0.35$ ;  $P = < 0.05$ ). Those with mild symptom ratings were significantly less likely to show cognitive impairment than those with moderate symptom ratings—Webster score 0–9: 61% definite cognitive impairment; Webster score 10–19: 95% cognitive impairment;  $\chi^2 = 4.6$ ,  $P < 0.05$ ). Of five patients with severe symptom ratings, two had deficits typical of SDAT and two had severe communication problems which limited assessment. The fifth showed mild deficits. There was a tendency for a greater proportion of patients who had a lengthy history to have more severe cognitive impairment in comparison with those with a short history but this trend was not significant.

As symptoms of PD became more severe there was a tendency for word fluency to be poorer ( $r = 0.3$ ;  $P < 0.1$ ). In unilateral cases, side of symptoms had no influence on cognitive deficits found.

The three patients who had undergone thalatomy all had severely depressed word fluency, an effect which has been shown to occur after bilateral ventral thalamic lesions. Effects of medication on cognitive functioning could not be isolated but two patients with a history of drug-induced confusional states showed specific cognitive deficits indistinguishable in character from the general sample.

The relationship between psychiatric diagnosis and psychological assessment is shown in Table IV. For the purposes of this study patients found at psychiatric

TABLE IV  
*Relationship between psychiatric and psychological assessment of cognitive functioning in 43 patients with idiopathic PD*

Cognitive functioning	Psychiatric diagnosis: number (%) with each degree of cognitive impairment						Insufficient data
	Dementia	Cognitive deficits	Depression	Anxiety	None		
SDAT deficits	2 (100%)	1 (10%)					3 (7%)
Other deficits		9 (90%)	5 (56%)	1 (100%)	12 (63%)		28 (65%) <sup>1</sup>
? deficits			2 (22%)		6 (32%)		8 (19%)
No deficits					1 (5%)		1 (2%)
Insufficient data			2 (22%)			(1)	3 (7%)
	2 (5%)	10 (23%)	9 (21%)	1 (2%)	19 (44%)	1 (2%)	

<sup>1</sup> Includes one patient with diagnosis of combined depression and mild cognitive deficits

assessment to have cognitive impairment are divided into two diagnostic groups. The first includes those with SDAT, the second includes those with mild or doubtful dementia and those given a diagnosis of dementia qualified as not typical of SDAT (mild or atypical cognitive impairment). Where a psychiatric diagnosis of SDAT was made this predicted that the patients would show SDAT deficits at psychological assessment. In all other psychiatric categories, the majority of patients showed specific cognitive impairments, this being also true where there was no psychiatric diagnosis though in this group over 33% were found to have no definite evidence of cognitive impairment.

The nature of cognitive impairment showed some significant differences between diagnostic groups. Those diagnosed at psychiatric assessment as having mild or atypical cognitive impairment showed a significant lowering of intelligence (NART: average 109.25, PM: average 94.625;  $P < 0.01$ ) whereas neither the depressives nor those with no psychiatric diagnosis showed any change in average level of intelligence. Those with a diagnosis of depression produced significantly fewer responses on the third letter of the word fluency test (depressives: average 8.1; non depressives: average 11.4;  $P < 0.05$ ).

However, deficits in information/orientation, secondary memory and total production of words in the word fluency test were equally likely whether psychiatric diagnosis was of mild or atypical cognitive impairment, depression or no disorder.

#### Characteristics of patients not seen

Among those patients who died before they could be psychologically assessed a greater number had severe PD and a greater number received a psychiatric diagnosis of dementia than among the sample seen: 9.3% of all patients given a comprehensive psychiatric assessment received a diagnosis of SDAT with a further 2.7% receiving a diagnosis of dementia (atypical). Among those who did not attend or were not sent an appointment a greater proportion had moderate symptoms of PD and fewer had mild symptoms, but the proportion in the various psychiatric categories of diagnosis did not differ from the sample seen except for a greater number of cases with combined depression and mild cognitive impairment.

## Discussion

### Prevalence of dementia

Deficits typical of SDAT were found in 7% (three) of the current sample. This figure is lower than any estimate reported prior to 1985 except for a rate of 0 found by Patrick & Levy (1922).

Brown & Marsden (1984) present clearly the reasons why figures produced in many past studies may overestimate the prevalence of dementia. When studies of only those with idiopathic PD were examined the reported prevalence rate fell to 24.5%; and a correction for the use of inappropriate assessment measures led them to suggest that the true prevalence may be 15–20%. Less (1985) recently re-worked data in seven studies to include only patients whose diagnosis of dementia complied with DSM-III criteria and produced figures between 7 and 15%.

Thus re-examination of previous studies to correct for composition of sample, criteria for diagnosis and use of inappropriate assessment techniques produces a dramatic lowering in estimated prevalence to a similar level to that found in the prospective study reported here.

Since Brown & Marsden's critique was published, two short reports of equally low figures (15% and 8%) of prevalence for dementia have been given in published letters (Lees 1985; Taylor *et al* 1985). Thus clear evidence is emerging that the true prevalence rate for a clinical picture of dementia of Alzheimer's type in PD is substantially lower than was previously thought.

Two further points concerning these conclusions need to be made. Firstly we re-emphasise that the definition of dementia adopted influences the prevalence rate found. The term dementia, even defined as strictly as in the DSM-III criteria has disadvantages where an attempt is being made to distinguish between different patterns of cognitive

impairment, since it leads to the assembling of a heterogeneous sample of cognitively impaired individuals.

Operational definitions, which group together individuals who are impaired in stated aspects of cognitive functioning are more informative in this respect, and may clarify distinctions in this situation between patients who at psychiatric assessment appear to have SDAT and those who are felt to have an 'atypical' dementia.

Secondly, in this study despite using an operational definition of cognitive impairment we have tied our definition to a neurological level of explanation by using the term SDAT which implies the presence of certain cognitive pathology. The underlying pathology, however, cannot be assumed since identical cognitive impairments may be due to differing underlying pathology. Perry *et al* (1985) recently found that the pathological changes in a group of clinically demented PD patients differed from those in patients having SDAT in the absence of PD.

We therefore conclude that a lower proportion of PD patients have impairments typical of SDAT than was previously thought; but that the level of prevalence is nevertheless above that expected in the population of this age. Kay *et al* (1970) reported a prevalence rate of 2.4% among their population sample aged 65–69 years. This increased level of prevalence does not necessarily imply an increased risk among PD patients of developing SDAT but may indicate that a number of patients show cognitive deficits usually associated with SDAT but in their case consequent upon other cortical changes that may occur as a complication of PD.

#### Prevalence of other types of cognitive impairment

The results of the present study emphasise that many PD patients with no global dementia face difficulties in cognitive functioning. Taylor *et al* (1985) and Lees & Smith (1983) also found that a majority of patients with idiopathic PD showed subtle cognitive deficits not amounting to dementia. Risk of cognitive dysfunction appeared to increase to some degree with length and severity of illness, confirming some past findings (reviewed by Mayeux, 1982) but being contrary to the view recently put forward by McCarthy *et al* (1985) that there is correspondence between incidence of cognitive deficits in early and late PD. This issue may only be clarified by a longitudinal study of a patient cohort.

#### Interlink between psychiatric and psychological assessment

The results of this study suggest that where a psychiatric diagnosis of dementia has been made, the

patient is also likely to show SDAT deficits at psychological assessment. However, in all other cases, degree of cognitive dysfunction is almost independent of psychiatric diagnosis. This is such that the patient who receives no psychiatric diagnosis is almost as likely to show definite impairments as the patient who receives a diagnosis of affective disorder or mild or atypical cognitive impairment.

The dissociation, however, is not complete since the nature of the cognitive dysfunction may vary in some respects. Where a psychiatric diagnosis of mild or atypical cognitive impairment is made it seems that the psychiatrist is responding to a decline in general intelligence in the absence of clear-cut memory impairment, but more subtle impairments in word fluency and secondary memory may pass unnoticed. It should therefore be useful during psychiatric assessment of a patient with PD to include a simple test of word fluency in the absence of a full psychological assessment of cognitive functioning.

Where a patient is depressed there may be typical cognitive deficits as a result of the depression but these do not account for all the impairment found among this diagnostic group.

#### Conclusions

Results are now emerging from a number of sources, including the present prospective study, to suggest that the prevalence of deficits typical of SDAT in PD is lower than was previously thought, affecting only 5–15% of patients. More subtle forms of cognitive impairment may be much more common but can be missed during psychiatric examination. Their nature and course needs to be clarified through longitudinal follow-up and clearly designed psychological investigations.

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#### References

- AMERICAN PSYCHIATRIC ASSOCIATION (1980) *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.) DSM-III. Washington DC: Division of Public Affairs, APA.
- BENTON, A. L. (1968) Differential behavioural effects of frontal lobe disease. *Neuropsychologia*, **6**, 53–60.
- BOLLER, F. (1980) Mental status of patients with Parkinson's disease. *Journal of Clinical Neuropsychology*, **2**, 157–172.
- BROWN, R. G. & MARSDEN, C. D. (1984) How common is dementia in Parkinson's disease? *Lancet*, **ii**, 1262–1265.

- KAY, D. W. K., BERGMANN, K., FOSTER, E. M., McKECHNIE, A. A. & ROTH, M. (1970) Mental illness and hospital usage in the elderly: a random sample followed up. *Comprehensive Psychiatry*, **11**, 26–35.
- LEES, A. J. (1985) Parkinson's disease and dementia, *Lancet*, **1**, 43–44.
- & SMITH, E. (1983) Cognitive deficits in the early stages of Parkinson's disease, *Brain*, **106**, 257–270.
- MAYEUX, R. (1982) Depression and dementia in Parkinson's disease. In *Butterworths International Medical Reviews, Neurology 2: Movement Disorders* (eds C. D. Marsden & S. Fahr). London: Butterworth Scientific.
- MCCARTHY, R., GREY, M. & FINDLEY, L. J. (1985) Parkinson's disease and dementia, *Lancet*, **1**, 407.
- MORRIS, R., WHEATLEY, J. R. & BRITTON, P. G. (1983) Retrieval from long-term memory in senile dementia: Cued recall revisited, *British Journal of Clinical Psychology*, **22**, 141–142.
- NELSON, H. E. & O'CONNELL, A. (1978) Dementia: the estimation of premorbid intelligence levels using the New Adult Reading Test. *Cortex*, **14**, 234–244.
- PATRICK, H. T. & LEVY, D. M. (1922) Parkinson's disease: A clinical study of 146 cases, *Archives of Neurology and Psychiatry*, **7**, 711–720.
- PATTIE, A. H. & GILLEARD, C. (1979) *Clifton Assessment Procedures for the Elderly (CAPE)*, Sevenoaks: Hodder & Stoughton.
- PERRY, E. K., CURTIS, M., DICK, D. J., CANDY, J. M., ATACK, J. R., BLOXHAM, C. A., BLESSED, G., FAIRBAIRN, A., TOMLINSON, B. E. & PERRY, R. H. (1985) Cholinergic correlates of cognitive impairment in Parkinson's disease: comparisons with Alzheimer's disease. *Journal of Neurology, Neurosurgery and Psychiatry*, **48**, 413–421.
- RAVEN, S. C., COURT, J. H. & RAVEN, J. (1977) *Manual for Raven's Progressive Vocabulary Scales*, London: H. K. Lewis.
- TAYLOR, A., SAINT-CYR, J. A. & LANG, A. E. (1985) Dementia prevalence in Parkinson's disease, *Lancet*, **1**, 1037.
- TERMAN, L. M. & MERRILL, M. A. (1973) *Manual for Stanford-Binet Intelligence Scale*. Third Revision.
- WEBSTER, D. D. (1968) Critical analysis of the disability of Parkinson's disease, *Modern Treatment*, **5**, 257–282.
- WECHSLER, D. (1958) *The Measurement and Appraisal of Adult Intelligence*, 4th ed, Baltimore: Williams & Wilkins.

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