

Incidental and Intentional Recall in Parkinson's Disease: An Account Based on Diminished Attentional Resources*

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ABSTRACT

The recall of common objects and their spatial location was examined in 65 patients with Parkinson's disease (PD) under conditions in which available attentional resources were manipulated by secondary task demands. PD patients were impaired at item recall particularly under intentional learning conditions but were unimpaired at recall of spatial location. These findings were similar in newly diagnosed, untreated cases as well as patients who had suffered with the disease for an average of 9.6 years. Test performance was not improved by levodopa therapy, despite it benefiting motor control, and was not impaired by anticholinergic medication. Item recall correlated significantly with other memory measures (particularly tasks of working memory) but only weakly with indices of physical disability and traditional frontal-lobe measures. Spatial recall, by contrast, correlated with memory quotient but no other cognitive measure and depression and disease duration failed to correlate significantly with performance on either recall task. These results are attributed to a deficit in attentional resources in PD that impairs performance most markedly for tasks and conditions that make the greatest demands upon effort.

Visuospatial deficits have been reported in PD in the areas of visuoperception, construction, and spatial orientation (Brown & Marsden, 1988a; Sagar & Sullivan, 1988). However, the nature of the psychological processes underlying these impairments and their relationship to motor control remains controversial. In a recent paper Levin et al. (1991) showed that facial recognition, mental object assembly, and visuoconstruction performance declined inevitably with disease duration but that the ability to formulate angular judgements and to identify geometric figures declined only in those patients who became demented. Association between visuospatial test performance and motor control has been dem-

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onstrated in PD (Danta & Hilton, 1975; Mortimer, Pirozzolo, Hansch, & Webster, 1982). Indeed it is suggested that many of the deficits reported are, at least in part, attributable to motor impairments (Stelmach, Phillips, & Chau, 1989). In 1984, however, Boller et al. showed that nondemented PD patients were impaired on tasks of visuoperception even when motor response was minimized. More recently it has been suggested that spatial orientation deficits displayed by PD patients may owe more to impaired set-shifting capacity than to a specific visuospatial disturbance (Brown & Marsden, 1986).

Memory for spatial location has been investigated extensively within neuropsychology in the last fifteen years (although not in PD); in summary these studies have shown that the information is encoded largely without effort by normal subjects (Mandler, Seegmiller, & Day, 1977); that the elderly are unimpaired at spatial recall relative to item recall (McCormack, 1982); that amnesic patients are particularly compromised in processing spatial information (Mayes, Meudell, & MacDonald, 1991; Shoqeirat & Mayes, 1991); and that lesions to the right hippocampus are particularly detrimental to spatial recall (Smith & Milner, 1981). In addition, according to the work of Hasher and Zacks (1979), processes governing memory for spatial location lie towards the automatic end of a continuum of effort-demanding processes; thus, recall of spatial location is relatively unaffected by those factors that influence available attentional resources. Recall of the spatial location of an item can often be achieved despite performance of a secondary task and indeed without any intentional endeavour to remember the information. Memory for the item itself, however, demands a greater degree of mental effort and factors that compete for, or reduce attentional resources (e.g., age, secondary task requirements) will therefore have a significantly adverse effect upon the probability of recall of the item.

In a study of effort-demanding and automatic processes in untreated PD (Weingartner, Burns, Diebel, & LeWitt, 1984) patients were selectively impaired on tasks of free recall (effort-demanding) but were spared in judgement of event frequency and the mode by which stimuli were presented (tasks dependent upon automatic processes). Recall of spatial location was not assessed. These findings are of particular note in light of the prominent dopaminergic deficit in PD and finding by Newman, Weingartner, Smallberg, and Calne (1984) that elderly subjects could benefit from dopaminergic medication on those tasks dependent upon effortful but not automatic processing. Impaired spatial and item recognition has been demonstrated in medicated but not unmedicated PD (Sahakian et al., 1988). Taylor, Saint-Cyr, and Lang (1986) also found a deficit on a delayed recognition test of spatial location in their sample of medicated patients; in this case, however, results were confounded by temporal order task demands.

One unifying hypothesis which may explain differences in performance according to the nature of the spatial task is the attentional resource model, as developed by Brown and Marsden (1988b, 1991). They demonstrated that patients were disproportionately impaired on a Stroop task in the absence of external cues, which otherwise serve to guide response. This profile of performance

was mimicked by normal subjects when they were required to perform a secondary task concurrently with the Stroop. The authors concluded that their results were most compatible with the notion of a deficit in the 'Supervisory Attentional System' (Norman & Shallice, 1980) in PD. Other studies of cognitive impairment in PD have also focused upon attentional deficits (Downes et al., 1989; Morris et al., 1988; Sagar, Sullivan, Gabrieli, Corkin, & Growdon, 1988) which have been attributed to frontal-lobe dysfunction that accompanies the disease (Taylor, Saint-Cyr, & Lang, 1986).

The present study was designed to assess memory for item and spatial location in PD under learning conditions in which the availability of attentional resources was manipulated. A large patient sample was studied to explore the influence of disease duration and specific drug therapy upon performance. Test results were also examined in relation to performance on other tasks of motor control, memory, and executive function.

METHODS

Subjects

Sixty-five PD patients were recruited from the neurology outpatient clinics of the Royal Hallamshire Hospital, Sheffield. All patients fulfilled the diagnostic criteria of the PD Society: akinesia plus rest tremor, rigidity or postural instability and absence of any other condition that may produce signs of parkinsonism. No patient had undergone any neurosurgical operation. Patients were not selected on the basis of any behavioural criteria. These patients were classified into three subgroups: 20 newly diagnosed, untreated patients (*de novo*) were recruited from consecutive referrals; 30 patients who had been recruited into a longitudinal study of cognition in the same way as the *de novo* cases, but had since been randomized to one of three anti-parkinsonian monotherapies: levodopa (Madopar), bromocriptine or benzhexol (early treated); and 15 chronically medicated patients who had been maintained for several years on a variety of therapies (chronics). A group of 30 healthy control subjects (HCS) matched for age and sex was obtained from a variety of sources: spouses of PD patients, nonprofessional hospital staff, and patients without hearing impairment from an ear-nose-throat clinic.

Exclusion criteria for all subjects were head injury with loss of consciousness in excess of one hour, history of thyroid disease or diabetes, electroconvulsive therapy, major psychiatric disorder, psychoactive medication other than minor tranquilizers and hypnotics, alcohol consumption in excess of 56 units per week (Royal College of Psychiatrists, 1986), or any other condition known to impair central function other than PD. All subjects were self-declared as right-handed except 1 PD patient who was left-handed.

PD subgroups did not differ significantly from HCS in age ($F(3,91) = 0.27, p > .1$) or premorbid IQ, as measured by the National Adult Reading Test (NART; Nelson & O'Connell, 1978) ($F(3,91) = 2.59, p > .05$) and no patient was demented according to DSM-III criteria of the American Psychiatric Association (1980). The PD subgroups did not differ significantly from each other with respect to global cognitive capacity (score on the memory and orientation section of the Blessed Dementia Scale, BDS; Blessed, Tomlinson, & Roth, 1968) ($F(2,62) = 0.20, p > .1$) or severity of depression (score on the Beck Depression Inventory, BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) ($F(2,37) = 1.05, p > .1$). The chronic subgroup, however, had a significantly longer disease duration than both the *de novo* ($p < .0001$) and early treated ($p < .0001$) patients and also differed from the *de novo* subgroup ($p < .01$) in clinical motor disability (score on the Kings College Rating Scale, KCRS; Brown, Marsden, Quinn, & Wyke, 1984).

Table 1. Characteristics of Subject Groups: Mean and (Range).

Subject Group	Sex M/F	Age (years)	Premorbid IQ (NART)	Duration of Disease (years)	Motor Disability Rating (KCRS)	Dementia Rating (BDS)	Depression Rating (BDI)
Control	15/15	61.1 (43.7-76.1)	110.3 (94-124)	—	—	1.1 (0-4)	5.8 (0-14)
De Novo PD	8/12	60.8 (39.7-77.1)	107.1 (89-122)	1.8 (0.3-5.0)	18.3 (9-38)	3.4 (0-9)	13.5 (1-32)
Early Treated PD	16/14	61.7 (42.1-80.1)	104.6 (90-119)	2.2 (0.9-5.7)	24.4 (3-49)	3.5 (0-10)	9.7 (1-18)
Chronic PD	12/3	63.4 (50.9-74.2)	105.1 (93-119)	9.6 (3.0-20)	29.2 (13-45)	3.9 (1-7)	10.4 (0-19)

The early treated subgroup comprised 10 patients who had been randomized to Madopar (mean daily dose = 506 mg, range 250 - 1000 mg), 10 randomized to bromocriptine (mean daily dose = 24 mg, range 12.5 - 40 mg) and 10 to benzhexol therapy (mean daily dose = 7 mg, range 2 - 15 mg). These treated subgroups (Table 2) did not differ from each other in their duration of treatment ($F(2,27) = 2.29, p > .1$) or from the de novo patients with respect to age ($F(3,46) = 0.63, p > .1$), global cognitive capacity ($F(3,46) = 0.37, p > .1$) or depression ($F(3,22) = 0.83, p > .1$). Those patients taking benzhexol, however, were worse than the de novo ($p < .01$) and Madopar-treated cases ($p < .05$) in terms of clinical motor disability.

Procedure

Incidental and Intentional Learning of Test Items and Their Spatial Location

Test material consisted of 20 line drawings, displayed in a 4 X 5 matrix on an 8" X 7.5" white board. The items were selected from 14 categories of Snodgrass and Vanderwort

Table 2. Characteristics of the Early Treated PD Subgroup: Mean and (Range).

Subject Group	Sex M/F	Age (years)	Premorbid IQ (NART)	Duration of Treatment (months)	Motor Disability Rating (KCRS)	Dementia Rating (BDS)	Depression Rating (BDI)
Levodopa	6/4	64.4 (52.0-70.2)	106.4 (96-119)	14.3 (4.3-24)	18.0 (3-38)	3.1 (0-5)	7.2 (1-15)
Bromocriptine	5/5	59.0 (48.8-69.6)	102.1 (90-116)	8.9 (3.2-11.1)	25.3 (14.5-44.5)	3.3 (0-8)	11.8 (6-17)
Anti-Cholinergics	5/5	61.7 (42.1-80.1)	105.2 (93-114)	10.6 (3.1-30)	30.6 (10-49)	4.2 (1-10)	11.2 (3-18)

(1980); 10 items were common within their categories (ranking 1-9) and 10 were uncommon (ranking 13-76). Common and uncommon items were balanced in their distribution on the test matrix. There were three forms of the test, matched for item category and frequency.

The test procedure comprised three conditions:

1. **Incidental spatial recall.** Subjects were asked to name each of the 20 items in turn and to estimate its size in one dimension. The order of presentation of items was spatially random, but balanced for item category and frequency, and was consistent across subjects. Approximately 45 seconds after the presentation of the last item, subjects were presented with a blank 5 X 4 matrix of identical dimensions to the test grid which was used for recall of spatial location. Identical line drawings to the test stimuli were presented one at a time, in reverse order from that used in the acquisition phase. Subjects were required to indicate the location of the item by pointing to the corresponding cell of the test matrix. Recalled location was recorded on a computer via a pressure sensitive pad beneath the test matrix (Keyport). Finally, subjects were asked to recall without warning the list of items presented.
2. **Intentional spatial recall (dual task condition).** The procedure was identical to that of the incidental condition except that subjects were instructed initially to remember the location of each item as well as estimating its size. The time taken to carry out this condition was recorded. Spatial recall was again followed by recall of the list of test items.
3. **Intentional spatial recall (control condition).** This condition followed an identical procedure to the previous two except that subjects were required only to remember spatial location; no estimates of size were made. The time provided for the task was, however, made equal to that of the intentional (dual task) condition and divided equally among the 20 items. Finally as in the previous two conditions, subjects were required to recall the test items.

Different forms of the test were used for each condition, balanced across subjects. Responses were scored for each item as 5 for a correctly identified location, 2.5 for a mislocation of one cell in the vertical or horizontal planes, and 1.25 for a mislocation by one cell in both planes; all other responses scored zero. Response latency was also recorded by the computer (to the nearest 10 ms) measured from the moment a cue was given to the experimenter to present the test item to the moment that the subjects' finger registered a response on the keyport pad.

Motor Disability. A neurologist gave the Kings College Rating Scale (KCRS; Brown et al., 1984), which is a quantitative measure of clinical motor disability, including akinesia, tremor, rigidity and paucity of movement. Higher scores indicate poorer performance. Manual dexterity was measured under unimanual conditions (for each hand separately) by the Fine Finger Movements Test (Corkin, Growdon, Sullivan, & Shedlack, 1981). The task required the subjects to rotate a mounted spindle between the thumb and first finger as quickly as possible for 30 seconds.

Memory. The Wechsler Memory Scale (WMS) was administered according to published instructions (Wechsler & Stone, 1945). Forgetting in short-term memory was examined with the Brown-Peterson distractor paradigm in which recall of consonant trigrams was assessed over various distraction-filled intervals (Brown, 1958; Peterson & Peterson, 1959). The procedure employed retention interval times of 0, 3, 6, 9, 15 and 30 seconds with serial three subtractions forming the distraction element.

Executive Functions. "Frontal-lobe" capacity was assessed using the Milner (128 cards; 1963) version of the Wisconsin Card Sorting Test (WCST). Performance was scored according to the method of Heaton (1981) to yield the measures of categories, perseverative responses and cards sorted to achieve the first category. Cognitive sequencing and working memory were assessed with a test of digit ordering (Cooper, Sagar, Jordan, Harvey, & Sullivan, 1991). In this task, subjects were read a random selection of seven digits (e.g., 5-3-6-2-7-2-1) and were required to reorder the items in memory and repeat them in ascending fashion (in this example, 1-2-2-3-5-6-7). Each subject was given 15 trials. For each trial, one point was awarded for each digit placed in its correct position until a response broke the ascending sequence (e.g., 6 followed by 4). Maximum score was 7; if a subject reported more than 7 digits, only the first 7 were scored. Responses that did not come from the test presentation but, nevertheless, maintained ascending sequence were tolerated but not scored.

Statistical Analysis

Differences between data sets were evaluated by one-way and repeated measures analyses of variance (ANOVA). Planned comparisons between sets of data were made using *t* tests for two independent samples (two-tailed, unless otherwise stated). Correlations were determined with Pearson product-moment correlation coefficient, *r*.

RESULTS

Latency of Response and Duration of Test Procedure

Latency data was available for 59 of the PD patients and for 26 HCS. A two-way ANOVA of Group (PD, HCS) by Condition (incidental, dual intentional, control) showed a significant main effect of Condition ($F(2,166) = 21.85, p < .0001$) but the main effect of Group ($F(1,83) = 2.59, p = .1$) and the Group by Condition interaction ($F(2,166) = 0.02, p = .98$) were both insignificant, implying that the PD patients were not physically hampered in making their responses in any condition. Response latency was slower in the incidental condition than the dual intentional ($p < .01$) or control conditions ($p < .0001$); responses in the control condition were also faster than those made in the dual intentional condition ($p < .001$). The time required by PD patients to name and estimate the size of the test items did not differ significantly from that of HCS ($t = 1.16, p = .3$). Thus differences in test performance are also unlikely to be related to the confounding influence of test duration.

Spatial and Item Recall: PD Versus HCS

In preliminary analyses the total PD group ($N=65$) was compared with HCS for spatial and item recall separately. Spatial recall was examined with a three-way ANOVA of Group (PD, HCS) by Condition (incidental, dual intentional, control) by Familiarity of Stimuli (More, Less). This highlighted significant main effects of Condition ($F(2,186) = 63.41, p < .0001$) and Familiarity of Stimuli ($F(1,93) = 67.58, p < .0001$) but the main effect of Group ($F(1,93) = 1.48, p = .2$) and all two- and three-way interactions failed to reach significance (Fig. 1). Paired comparisons showed that recall of spatial location did not differ between PD and HCS for the incidental ($p = .7$) or dual intentional conditions ($p = .5$) but

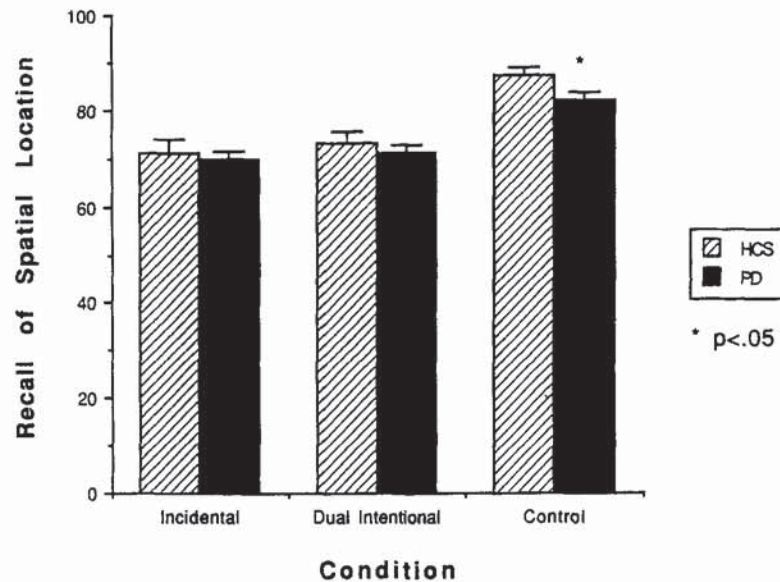


Fig. 1. Mean recall of spatial location for each of the three experimental conditions (incidental, dual intentional and control); vertical bars represent the SEM. PD patients were unimpaired overall but did show a mild deficit compared to HCS in the control condition.

the performance of the PD group was inferior to that of HCS in the control condition ($p < .05$). The recall of spatial location, for all subjects, was superior in the control condition than in incidental ($p < .0001$) or dual intentional conditions ($p < .0001$) but there was no significant difference in performance at these latter two conditions ($p = .2$). The main effect of Familiarity of Stimuli was due to the subjects' superior recall of the spatial location of the less familiar items.

Item recall was investigated with a two-way ANOVA of Group (PD, HCS) by Condition (incidental, dual intentional, control) which showed significant main effects of Group ($F(1,93) = 17.58, p < .001$) and Condition ($F(2,186) = 3.50, p < .05$) but the interaction term was not significant (Fig. 2). PD patients recalled fewer test items than HCS under incidental ($p < .05$); dual intentional ($p < .0001$) and control conditions ($p < .01$). The main effect of Condition was attributable to the inferior performance of subjects at the dual intentional condition relative to the control condition ($p < .05$); performance in the incidental and dual intentional conditions did not differ significantly from each other.

The PD group as a whole was thus impaired at free recall of the items but not at recalling their spatial location. To establish whether these results were attributable to a factor of scaling (a selective deficit in the PD group in the free recall condition as a simple consequence of the more taxing nature of the task) or

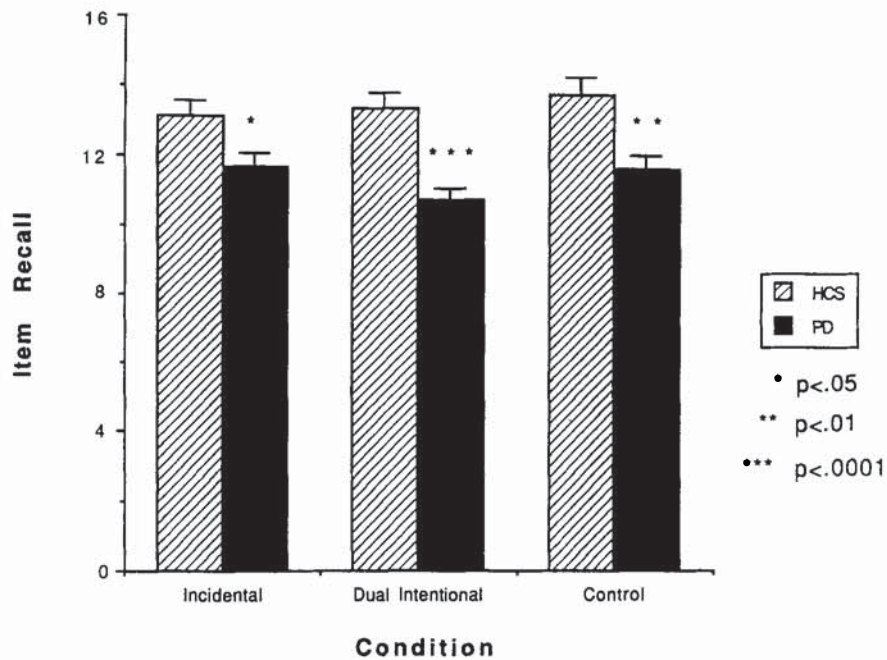


Fig. 2. Mean item recall for each of the three conditions (incidental, dual intentional and control); vertical bars represent the SEM. PD patients were significantly impaired, in all conditions.

whether they represented a qualitative dissociation of performance, we carried out a further analysis comparing the results of the two test elements directly. The data were standardized by a conversion to Z scores and then subject to a three-way ANOVA of Group (PD, HCS) by Task (item, spatial location) by Condition (incidental, dual intentional, control). A significant main effect arose for Group ($F(1,93) = 9.54, p < .01$) and Group interacted significantly with Task ($F(1,93) = 6.62, p < .05$). The PD group performed worse than the HCS on the test as a whole and were selectively impaired at item ($p < .0001$) but not spatial recall ($p = .2$) (Fig. 3). Thus the selective deficit for item recall found in the PD group appears genuinely disproportionate to the mild and insignificant deficit in spatial recall. The nature of the data transformation for this analysis abolished the significance of the main effects of Task and Condition and the interaction between these terms, since in each of these cases the mean of the transformed results was zero.

When overall test performance for each condition was analysed separately, again using data transformed to Z scores, PD patients showed significant impairment under dual intentional ($F(1,93) = 9.81, p < .01$) and control ($F(1,93) = 8.67, p < .01$) conditions but were unimpaired relative to HCS at the incidental condi-

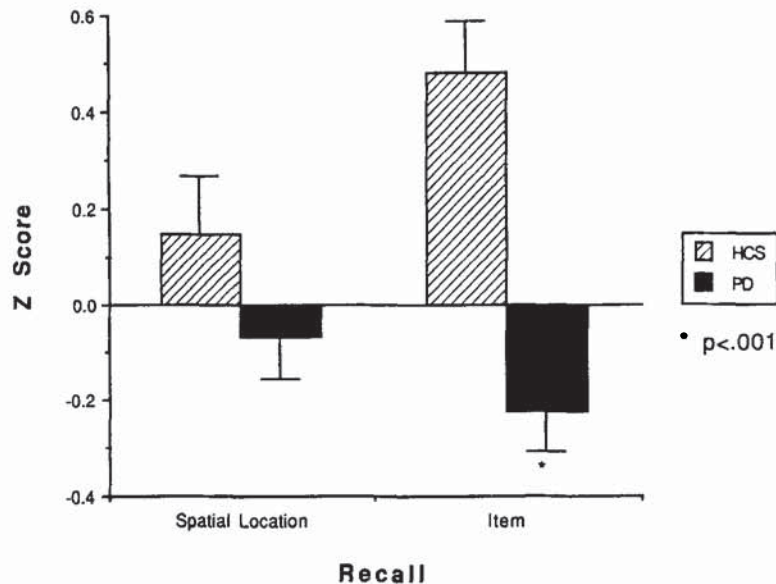


Fig. 3. Mean Z score for spatial location and item recall; vertical bars represent the SEM. PD patients were disproportionately impaired at recall of the test items compared with recall of the spatial location of the items.

tion ($F(1,93) = 3.33, p > .05$). Since deficits were found for the PD group under intentional but not incidental learning conditions and because their performance for the two intentional conditions was broadly comparable, despite dual task demands in one case, data for these conditions were pooled into a single (Intentional) measure (for spatial and item information separately). This made possible a direct comparison of group performance under incidental and intentional learning conditions. A three-way ANOVA of Group (PD, HCS) by Task (item, spatial location) by Condition (incidental, intentional) of Z-transformed data highlighted a main effect of Group ($F(1,93) = 7.99, p < .01$) and interactions between Group and Task ($F(1,93) = 5.30, p < .05$) and Group and Condition ($F(1,93) = 5.19, p < .05$). Once again the main effect of Group was attributable to inferior performance by the PD patients, in item ($p < .001$) but not spatial recall ($p = .3$). The Group by Condition interaction was due to the PD patients being significantly impaired under the intentional condition ($p < .01$) but showing only a mild and insignificant test deficit for the incidental condition ($p = .07$) (Fig. 4). As in the previous analysis, the main effects of Task and Condition and the interaction between them were nullified by the data transformation.

Finally, the recall of spatial location data was analysed with respect to the items' serial position. In the acquisition phase, the experimenter pointed to each

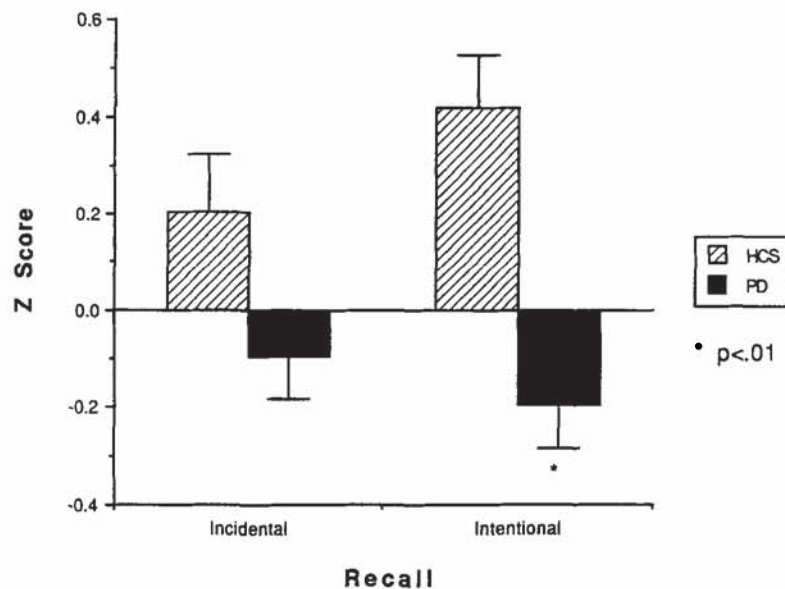


Fig. 4. Mean Z score for incidental and intentional learning conditions (combined item and spatial location data); vertical bars represent the SEM. PD patients were disproportionately impaired under the intentional learning condition.

of the items in a random (but constant) order which was reversed when the subject was required to recall their spatial location. Hence, for example, if an item was 5th in order in the acquisition phase it was 15th during the test of spatial location. For the purpose of analysis, data were pooled and averaged for each successive pair of items, so that items 19 and 20 of the acquisition phase became data point 1 of the spatial recall test session. To examine the effect of serial position on the probability of recall a three-way ANOVA of Group (PD, HCS) by Condition (incidental, dual intentional, control) by Order (1,2,...,10) was performed. This highlighted main effects of Condition ($F(2,186) = 66.86, p < .0001$) and Order ($F(9,837) = 14.45, p < .0001$) but the main effect of Group and all two- and three-way interactions were insignificant. As a previous analysis showed, recall of spatial location was superior in the control condition than incidental ($p < .0001$) or dual intentional conditions ($p < .0001$). The main effect of Order was attributable to a recency effect for items processed late in the acquisition phase and tested early in the recall session. A primacy effect for items processed towards the beginning of the acquisition phase and recalled late in the test period was less evident but the profile of performance was clearly similar for patients and controls (see Fig. 5) and confirmed by the insignificant Group by Order interaction.

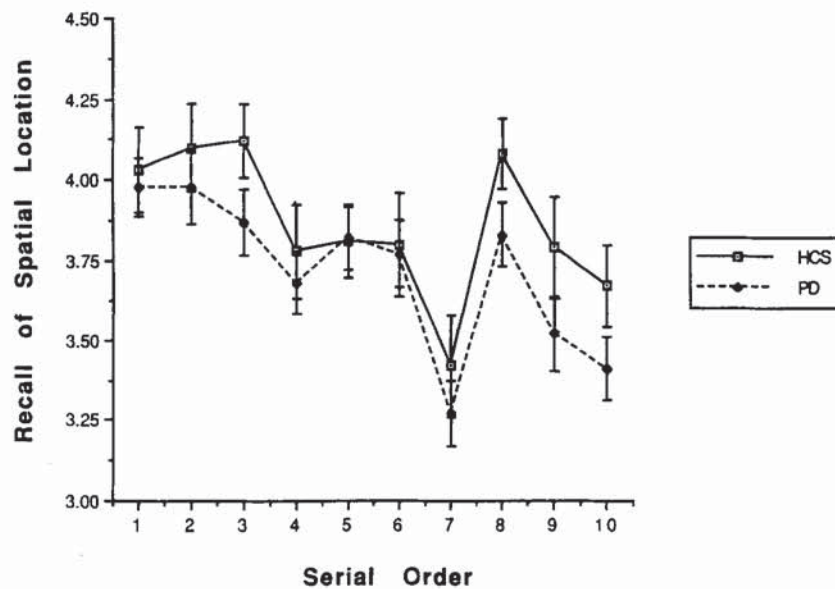


Fig. 5. Mean recall of spatial location according to serial position; low numbers represent recent items. Data is pooled from all three learning conditions; vertical bars represent the SEM. PD patients recalled the items' spatial location as well as did HCS and displayed the same effect of recency.

Spatial and Item Recall: HCS versus the PD Subgroups

The following series of analyses were carried out to examine spatial and item recall for each of the PD subgroups and to investigate the possibility that test performance might differ selectively with respect to disease chronicity or treatment. A two-way ANOVA of Subgroup (*de novo*, early treated, chronic, HCS) by Condition (incidental, dual intentional, control) for the spatial location results led only to a main effect of Condition ($F(2,182) = 66.86, p < .0001$). Once again performance at the control condition was superior to that at the incidental ($p < .0001$) or the dual intentional condition ($p < .0001$). The insignificant main effect of Subgroup ($F(3,91) = 0.96, p = .4$), however, confirmed that the subgroups performed the spatial location element of the task comparably (see Fig. 6). Indeed posthoc analysis failed to reveal any significant differences between the subgroups for these data. The equivalent two-way ANOVA of Subgroup (*de novo*, early treated, chronic, HCS) by Condition (incidental, dual intentional, control) for the item recall analysis showed main effects of Subgroup ($F(3,91) = 5.86, p < .01$) and Condition ($F(2,182) = 3.48, p < .05$). The HCS recalled more items than the *de novo* ($p < .01$), the early treated ($p < .01$) and the chronic groups ($p < .01$) but the PD subgroups did not differ significantly from each other (see

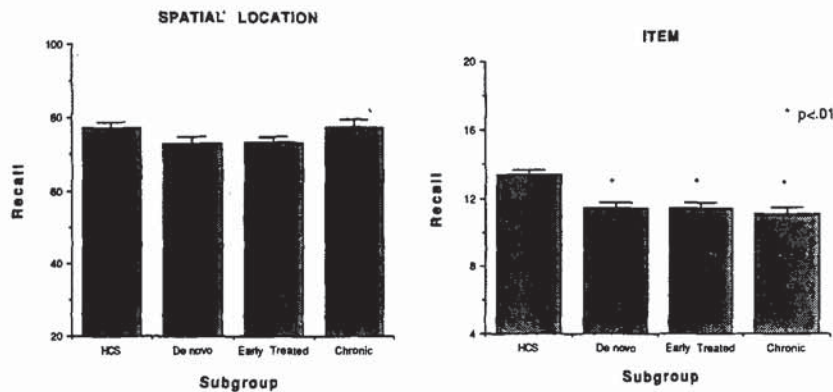


Fig. 6. Mean recall of item and spatial location for HCS and the three PD subgroups (de novo, early treated and chronic); vertical bars represent the SEM. Data are pooled and averaged across the three learning conditions. All PD subgroups showed normal recall of spatial location but were impaired relative to HCS at item recall. The PD subgroups did not differ significantly from each other on either recall task.

Fig. 6). The main effect of condition was due to superior recall under the control condition relative to the dual intentional condition ($p < .05$).

When the data for the subgroups was transformed to Z scores and subject to a three-way ANOVA of Subgroup (*de novo*, early treated, chronic, HCS) by Task (item, spatial location) by Condition (incidental, dual intentional, control) the above results were confirmed. A main effect of Subgroup ($F(3,91) = 3.19$, $p < .05$) arose from superior performance by the HCS relative to the *de novo* ($p < .05$) and early treated patients ($p < .01$) and a significant Subgroup by Task interaction ($F(3,91) = 3.46$, $p < .05$) again reflected comparable performance by the groups in recall of spatial location but superior item recall by the HCS relative to each of the PD subgroups ($p < .01$), which did not differ from each other.

A repetition of the analysis carried out on the overall PD group, examining the effect of learning condition upon performance, was also performed for the PD subgroups. The three-way ANOVA of Subgroup (*de novo*, early treated, chronic, HCS) by Task (item, spatial location) by Condition (incidental, intentional) yielded a borderline significant main effect of Subgroup ($F(3,91) = 2.66$, $p = .052$) and significant interactions of Subgroup with Task ($F(3,91) = 2.84$, $p < .05$) and Subgroup with Condition ($F(3,91) = 2.97$, $p < .05$). HCS performed better overall than the *de novo* ($p < .05$) and early treated ($p < .05$) subgroups. The PD subgroups were selectively impaired, relative to HCS, at item ($p < .01$) but not spatial location recall and the *de novo* ($p < .05$) and early treated subgroups ($p < .01$) were impaired, relative to HCS, under intentional conditions but all subgroups performed comparably under the incidental learning condition.

The Effects of Treatment

Only those patients receiving levodopa therapy showed evidence of improved motor control on treatment. Their score on the Fine Finger Movements Test (for left- and right-hand performance averaged) improved from a pretreatment level ($p < .01$) and was significantly better than the score achieved by the *de novo* subgroup ($p < .05$). Despite this improvement in motor control however, the cognitive measures of spatial and item recall failed to demonstrate any benefit. A three-way ANOVA of Subgroup (de novo, levodopa, bromocriptine, anticholinergic) by Task (item, spatial location) by Condition (incidental, intentional), performed on data transformed to Z scores, yielded neither significant main effects nor interactions. No subgroup(s) performed significantly better on the task overall, at spatial relative to item recall, or under incidental conditions relative to intentional ones.

Correlational Results

Performance for spatial location and item recall (averaged across the three conditions) was correlated with a number of clinical and cognitive variables for the PD group as a whole (Table 3). Recall of spatial location correlated significantly with MQ ($r = .31$, $p < .05$) but failed to show significant association with age, duration of disease, scores on the KCRS, Fine Finger Movements Test (left hand, right hand or averaged), Blessed Dementia Scale, Beck Depression Inventory, NART (premorbid IQ), Brown-Peterson test, Digit Ordering Test or WCST (categories, perseverative responses or cards sorted to achieve the first category). Item recall, conversely, correlated significantly with age ($r = -.40$, $p < .001$), scores on the KCRS ($r = -.28$, $p < .05$), Blessed Dementia Scale ($r = -.53$, $p < .0001$), NART ($r = .28$, $p < .05$), WMS (MQ) ($r = .65$, $p < .0001$), Brown-Peterson Test ($r = .37$, $p < .01$), Digit Ordering Test ($r = .67$, $p < .0001$) and with perseverative responses on the WCST ($r = -.25$, $p < .05$). Item recall did not correlate significantly with Fine Finger Movements score (left hand, right hand or averaged), disease duration, Beck Depression Inventory score or WCST score (categories or cards sorted to achieve the first category). The correlation between spatial and item recall was also significant ($r = .52$, $p < .0001$).

DISCUSSION

The PD patients in this study were impaired at recall of item but not spatial location and deficits were more pronounced under intentional than incidental learning conditions. The PD subgroups, which differed in disease chronicity and treatment, performed comparably to each other for both tasks and under all test conditions. These findings support the notion that effortful but not automatic processes are compromised in PD but the deficits do not respond to dopaminergic medication.

Spatial recall was comparable between the incidental and dual intentional

Tabel 3. Correlation Table.

	Recall			
	Location		Item	
	r	p	r	p
Age	-.23		-.40	<.001
Disease Duration	.15		.01	
KCRS	-.03		-.28	<.05
Fine Finger Movements				
– Left Hand	.02		.20	
– Right Hand	.02		.02	
– Combined	.02		.13	
BDS Score	-.18		-.53	<.0001
BDI Score	-.08		-.27	
NART	-.11		.28	<.05
MQ	.31	<.05	.65	<.0001
Brown-Peterson	.03		.37	<.01
Digit Ordering	.23		.67	<.0001
WCST				
– Categories	.10		.20	
– Perseverative Response	-.18		-.25	<.05
– Cards To 1st Category	-.01		-.08	

conditions (when subjects were required to direct their attention to the secondary estimation task) but improved when these demands were lifted (control condition). These results suggest that although spatial location information was to a large extent encoded automatically, performance could benefit from allocation of additional attentional resource. It is interesting to note that the control condition was the only situation where the spatial recall of the patients fell below that of HCS. They were less able to utilize the available and undisturbed study time to encode item position effortfully. This observation is compatible with the notion that PD patients have poor self-generated attentional capacity or strategy organization. In general, however, the performance of the PD patients for spatial recall was equivalent to that of the HCS. They displayed the same pattern of performance in relation to serial position of the items as normal subjects and were also more likely to forget the more familiar test items than they were the less familiar ones.

PD patients displayed deficits for the more effort-demanding task (item recall) and when learning conditions were most strenuous (intentional). Patients were impaired for the dual intentional and control conditions which were assessed subsequent to the initial incidental task. In principle, this pattern of deficit could be explained by a disproportionate susceptibility to proactive interference

(PI) in the PD group. However, this explanation would predict greatest deficits under the final control condition, whereas the dual intentional condition produced their most significant impairment. Furthermore, a recent study by Sagar, Sullivan, and Cooper (1991) has shown that *de novo* PD patients, at least, show a normal pattern of PI build-up and release. Thus, it seems probable that deficits arising under intentional learning conditions occurred as a result of the additional demands made upon a limited processing resource rather than an artefact of condition order.

If "effort" can be gained from dopaminergic medication it was not evident from the results of this study. Those patients receiving levodopa, who showed consequent improvement in motor control, did not show any sign of superior test performance (on either item or spatial recall) relative to the other early treated subgroups or the *de novo* patients. Furthermore the extent of association between item recall and disease severity was minimal; it showed only a borderline significant correlation with clinical motor disability and insignificant associations with performance on the test of Fine Finger Movements and disease duration. Significant associations did arise however, between item recall and other memory measures, particularly those most dependent upon working memory function. Recall of spatial location, by contrast, not only dissociated from indices of clinical disability but largely from the other cognitive test results as well. Of particular interest is the observation that impaired spatial memory did not associate more strongly with left-sided disability (reflecting right hemisphere pathology) than with right, at least as judged from the results of the Fine Finger Movements Test. This may reflect the fact that true hemi-parkinsonism is rare, since hemisphere dysfunction is normally bilateral even in clinically unilateral cases and that spatial recall was overall unimpaired in this study. Severity of depression did not associate significantly with spatial recall but the correlation with item recall approached significance. This finding is in accord with the work of Hasher and Zacks (1979) which showed that effortful processes are more likely to be affected by factors that reduce attentional capacity (such as depression) than are automatic processes. It should be noted however that the patients assessed in this study were not clinically depressed and that in a larger investigation of cognitive impairment in untreated PD (Cooper et al., 1991) memory and affect dissociated.

Not only did dopaminergic medication fail to confer selective benefit upon processes of free recall but the anticholinergic-treated subgroup also performed comparably to the other patients. Evidence from the literature has highlighted the detrimental effects of this form of medication upon memory processes in normal subjects (Drachman, 1977) and in PD (De Smet et al., 1982; Dubois et al., 1987; Sadeh, Braham, & Modan, 1982) but the patients in our study did not show any sign of a disproportionate deficit relative to the other treated subgroups. In a recent study of Brown-Peterson task performance in PD (Cooper et al., 1992) we showed that anticholinergic medication led to a selective impairment in the immediate recall of information (up to 6 s retention) but recall of items tested later was unaffected. Patients also performed the first trial of an associative learning

task poorly but attained a comparable level of performance to the other patients for the subsequent trials. It is therefore conceivable that the anticholinergic-treated patients in the present study (a subsection of those tested in Cooper et al., 1992) failed to demonstrate a deficit in recall of either item or spatial information owing to the nature of the task demands. Spatial recall began approximately 45 s after the acquisition phase had finished, thereby allowing the material to be encoded without competition for attentional resources. Item recall was tested after spatial recall and subsequent to a second presentation of the test stimuli. In neither case was the retention interval as short as that reported by Cooper et al. (1992) at which anticholinergic medication exerted its most deleterious effect.

Evidence for the role of the frontal lobes in spatial memory has come from both human and animal studies. In man, frontal-lobe lesions have been shown to impair learning of stylus mazes (Corkin, 1965; Milner, 1965) and to interfere with a task of egocentric spatial orientation requiring left-right discrimination (Semmes, Weinstein, Ghent, & Teuber, 1963). A vast body of literature has discussed the role of the prefrontal cortex in spatial memory in primates (reviewed by Fuster, 1989; Goldman-Rakic, 1987) and has highlighted the importance of the principal sulcus for normal performance on delayed-response tasks. The studies of Smith and Milner (1981, 1984) however, showed that delayed item recall was impaired in patients suffering with temporal-lobe and frontal-lobe lesions but spatial recall deficits were contingent upon radical excision of the right hippocampal region. The lack of a spatial memory impairment following frontal-lobe lesions appears to contradict the work of Goldman-Rakic and her colleagues but this discrepancy may be attributable to differing task demands. Delayed-response and delayed-alternation paradigms (that consistently highlight the role of the principal sulcus in spatial memory) generally involve a delay period of a few seconds, whereas in the procedure adopted by Smith and Milner (1981, 1984), spatial recall was examined subsequent to item recall and at least 1 min after stimulus presentation. Zola-Morgan and Squire (1985) showed that conjoint hippocampal and amygdala lesions impaired delayed-response performance in monkeys at delays of 15 s or more. Thus spatial memory deficits following frontal-lobe damage may be selectively confined to those tasks that demand immediate access to recently presented information, a condition that was not investigated by the work of Smith and Milner (1984).

In the present study, spatial recall in PD was unimpaired and performance on the WCST, generally regarded as an index of frontal-lobe function, failed to show significant association with spatial recall. These results may again reflect the fact that spatial recall followed approximately 45 s after the last item had been processed and thus subsequent to the period most compromised by prefrontal lobe dysfunction. If effort-demanding memory processes, compromised in PD, are attributable to frontal lobe dysfunction (Taylor et al., 1986) one must assume that the WCST is at best a poor gauge of this detriment, since correlations between this test and item recall were minimal. Performance on the Digit Ordering Test correlated significantly with item recall although not with spatial recall

suggesting that effort demanding processes may be related to working memory, which itself may be served by the prefrontal cortex.

Impaired item recall with preserved spatial recall was reported by McCormack (1982) in an elderly normal population, a pattern of performance that was explained in terms of reduced attentional processing resource by Rabinowitz, Craik and Ackerman (1982). According to this view, if available processing resources are diminished, an elderly subject will encode an item in a generalized and stereotypical fashion. The resulting memory trace is less likely to be successfully retrieved than one more richly processed. This processing resource model can also explain performance patterns of younger adults whose attentional capacity is experimentally reduced by secondary task requirements. The PD patients in our study were impaired when processing resources were most in demand, at item recall and in situations where the learning condition stressed intentional effort. A reduction in the level of central processing resources in PD has recently been proposed by Brown and Marsden (1991). They concluded that deficits lay within a theoretical limited-capacity central processor but did not postulate an anatomical substrate for this hypothetical system. The present results show that PD patients are differentially impaired for those tasks and conditions which are the most effort demanding. Although alternative explanations exist, the findings support the notion of diminished central processing resources in PD. This can be attributed directly to disease pathology which is common to all groups, since performance was poor in newly diagnosed, untreated early cases as well as patients on short-term and long-term medication.

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