Selective Deficits in Alzheimer and Parkinsonian Dementia: Visuospatial Function

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ABSTRACT: Deficits in visuospatial cognition are frequently cited as an important component of the cognitive changes accompanying Parkinson's disease. To characterize possible differences between Parkinson's (PD) and Alzheimer's (AD) dementia, patients from both groups, matched for overall dementia severity, age and education, were contrasted neuropsychologically. Visuospatial tasks dissociated from memory, were significantly compromised in both patient groups. Differential impairment was evident on visuospatial abstraction and reasoning (Object Assembly), which was most deficient in PD. Visuospatial cognition associated with memory, classified both patient groups as impaired compared to controls, but AD patients demonstrated substantially lower performance levels than those with PD. Parkinsonian dementia thus appears to have some distinct features compared to Alzheimer's disease, which may indicate differences in underlying pathogenic mechanisms.

INTRODUCTION

Intellectual deficits are now recognized as an important feature of Parkinson's disease.\(^1\,2\,3\) The incidence, severity and precise characteristics of the cognitive dysfunction, nevertheless, remains controversial.\(^4\,5\) In particular, the question of a parkinsonian dementia, separate and distinct from the dementia of the Alzheimer-type, has been difficult to assess. Attempts to characterize parkinsonian dementia have included studies of neuropathological features,\(^6\,10\) transmitter abnormalities,\(^4\,11\) radiographic alterations,\(^12\) and neuropsychological changes.\(^13\) While certain relatively unique features of parkinsonian dementia have been ascertained (e.g. Lewy-body type cortical changes),\(^6\) it is unclear whether differences between Parkinson's and Alzheimer's dementia outweigh communitiies.

Visuospatially mediated tasks were therefore chosen to further investigate the question of potential differences between Parkinson's and Alzheimer type dementia. To control for possible confounds, patients from both groups were matched for age, education and overall level of dementia (Mattis Dementia Rating Scale).\(^17\)

METHODS

Eleven demented patients (DSM III-R criteria)\(^18\) with idiopathic Parkinson's disease, (10 men, 1 woman, mean ± SEM age 69 ± 1.4, range 59-76 years), 11 patients with Alzheimer's disease were included. All patients were in the severe or moderate stage of dementia. Using established dementia staging, \(n=1\) patient was in the severe stage, \(n=9\) patients were in the moderate stage. All patients underwent a detailed clinical and neuropsychological evaluation. Visuospatial tasks included: Object Assembly,\(^14\) Recognition memory,\(^19\) the Mazes subtest of the Wechler Memory Scale,\(^20\) and the Trail making Test,\(^21\) which were administered at the beginning and end of the neuropsychological battery. Performance was measured using the Wechsler Adult Intelligence Scale,\(^22\) the Mattis Dementia Rating Scale,\(^17\) the California Verbal Learning Test,\(^23\) the Brief Visuospatial Memory Test,\(^24\) the Digit Symbol Substitution Test,\(^25\) and the Digit Span test from the Wechsler Memory Scale.\(^26\)

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RESULTS

Evaluation of overall intellectual status (Verbal, Performance and Full Scale IQ of the WAIS-R) attested to a substantial and comparable functional reduction in both patient groups (p < .01; Table 1). Overall memory (Memory Quotient) was also deficient in both groups compared to controls (p < .01) but evidenced a greater deficit for Alzheimer’s compared to parkinsonian patients (p < .02; Table 1).

Visuospatial cognition, not principally associated with memory (as measured by WAIS-R Performance subscales, Mosaic Comparison Test, Rey-Osterrieth Complex Figure-Copy), was significantly compromised in both patient groups compared to controls (p < .01, Figure 1). Performance differences between the 2 groups were limited to Object Assembly (WAIS-R Performance subscale), where parkinsonian patients showed a substantially greater deficit than those with Alzheimer’s disease (p < .05, Figure 1). Analysis of process (time taken to complete the Object Assembly task, total number of pieces assembled, number of erroneous connections, differences as a function of design difficulty and contrast of number of perfectly assembled designs) failed to reveal any differences between demented Parkinsonians and Alzheimer’s patients.

Visuospatial cognition with principal memory components (Visual Reproduction, Wechsler Memory Scale; Object Recognition and Placement; Rey-Osterrieth Complex Figure, 3 minute delayed recall; Street Map) was performed less well by both patient groups compared to controls (p < .01). However, Alzheimer’s patients tended to show relatively greater impair-

Table 1: Subject Characteristics.

<table>
<thead>
<tr>
<th></th>
<th>Normal Controls (N=11)</th>
<th>Alzheimer’s Patients (N=11)</th>
<th>Parkinsonian Patients (N=11)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Education</td>
<td>17 ± 3.1 (12-20)</td>
<td>18 ± 1.5 (16-20)</td>
<td>16 ± 1.0 (12-20)</td>
</tr>
<tr>
<td>Symptom Duration</td>
<td>5 ± 1.0 (1-10)**</td>
<td>13 ± 4.7 (6-18)</td>
<td>3 ± 0.1 (2-3)</td>
</tr>
<tr>
<td>Hoehn and Yahr Score</td>
<td>3 ± 0.1 (2-3)</td>
<td></td>
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<tr>
<td>Wechsler Tests</td>
<td></td>
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<tr>
<td>Verbal IQ</td>
<td>128 ± 2.8 (108-144)</td>
<td>97 ± 3.4 (79-123)*</td>
<td>98 ± 2.3 (86-111)*</td>
</tr>
<tr>
<td>Performance IQ</td>
<td>128 ± 3.2 (117-142)</td>
<td>81 ± 2.4 (65-90)*</td>
<td>78 ± 3.4 (66-103)*</td>
</tr>
<tr>
<td>Full Scale IQ</td>
<td>132 ± 2.9 (120-148)</td>
<td>89 ± 2.5 (80-109)*</td>
<td>93 ± 5.0 (81-133)*</td>
</tr>
<tr>
<td>Memory Quotient</td>
<td>137 ± 3.3 (110-143)</td>
<td>76 ± 2.8 (64-93)**</td>
<td>94 ± 6.4 (64-143)*</td>
</tr>
</tbody>
</table>

* Patients different from Normal Controls at p < .01.
** Alzheimer’s patients different from parkinsonian patients at p < .02.
*** Alzheimer’s patients different from parkinsonian patients at p < .001.
ment levels on most of these tasks than parkinsonian patients. Object Recognition and Placement as well as the Street Map Test, were significantly less well performed by those with Alzheimer’s disease (p < .05, Figure 2). Delayed recall of the Rey-Osterrieth Complex Figure was borderline deficient in the Alzheimer’s, compared to the parkinsonian patients (p < .07, Figure 2).

**DISCUSSION**

The present results showed characteristic differences in cognitive profiles between patients with Alzheimer’s and Parkinson’s disease, having equivalent degrees of overall intellectual dysfunction, confirming earlier observations in other cognitive realms.32

Visuospatial tasks not principally associated with memory, evidenced some differentiation between the two demented groups. Object Assembly, a visuospatially mediated task necessitating the ability to abstract from an incomplete stimulus and visuospatial reasoning, both skills which have been associated with frontal function,33 was significantly impaired in the Parkinsonians compared to controls as well as to Alzheimer’s patients; the latter were also less proficient than controls. A comparison of the time taken to complete the task as well as additional task analysis, failed to reveal differences between the two groups, which strengthens the argument that visuospatial abstraction and reasoning abilities overall, rather than motor slowing or differential errors as a function of difficulty, were the source of observed differences. While this test’s semantic memory component may have played a role in observed results, the remaining memory profile of the two patient groups would actually favor parkinsonians. It is therefore likely that the visuospatial abstraction and reasoning components of this test were the source of observed performance differences. However, these two patient groups failed to differ on Picture Arrangement, a test associated with sequential thinking and social sophistication,34 both of which may also be related to frontal lobe function. Nevertheless, Object Assembly and Picture Arrangement may use different “cognitive routines”. While the former would appear to rely on the combination of visuospatial abstraction and reasoning, the latter necessitates principally social sophistication and reasoning. In as far as visuospatial cognition might be impaired in Parkinson’s disease (e.g.),14 one might speculate that visuospatial abstraction is the most “vulnerable” of these tasks in the context of parkinsonian dementia and therefore the most likely to differentiate between the dementia subtypes under investigation here. In addition, Block Design, another timed
visuospatially mediated test, generally more associated with posterior dysfunction, was comparably performed by both patient groups. Since all of these tasks are timed, reduced speed is unlikely to account for these differential findings. The two other visuospatially mediated items without principal memory components (Rey-Osterrieth Complex Figure and Mosaic Comparisons), failed to differentiate between the two demented groups.

Visuospatial tasks with principal memory components classified Alzheimer’s patients as more impaired than parkinsonian patients. This was true for overall memory function (Memory Quotient) in spite of comparable intellectual levels (Full Scale, Verbal and Performance IQs). Alzheimer’s patients were more deficient regardless of which memory systems were tested (episodic memory, context bound knowledge such as the Rey-Osterrieth Complex Figure, Recall, versus semantic memory, context-free, general principles like the Street Map). The relative failure of the Visual Reproduction subscale of the Wechsler Memory Scale to differentiate between the two groups, might be related to the potential motor disadvantage of parkinsonian patients in face of the graphic demands of this task, as well as item complexity. Recall of simple geometric figures (Visual Reproduction) may not have taxed memory sufficiently, while recall of the complex Rey-Osterrieth Figure, in spite of graphic motor demands on the Parkinsonians, tended to classify Alzheimer’s patients as relatively more impaired than those with Parkinson’s disease.

Neuroanatomical evidence may help explain the observed double dissociation between performance on Object Assembly (a visuospatially mediated task of abstraction and reasoning, where Parkinsonians evidenced the relatively greatest deficit) and visuospatial memory (where Alzheimer’s patients were least accurate). Frontal deficits appear to be a consistent feature of Parkinson’s disease. Preclinical evidence with primates implicates a loss of striatal-frontal connectivity in these impairments, likely one of the pathognomonic aspects of Parkinson’s disease. The frontal deficits noted in Alzheimer’s patients, although milder than in Parkinsonians, may be indicative of frontal cortical involvement, observable in some individuals with Alzheimer’s disease. Differential spatial memory

![Image of graphs showing memory tasks performance]

*Patients Significantly Different from Controls at p<.01
**Parkinson Patients Significantly Different from Alzheimer Patients at p<.05
deficits in the two groups, might reflect the well established involvement of septo-hippocampal systems in Alzheimer’s patients, while Alzheimer-type changes in the septo-hippocampal system are reported to occur in demented parkinsonian patients, they are not always evident. The observed milder impairments in Parkinson’s patients may alternatively implicate dorsolateral striatal-frontal connections, which have tentatively been linked to spatial memory.

Both communalities and differences in these two dementia groups might also be related to characteristic neurotransmitter abnormalities. While a whole host of transmitter deficits are implicated in both disorders, their relative contribution may be of importance. The deficit in dopaminergic neural transmission is the central contributor to parkinsonian motor symptoms, and may play some limited role in cognition. The functional role of dopamine in Alzheimer’s disease is less secure and earlier attempts of palliative treatment with L-Dopa failed to yield encouraging results. Cortical noradrenergic deficiencies, on the other hand, are more common to both groups. Deficits in this system, which has been associated with learning and memory, could thus be a necessary but not sufficient condition for dementia symptoms. Compromises in cholinergic function, one of the central neurotransmitter deficits in Alzheimer’s disease, have also been observed in Parkinson’s disease, but do not appear to be a consistent feature. The complexities of transmitter involvement in the primary degenerative dementias make conclusions difficult, a more catecholamine based dementia in Parkinson’s disease, compared to a more acetylcholine related dementia in Alzheimer disease, might account for some of the neurobehavioral differences observed here.

Parkinson’s and Alzheimer’s patients matched for overall level of dementia evidenced certain communalities in neurobehavioral deficits. Nevertheless, the important differences in profiles observed here, mainly the dissociation between visuospatial cognition involving abstraction versus memory-related function in the 2 disorders, point to differences in the underlying dementing process from Alzheimer’s disease in the majority of parkinsonian patients in the present study. Reports of multiple pathophysiological profiles in demented Parkinsonians, however, caution against simplistic interpretations.

REFERENCES


