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Basal ganglia disease and visuospatial cognition: Are there disease-specific impairments?

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Visuospatial deficits in basal ganglia disease may be a non-specific function of the severity of dementia or they could reflect disease-specific impairments. To examine this question, Huntington (HD) patients, demented and non-demented Parkinson (PD) patients and healthy controls were examined with neuropsychological tests emphasising visuospatial abilities. Global intellectual function and general visuospatial cognition were less efficient in the two demented patient groups relative to both controls and non-demented PD patients and they did not differ significantly between non-demented Parkinsonians and controls nor between demented PD and HD patients. However, HD patients but not demented PD patients were impaired on a test of person-centred spatial judgement compared to non-demented subjects while demented PD patients scored significantly lower than HD patients on a test of field independence. Factor analysis yielded a factor reflecting general visuospatial processing capacity which discriminated between demented and non-demented PD patients but not between demented PD and HD patients. A unique factor associated with the manipulation of person-centred space discriminated between demented PD and HD patients. These results suggest general visuospatial processing is impaired as a non-specific function of dementia presence in HD and PD. Abnormalities in circumscribed aspects of visuospatial function, on the other hand, may differentiate between HD and PD, suggesting differential involvement of the basal ganglia in the respective illnesses.

Keywords: Basal ganglia disease – Dementia – Huntington's disease – Parkinson's disease – Visuospatial impairment

INTRODUCTION

Visuospatial function, i.e. cognitive processes mediated by visual input rather than language, is consistently found to be affected in basal ganglia disorders (for example, Brouwers *et al.*, 1984; Beatty, 1989; Mohr *et al.*, 1991; Levin *et al.*, 1991; Drachman *et al.*, 1982; Filoteo *et al.*, 1995). Indeed, disability in this sphere is among the most uniformly reported cognitive complication in patients with HD or PD (Mohr *et al.*, 1995; Purdon *et al.*, 1994). A more global dementia is also associated with these two basal ganglia diseases. In HD, the presence of dementia is well established (Granholm and Butters, 1988; Starkstein *et al.*, 1988; Saint-Cyr *et al.*, 1988; Weinberger *et al.*, 1988; Goldberg *et al.*, 1990) and a further investigation reported a significant association between impairment of general visuospatial ability and magnitude of dementing symptoms in HD patients (Mohr *et al.*, 1991). In addition, cognitive deficits in specific aspects of visuospatial function have been demonstrated (Fedio *et al.*, 1979; Jason *et al.*, 1988), particularly when manipulation of person-centred space (as opposed to environment-or object-centred space (Olson and Gettner, 1995, Brouwers *et al.*, 1984; Potegal, 1971)) is required. Severity of this deficit has been linked to duration of symptomatic expression of disease in HD patients (Mohr *et al.*, 1991). In PD, dementia is present in only 20 to 30% of cases and the relationship between functional decrements in general visuospatial cognition and the presence of dementia is less certain. Within the various components of visuospatial function, there is some evidence suggesting that spatial orientation remains intact in

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non-demented Parkinsonians (Raskin *et al.*, 1992) but retention of other visuospatial abilities is less certain.

The following study was designed to examine whether visuospatial deficits in basal ganglia disease are a non-specific function of dementia severity or whether they reflect disease-specific impairments. PD offers unique possibilities for such a study due to the presence of subgroups with and without dementia (Girotti *et al.*, 1988; Mohr *et al.*, 1990; Mohr *et al.*, 1995; McFadden *et al.*, 1996). Accordingly, for this study we compared neuropsychological function in non-demented and demented PD patients as well as demented PD and HD patients (matched for dementia severity), relative to normal controls. Thus the effect on visuospatial functioning of presence or absence of dementia and of distinctive motor disease involving the basal ganglia could be ascertained independently.

METHODS

Twenty-one patients with HD, diagnosed on the basis of a positive family history, evidence of caudate atrophy on computer tomography and fulfilling the clinical criteria for the illness (Folstein, 1989) consented to participate after full disclosure of all risks and potential benefits associated with this study (Table Ia). Results of genetic testing were unavailable for this group. Data from 19 of these individuals have been reported in a previous study (Mohr *et al.*, 1991). Nineteen patients with idiopathic PD, diagnosed on the basis of the presence of at least two of the three cardinal features of the illness (tremor, rigidity and bradykinesia) and the absence of any known cause of secondary Parkinsonism also provided informed consent for study participation (Table Ia). Presence and severity of dementia was evaluated in all patients prior to study entry with the Mattis Dementia Rating Scale (Lukiw *et al.*, 1987) and DSM-III-R criteria for dementia (American Psychiatric Association, 1987). HD patients were matched for dementia severity to the 11 PD patients identified as demented on the basis of the aforementioned criteria (Table Ib).

None of the study subjects was on a regular regimen of centrally active medication for at least two months prior to study entry with the exception of dopamine-enhancing agents used by PD patients. None of the latter was treated with anticholinergics or monoamine oxidase B inhibitors. Dopamine precursor therapies were withheld at least three hours prior to neuropsychological testing and assessments proceeded only after a neurological examination deemed a patient 'off', in order to test the presence of disease-specific impairments reliably. The scoring of tests was not dependent on speed and accuracy of motor function. In the case of the one exception to this rule (Performance subtests of the WAIS-R which were used in the examination of visuospatial cognition), age-scaled norms were utilised for purposes of data analysis. The appropriate dose of levodopa/carbidopa was reinstated immediately following test completion.

Nineteen neurologically healthy subjects whose status was determined by history and physical and

TABLE Ia: Demographics of study subjects (mean years ± SEM, range)

	Normal controls	Parkinson's Non-demented	Parkinson's Demented	Huntington's
Male/Female	10/9	6/2	10/1	11/10
Age	43.0 ± 2.8 (20–65)	62.1 ± 3.2 (51–77)	66.4 ± 2.5 (46–76)	45.8 ± 2.8 (25–75)
Education	14.6 ± 0.7 (7–20)	15.6 ± 0.7 (12–18)	15.8 ± 0.9 (12–20)	13.6 ± 0.7 (6–20)
Symptom duration		11.3 ± 1.3 (6–17)	12.5 ± 1.5 (4–18)	8.0 ± 1.1 (1–22)

TABLE Ib Intellectual and memory profiles (mean ± SEM, range)

	Normal controls N = 19	Non-demented PD N = 8	Demented PD N = 11	HD N = 21
WAIS-R Verbal IQ	116 ± 3.4 (86–146)	115 ± 4.1 (100–136)	94 ± 3.8 (65–115)[a,b]	88 ± 2.7 (67–111)[a,b]
WAIS-R Performance IQ	111 ± 2.7 (86–133)	98 ± 3.4 (86–115)	83 ± 3.7(70–104)[a]	81 ± 3.3 (58–120)[a,b]
WAIS-R Full Scale IQ	115 ± 3.1 (87–142)	110 ± 4.0 (93–129)	95 ± 4.8 (81–133)[a]	84 ± 2.9 (59–111)[a,b]
Wechsler Memory Scale	120 ± 4.0 (81–143)	123 ± 5.6 (101–143)	93 ± 6.1 (64–143)[a,b]	85 ± 4.4 (61–132)[a,b]
Mattis Dementia Rating Scale	142 ± 0.4 (140–144)	141 ± 0.6 (139–143)	127 ± 2.4 (116–137)[a,b]	125 ± 2.8 (93–143)[a,b]

[a]different from normal controls at $p < 0.05$, Bonferroni correction;
[b]different from non-demented PD at $p < 0.05$, Bonferroni correction.

neurological examination also provided informed consent for study participation. They were matched to HD patients for age, and to both HD and PD patients for educational attainment (Table Ia).

Intellect and memory were assessed in all subjects with the Wechsler Adult Intelligence Scale-Revised (WAIS-R; (Wechsler, 1981)) and the Wechsler Memory Scale (WMS; (Wechsler and Stone, 1945)). The original, rather than the revised version of the latter, was used since the current patient sample was enrolling as part of a longitudinal study which commenced when the Wechsler Memory Scale-Revised (Wechsler, 1987) was not yet available. Visuospatial cognition was examined by neuropsychological tests shown to be sensitive for assessment of this realm and included tests of perceptual organisation, non-verbal reasoning, visual attention to detail, visuospatial concept formation and visuospatial integration (WAIS-R) as well as field dependence, visual orientation, manipulation of person and object-centred space, and consistency of visual judgment as described below. The Embedded Figures Test examines field dependence and visual search by asking subjects to identify a simple geometric design embedded in one of four complex patterns (Kapur and Butters, 1977). The Rod and Frame Test examines field dependence of upright orientation by asking subjects to set upright, from a 20° left or right tilt, a rod within a tilted frame without reference points (Oltman, 1968). The Mental Rotation Test examines manipulation of object-centred space by asking subjects to mentally rotate complex geometric patterns represented three-dimensionally to compare with a target design (Shepard and Metzler, 1971; Vanderberg and Kuse, 1978). The Street Map Test examines simple right and left orientation and manipulation of person-centred space by asking subjects to identify right and left turns on a simulated map of a small town with orientation facing initially away, then towards the subject (Money, 1976). The Mental Reorientation Test examines complex right and left orientation by asking subjects to identify which hand of a represented figure, rotated in the front/back, left/right and up/down planes, is black (Ratcliff, 1979). The Extended In-Front-Of Test examines consistency of visual judgment, field dependence and manipulation of person-centred space by asking subjects to identify the location in front, behind and next to an object or person(s) placed in the centre of a 3×3 grid. The object (which may or may not have an inherent directionality, i.e. ball versus car or chair) or person(s) are changed in their orientation from picture to picture, and the influence of type and orientation of the centre figure on the choice of the 'in front', 'behind' and 'next to' locations is measured. Consistency indicates that subjects did not alter their choice, irrespective of the centre figure (Vaid *et al.*, 1979).

Data analysis was performed in three segments. In the first, differences in intellectual, memory and visuospatial performance among all four study groups (HD, demented PD, non-demented PD and controls) were analysed with analysis of variance using Bonferroni-adjusted significance levels. Since multiple test scores can be derived from the visuospatial tests in the battery, in those cases where within-task correlations were significant ($r > 0.6; p < 0.01$), variables were pooled and summary scores used to reduce the number of measures. The second segment used factor analysis of the visuospatial processing variables to identify the presence of separate domains of visuospatial function. Two data sets were analysed; firstly, data from all of the Parkinsonian patients (demented and non-demented) and secondly, data from all demented patients (PD and HD). Principal components factor analysis was calculated with varimax rotation. Factors which explained at least 10% of the common variance were maintained. This figure of 10% or greater denotes an Eigen value of at least 1.6. The third segment compared the patients' factor scores using t-tests. A comparison between the two factor solutions using correlational analysis was also performed. In addition, Alpha Factoring and Carmines Theta (Kim and Mueller, 1978) were performed to evaluate the consistency between the solutions using different factor techniques.

While older patients tend to perform less well on neuropsychological tests, age was not used as a variable in the data analysis. Neither intellectual, memory nor visuospatial cognitive profiles were different for the non-demented PD and normal control comparison. For the HD and demented PD comparison, there was only one test in which the younger HD patients scored significantly better than the older demented PD patients. Moreover, these patients were matched for overall dementia severity. Thus, it is unlikely that age played a role in the findings.

RESULTS

Global intellectual and memory status

Intellectual (WAIS-R Full Scale, Verbal and Performance IQs) and memory (WMS MQ) function were significantly compromised in both demented PD and HD patients compared to controls ($p < 0.05$, Table Ib). Scores on these tests did not differ significantly between non-demented PD patients and controls.

TABLE Ic. Visuospatial cognition profiles (mean ± SEM, range)

	Normal controls N = 19	Non-demented PD N = 8	Demented PD N = 11	HD N = 21
WAIS-R				
Picture Completion	11 ± 0.4 (8–14)	11 ± 0.7 (8–14)	9 ± 0.8 (6–13)	8 ± 0.6 (4–14)[a,b]
Block Design	12 ± 0.7 (7–18)	12 ± 0.5 (10–14)	7 ± 1.0 (3–13)[a,b]	8 ± 0.6 (2–12)[a,b]
Picture Arrangement	12 ± 0.5 (8–16)	11 ± 0.8 (8–14)	8 ± 1.0 (2–14)[a]	8 ± 0.6 (3–15)[a]
Digit Symbol	12 ± 0.6 (6–16)	10 ± 0.8 (6–12)	6 ± 1.0 (1–11)[a,b]	6 ± 0.6 (2–11)[a]
Object Assembly	12 ± 0.6 (7–16)	11 ± 0.9 (6–14)	6 ± 0.7 (3–10)[a,b]	6 ± 0.6 (1–11)[a,b]
Mental Rotation Test	23 ± 2.9 (4–40)	14 ± 4.0 (2–36)	6 ± 1.0 (0–11)[a]	8 ± 1.2 (0–18)[a,b]
Street Map Test				
Overall Correct	15 ± 0.2 (12–16)	15 ± 0.4 (13–16)	13 ± 0.7 (9–15)	11 ± 0.8 (1–15)[a,b]
Away from self – toward self	1 ± 0.3 (–1–5)	1 ± 0.4 (0–2)	1 ± 0.8 (–5–6)	3 ± 0.7 (–3–8)
In–Front–Of Test Consistency				
Orientation to front	3 ± 0.2 (1–4)	3 ± 0.4 (1–4)	2 ± 0.4 (0–4)	3 ± 0.2 (1–4)
Orientation to back	3 ± 0.2 (1–4)	3 ± 0.4 (1–4)	2 ± 0.3 (0–4)	3 ± 0.2 (1–4)
Orientation to person & group	1 ± 0.2 (0–2)	1 ± 0.3 (0–2)	1 ± 0.1 (0–1)	1 ± 0.1 (0–2)
Overall consistency	0 ± 0.0 (0–1)	0 ± 0 (0–1)	1 ± 0.3 (0–4)	2 ± 0.5 (0–6)[a]
Rod and Frame Test				
Frame orientation left	2 ± 0.2 (1–5)	3 ± 0.8 (1–8)	6 ± 1.0 (2–14)[a]	6 ± 1.1 (1–16)[a]
Frame orientation right	1 ± 0.2 (1–4)	2 ± 0.3 (1–4)	5 ± 0.8 (2–10)[a,b]	4 ± 0.7 (1–13)[a]
Mental Reorientation Test	8 ± 0.1 (7–8)	8 ± 0.2 (7–8)	6 ± 0.5 (4–8)[a,b]	5 ± 0.4 (3–8)[a,b]
Embedded Figures Test	18 ± 0.3 (14–20)	15 ± 1.0 (12–20)	10 ± 1.2 (3–17)[a,b,c]	13 ± 0.9 (5–19)[a]

[a]different from normal controls at $p < 0.05$, Bonferroni correction;
[b]different from non-demented PD at $p < 0.05$, Bonferroni correction;
[c]different from HD at $p < 0.05$, Bonferroni correction.

Both demented PD and HD patients performed more poorly than the non-demented PD patients on verbal and memory function (VIQ and MQ; $p < 0.05$). HD patients also showed significant decrements relative to non-demented Parkinsonians on global intellectual function (FSIQ) and global visuospatial processing (PIQ; $p < 0.05$). As expected, due to matching on the basis of the Mattis Dementia Rating Scale, no significant differences emerged between the two demented patient groups on any global measure of cognitive function.

Visuospatial cognition

Comprehensive visuospatial testing revealed general deficits in function for both demented PD and HD patients relative to both controls and non-demented PD patients ($p < 0.05$; Table Ic). This was evident on the visuospatial subtests of the WAIS-R and the Mental Reorientation Test. In some instances, significant differences were limited to the comparison between controls and demented PD and HD patients, as in the case of the Mental Rotation Test and the Rod and Frame Test (Table Ic).

Several specific differences are also worth noting. Only the HD patients were impaired on the Street Map Test compared to both controls and non-demented PD patients and HD patients showed a greater overall consistency compared to controls on the In-Front-Of Test. On the Embedded Figures Test, in contrast, demented PD patients scored significantly lower than controls, non-demented PD and HD patients (Table Ic).

Factor analysis: PD patients with and without dementia

Three factors, together accounting for 69% of the variance, were maintained when visuospatial performance of the two PD groups was analysed. Factor 1 explained 41% of the variance (varimax rotation) (associate alpha 0.96). The WAIS-R performance subtests, Mental Reorientation Test, Embedded Figures Test, Street Map Test, part of the In-Front-Of Test and the Rod and Frame Test (negatively) all loaded at levels greater than 0.50 on this factor (Table IIa). Measures of field dependence (parts of the Rod and Frame Test, positively, and the In-Front-Of Test) loaded on factor 2 which explained 15% of the variance (associate alpha 0.80). Finally, manipulation of object-centred space (Mental Rotation Test) and tests of person-centred space (accuracy difference between 'away' versus 'toward' direction on the Street Map Test, negatively, and consistency on part of the In-Front-Of Test) loaded on factor 3, which explained 13% of the variance (associate alpha 0.54).

A comparison of the demented and non-demented PD patients on these three factors using factor scores

TABLE IIa. Factor solution for demented PD and non-demented PD patients

Factor	Analysis	
	PCA & varimax	α factor & varimax
Factor 1 (41% of variance)		
WAIS-R Object Assembly	.898	.908
WAIS-R Picture Arrangement	.874	.865
WAIS-R Digit Symbol	.861	.834
WAIS-R Block Design	.843	.844
Mental Reorientation Test	.799	.783
WAIS-R Picture Completion	.792	.732
Embedded Figures Test	.780	.793
Street Map Test (overall correct)	.743	.690
In-Front-Of Test (consistency of orientation to back)	.615	.593
Rod and Frame Test (frame orientation left)	−.595	−.595
Rod and Frame Test (frame orientation right)	−.563	−.521
Factor 2 (15% of variance)		
In-Front-Of Test (overall consistency)	−.846	−.689
In-Front-Of Test (consistency of orientation to front)	.831	.805
Rod and Frame Test (frame orientation left)	.571	.472
Factor 3 (13% of variance)		
In-Front-Of Test (consistency of orientation to person and group)	.833	.629
Mental Rotation Test	.578	.379
Street Map Test (away from self – toward self)	−.571	−.447

Carmines theta of internal consistency = 0.9110; coefficient α for factor 1 = 0.96, 2 = 0.80, 3 = 0.54.

revealed significant differences only on factor 1 ($p < 0.01$; Table IIb) while no differences between these two groups could be ascertained on factors 2 or 3.

Factor analysis: Demented Parkinson's and Huntington's patients

Three factors, together accounting for 60% of the variance, were maintained when visuospatial performance of the two demented patient groups (PD and HD) was analysed. Factor 1 explained 31% of the variance (varimax rotation) (associate alpha 0.95). All WAIS-R Performance subtests, the Mental Reorientation Test and the Embedded Figures Test loaded greater than 0.60 on this factor (Table IIIa). Measures of manipulation of person-centred space (accuracy difference between 'away' and 'toward' direction on the Street Map Test and part of the In-Front-Of Test) and field dependence (Rod and Frame Test, negatively) loaded on factor 2, which explained 19% of the variance (associate alpha 0.84). Finally, factor 3, which explained 10% of the variance (associate alpha 0.59), loaded on the manipulation of object-centred space (Mental Rotation Test) and, negatively, on overall consistency on the In-Front-Of Test.

TABLE IIb. Factor solution: Contrasts of demented and non-demented PD

	Demented PD N = 11	Non-demented PD N = 8
Factor 1	−.563 (.263)	.774 (.196)*
Factor 2	−.115 (.350)	.158 (.272)
Factor 3	−.300 (.243)	.412 (.405)

*non-demented PD different from demented PD at $p < 0.01$.

Comparing demented PD and HD patients on these three factors using factor scores revealed statistically significant differences between groups only on factor 2 ($p < 0.05$; Table IIIb), while no statistically significant results emerged for factors 1 and 3.

Correlations of factor solutions

The two factor solutions (non-demented and demented PD patients and demented PD and HD patients) were compared using correlational analysis. Factor 1 of both solutions was significantly correlated ($r = 0.92$; $p < 0.01$). In addition, factor 2 of the PD (non-demented and demented) patients' solution correlated significantly with factor 3 of the demented (PD and HD) patients' solution ($r = 0.62$; $p < 0.05$). Other correlations between factors failed to reach statistical significance.

TABLE IIIa. Factor solution for HD and demented PD patients

Factor	Analysis	
	PCA & varimax	α & varimax
Factor 1 (31% of variance)		
WAIS-R Picture Arrangement	.876	.823
WAIS-R Picture Completion	.870	.890
WAIS-R Block Design	.848	.825
WAIS-R Object Assembly	.775	.705
WAIS-R Digit Symbol	.767	.732
Mental Reorientation Test	.665	.677
Embedded Figures Test	.617	.568
Factor 2 (19% of variance)		
In-Front-Of Test (consistency of orientation to front)	.794	.774
Rod and Frame Test (frame orientation right)	−.789	−.789
In-Front-Of Test (consistency of orientation to back)	.753	.638
Rod and Frame Test (frame orientation left)	−.642	−.608
In-Front-Of Test (consistency of orientation to person and group)	.547	.442
Street Map Test (away from self - toward self)	.458	.350
Factor 3 (10% of variance)		
Mental Rotation Test	.787	.509
In-Front-Of Test (overall consistency)	−.583	−.449

Carmines theta of internal consistency = 0.8686; coefficient α for factor 1 = 0.95, 2 = 0.84, 3 = 0.59.

TABLE IIIb. Factor solution: Contrasts of HD and demented PD

	HD N = 21	Demented PD N = 11
Factor 1	−.020 (.215)	.037 (.325)
Factor 2	.307 (.231)*	−.586 (.162)
Factor 3	.090 (.251)	−.171 (.189)

*HD different from demented PD at $p < 0.05$.

DISCUSSION

The commonly asserted notion that basal ganglia disease results in decrements in visuospatial function has received some support from this study but several important qualifiers were evident. While the presence of dementia appears to be the single most important determinant of visuospatial deficits, relative decrements do not appear to be uniform. Our data suggest possible differential effects specific to circumscribed basal ganglia nuclei rather than an undifferentiated effect of dementia only. Simple contrasts of individual test results were inadequate for providing insight with respect to functional changes in neural networks secondary to a specific disease. The use of factor analytical techniques allowed for a more detailed evaluation of the neuropsychological consequences of basal ganglia disease.

Presence of dementia affected all spheres of intellectual and memory function with demented patients showing deficits on these measures relative to controls and non-demented PD patients. This is an expected finding, corroborating mental status assessment with more detailed measures of intellectual function and memory. It further demonstrates that demented PD patients can be distinguished from non-demented PD patients on most global measures of intellectual and memory function.

Similarly, on most tasks of visuospatial cognition, both demented patient groups with basal ganglia disease (HD and demented PD) evidenced deficits relative to either controls or non-demented PD patients. These deficits, however, were not uniform. Although demented patients performed comparably on most measures, a number of exceptions were noted. On a test of field dependence (Embedded Figures Test), demented PD patients were impaired relative to those with HD. In contrast, HD patients were significantly impaired relative to both controls and non-demented PD patients on a task of simple right/left orientation (Street Map Test) while demented Parkinsonians did not show any significant decrements in this realm. Similarly, only the performance of HD patients on the In-Front-Of Test indicated higher overall consistency relative to controls. Demented PD patients, however, as well as those with HD, showed significant deficits in right/left orientation when the task became more complex, with rotations involving all planes (Mental Reorientation Test). Therefore, visuospatial task com-

plexity might play a role, with deficits perhaps occurring at lower levels of complexity in patients with HD relative to PD patients with comparable global dementia levels.

Important additional insights were offered by the examination of independent visuospatial domains revealed by factor analysis of two data sets, one looking at patients with comparable motor disease but different dementia status (demented and non-demented PD) and a second examining patients with comparable dementia status but different motor disease (HD and demented PD). Three optimal factors were revealed for each analysis. Correlational cross-comparisons showed that the first factor in both, which also explained the largest part of the observed variance (41% and 31%), was highly correlated ($r = 0.92$). This is suggestive of a common factor which could best be described as global visuospatial accuracy. This factor clearly discriminated between demented and non-demented PD patients but no differences were found between demented PD and HD patients who were matched for dementia severity. Factor 2 from the PD patients' solution was significantly correlated with Factor 3 from the demented patients' solution, again suggesting a common factor. This factor appears to reflect environment- and object-centred spatial judgment as well as field independence, based on variables loadings. Factor 3 from the PD patients' solution was a unique factor, i.e. it had no significant intercorrelations with the other factors, and loaded on measures of object- and person-centred spatial judgement. In the case of the person-centred judgement, both positive and negative loadings were noted. Factor 2 from the demented patients' solution was also a unique factor and, based on factor loadings, seems to reflect manipulation of person-centred space and field independence.

Global visuospatial function (factor 1) distinguished demented from non-demented PD patients while these PD subgroups performed comparably on factor 2 and factor 3. In contrast, neither global visuospatial function (factor 1) nor scores on factor 3 differentiated demented PD from HD patients but significant differences between these two groups were found on factor 2. These results suggest that efficiency of global visuospatial function varies as a function of dementia presence versus absence but it is not diagnostic for specific basal ganglia disease. Certain domain-specific decrements, however, appear more characteristic of a particular disease.

It is remarkable that the only factor which discriminated between demented PD and HD patients was a unique factor for this solution, i.e. the discriminating factor did not significantly correlate with any of the factors in the PD patients' (demented and non-demented) solution. Moreover, the Street Map Test and, more specifically, the difference between 'away' and 'towards' accuracy, loaded positively on this factor. We have shown previously that HD patients are differentially impaired on this measure of person-centred spatial judgement. In a study comparing Alzheimer (AD) to HD patients (Brouwers *et al.*, 1984), we found significant deficits for HD patients on this measure whereas, on measures of environment- or object-centred spatial functioning, AD patients were more impaired. In addition, Potegal (1971) compared HD and PD patients to controls on a related test of person-centred spatial localisation and found HD, but not PD, patients significantly impaired. This author suggested that destruction of the caudate nucleus might be implicated in this deficit. More recent research (Filoteo *et al.*, 1995) has also suggested that different patterns of neuropsychological deficits can be associated with neuropathology in different structures of the basal ganglia as seen in HD and PD.

Both structural and functional correlates of these disease processes may help explain these findings. Global efficiency of visuospatial function affected by the dementing process could reflect cortical dysfunction by way of deafferentation from subcortical structures or a more global atrophic process, also cortically based. In PD dementia, this may be due to cortical effects related to concomitant Lewy body disease, Alzheimer's disease or deafferentation of cortical structures (Mohr *et al.*, 1995). In either case, the functional outcome would yield similar results with respect to decrements in efficiency of visuospatial function. In HD, similar manifestations may be due to cortical atrophy, evident early in the illness but not necessarily linked with disease progression (De La Monte *et al.*, 1988).

Differential involvement of person-centred spatial judgement and field independence in the two demented patient groups could indicate that, while both have reduced cortical efficiency, subcortical structures are differentially involved. In a previous study (Brouwers *et al.*, 1984), comparing performance on spatial tasks between HD and AD patients, findings indicated that ego-centric and person-centred space (Street Map Test) were differentially affected in HD but not in AD, while AD patients were more impaired on construction visuospatial tasks. This suggests that the critical substrate for appropriate performance on the Street Map Test is subcortical and, in the context of current findings, seems to identify factor 2, field dependence and manipulation of person-

centred space, as a specific subcortical factor. Caudate atrophy in HD could underlie the deficits in spatial judgement, while the relatively greater integrity of this structure in those with PD may leave this specific sphere of visuospatial function largely intact, regardless of dementia status.

As a caution, this is an exploratory study and the results have to be viewed within the limitations of the design and methods. Moreover, the sample size of some of the subgroups (particularly the non-demented PD patients) was rather small which reduces the power of the comparisons made involving that subgroup. Nonetheless, impairment of visuospatial cognition in basal ganglia disease appears to have several components. Global decline is present as a function of dementia, while domain-specific decrements may reflect involvement of circumscribed structures characteristic of specific basal ganglia diseases. Further studies corroborating these findings with functional brain imaging technology are required.

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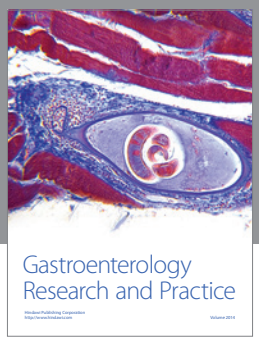

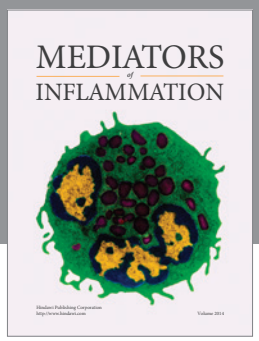

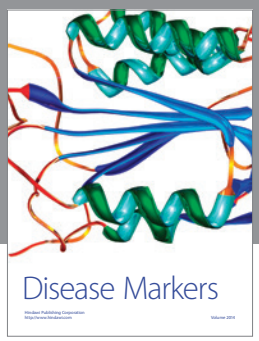

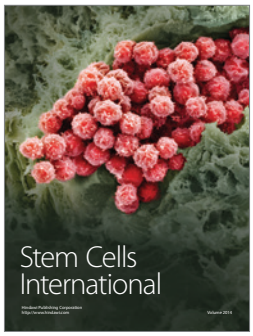

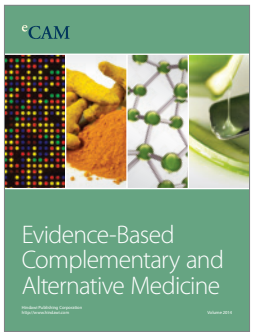

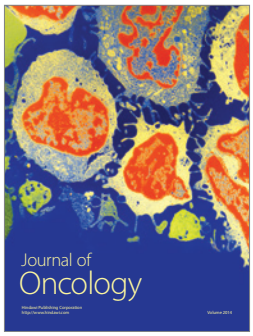

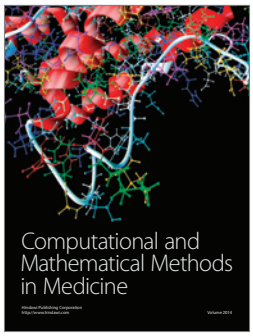

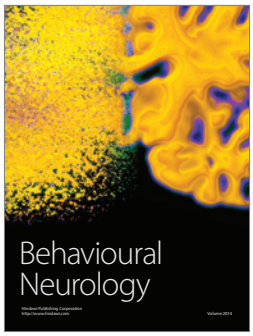

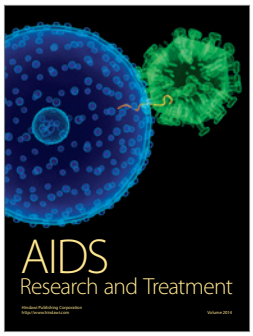

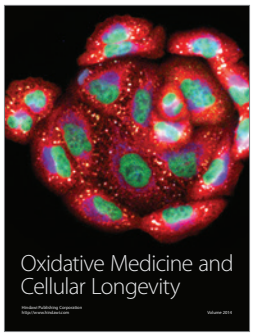

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