Impairment of motor planning in patients with Parkinson's disease: evidence from ideomotor apraxia testing

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SUMMARY Compared with a group of age matched controls, patients with Parkinson's disease scored significantly lower in testing for ideomotor apraxia. Imitation of movement sequences was affected more severely than performance of single movements. The degree of impairment was not related to severity of motor disability, but correlated strongly with the results of tests that measured visuospatial and visuoperceptive abilities. It is suggested that defective encoding and central processing of visuospatial information impairs memory for movement which is necessary for correct imitation of movements. Enhanced vulnerability to interference between successively presented items may cause further deterioration of performance in the copying of movement sequences.

Ideomotor apraxia has been defined as a failure to produce either a correct movement on verbal command or to imitate correctly a movement performed by the examiner when the incorrect performance cannot be explained by weakness, incoordination, akinesia, abnormal reflexes, impaired auditory comprehension, or impaired visual or tactile perception.[1] This had led to the conclusion that ideomotor apraxia is an inaccurate term to associate with Parkinson's disease because the patients manifest motor deficits.[2]

With regard to the examination of motor behaviour the distinction between apraxic errors and lower level deficits of motor control has been expressed as follows: "The diagnosis of ideomotor apraxia is not made if a patient fails to carry out certain movements at all, nor is mere clumsiness of movement sufficient to consider a patient apraxic. The diagnosis is made when a patient is impaired in the proper selection of the motor elements which constitute a movement and in the correct ordering of these elements in a motor sequence".[3] Errors that fit into this definition have been observed in patients with Parkinson's disease and seem to indicate a deficit in central motor planning.[2 4–6] The aim of the present study was to find out whether such a deficit would show up in con-

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ventional testing of ideomotor apraxia and to look into the relationship between apraxia scores and motor disability, visuospatial deficits, and general intellectual deterioration. Instead of discussing whether the demonstrated impairment in apraxia testing warrants a diagnosis of ideomotor apraxia, we thought that a more interesting question would be whether concomitant basal ganglia damage might be a contributing factor to ideomotor apraxia in patients with left hemisphere lesions affecting the cortex and the fibre systems connecting cortical areas.

Subjects

Forty two patients (17 female, 25 male) with idiopathic Parkinson's disease were examined. Twenty eight of them were out-patients, 14 were examined during hospitalisation for therapeutic reasons. Motor symptoms were bilateral in all cases, and their distribution was symmetric in 23. In 15 patients the right side of the body was more severely affected and four patients showed greater impairment on the left. All patients had at least some degree of bradykinesia, rigor, and gait disturbance, and tremor was conspicuous in 26 of them. Mean duration of illness was 8·95 years, and 14 patients had had symptoms for more than 10 years. Motor disability (see below) was rated mild for seven patients, medium for 18, and severe for 17, but no patient was confined to wheelchair or bed during the period of examination. All patients were treated with levodopa and benseracid; 18 of them also received anticholinergic drugs.

The control group consisted of 38 neurological patients (18 female, 20 male) who suffered from disease of the muscles, the peripheral nerves, or the spinal cord; none of

them showed evidence of cerebral damage. Fifteen were outpatients and 23 were hospitalised during the period of examination.

Methods

Motor disability was rated by one of the authors who was not aware of the results of the neuropsychological examinations. According to the criteria proposed by Birkmayer and Neumayer,[7] [8] patients were grouped for mild, medium, and severe disability.[8] Evaluation with this scale considers impairment in starting, walking, balance, posture, writing, speech, and facial expression. As a rough guide, a patient classified as "mildly impaired" falls within stage 1 or 2 of the Hoehn and Yahr scale,[9] and a severe impairment corresponds to stages 4 and 5 of this scale.

Neuropsychological examinations were administered during two sessions within one week. The test battery was chosen to measure general intellectual deterioration and dementia, visuospatial abilities, and ideomotor apraxia:

Memory was examined by free recall of a list of 12 unrelated concrete nouns and a list of 12 two-digit numbers. The score was the number of items correctly recalled after one presentation. Vocabulary was assessed by the Boston Naming Test (BNT)[10] which had been revised for use with German speaking subjects. The WAIS subtest picture completion was administered and evaluated in the usual way. The Rorschach test was given, and signs of organic brain syndrome were rated on a 4-point scale according to the criteria proposed by Piotrowsky.[11] Critical flicker fusion was measured as an index of vigilance and concentration.

Visuospatial abilities were evaluated by four tests: (1) Judgement of line orientation was scored without the proposed corrections for age, sex and educational level.[12] This test demands the estimation of the directions of two lines by comparison with a set of 11 angles. (2) Ratcliff's mannekin test of mental rotation,[13] which requires the distinction between the right and the left side of a human figure seen in different positions, was extended to include profile views of the mannekin in addition to frontal and dorsal views. The score was the number of correct responses for all positions, but subjects were asked to correct when they committed errors for the upright–dorsal position. (3) Assembly of geometric figures required the patient to copy geometric line patterns by means of four to six quadratic plates, each having either an oblique or a straight line on it. The maximum score of each item was half the number of plates involved. If the patient committed errors but corrected after comparison with the model, one point was subtracted; in case of persistent error no point was given. (4) The WAIS subtest figure assembly was administered without time limits, and neither Parkinson's disease patients nor controls received additional points for quick performance. In both assembly tests inaccuracy caused by temor was neglected for scoring.

In apraxia testing the patient was free to choose the preferred hand. There were four imitation items: six finger postures, each involving two or three fingers (for example form a ring with thumb and small finger; extend the thumb, the index and the small finger); six positions of the hand (for example hold the hand vertically under the chin with the ulnar side looking forwards;[14] touch the contralateral cheek with the back of the hand); six movement sequences which

required different combinations of simultaneous movements of arm, hand and fingers (for example touch the upper arm with the ulnar edge of the hand, then touch the forearm with the same ulnar edge, then touch the upper arm with the palm;[15] make a fist beside the head, then extend the arm and form a ring with thumb and middle finger;[14] point with the index to the ipsilateral eye, then place the palm upon the contralateral shoulder); and a sequence of finger tapping (opposing once the index, twice the middle finger, once the ring finger, twice the small finger to the thumb) which was demonstrated simultaneously to counting the number of tappings out loud. In all imitation items the patient was allowed to start performance only after the examiner had terminated the demonstration. Two points were given for a correct performance. In case of failure, demonstration was repeated and one point was credited for a correct performance on second trial. The finger tapping sequence was demonstrated in up to three trials and hence a maximum of three points could be obtained.

Six symbolic gestures and six pantomimes of object use were examined on verbal command only. Again, correct performance on first trial yielded two points. In case of failure the meaning of the gesture or the demand to pantomime the use of an implement was reexplained, and one point was credited for a correct second trial. Generally, slowness of execution and disturbance of movement by tremor or reduced motility were not considered as failures. The prevailing types of errors were the same in Parkinsonians and controls: confusion of fingers in imitation of finger postures; spatial errors in imitation of hand positions and of whole limb movement sequences, frequently occurring already with the first movement of a sequence; omission of tapping with the fourth and fifth finger and loss of the 1–1, 2–1–1, 2 rhythm in the finger tapping sequence; omission or incorrect patterns of movement of single digits in symbolic gestures, and body part as object errors in pantomine of object use.

Statistical analysis

Rating of organic brain syndrome and of motor disability obviously operated on an ordinal scale level. The interval scale level of some of the other subtests could be disputed, because rating was based on a combination of different criteria. For example, scoring combined the number of correct items with an evaluation of whether a task was achieved with or without correction. Therefore, nonparametric statistics based on rankings were choosen to determine the significance level of differences and to estimate the strength of correlations. Because multiple comparisons and correlations were being computed for the same data set a probability of 0·001 was considered to be a maximum for interpreting a single result as being significant. In interpreting partial correlations, however, a 0·01 level of significance was accepted since this procedure controls more strictly for common influences of nonspecific factors.

Results

Within each group scores of finger and hand positions as well as of geometric figure and WAIS figure assembly showed no significant differences (Wilcoxon test for repeated measures, p > 0·20). They were col-

Table 1 *Comparison between groups*

	Parkinsonian patients		Controls		p (Mann-Whitney U-test)
	Mean	Range	Mean	Range	
Age	67,6	35–83	66,3	41–86	0·47
Memory	7,6	0–15	9,1	4–18	0·10
Organic brain syndrome	1,3	0–3	0,9	0–3	0·089
Picture completion	9,3	1–14	10,2	6–15	0·29
Vocabulary (BNT)	49,6	22–60	52,2	40–60	0·28
Flicker fusion	36,0	29–42	37,1	33–42,5	0·064
Line orientation	17,8	0–30	21,3	11–29	0·046
Mental rotation	28,0	14–31	29,1	20–32	0·64
Figure assembly	23,3	1–35	28,8	11–35	0·0095
Finger and hand positions	19,8	11–24	21,4	16–24	0·039
Finger tapping sequence	1,6	0–3	2,4	0–3	0·0051
Movement sequences	7,6	1–12	9,7	4–12	0·0001
Symbolic gestures	11,5	8–12	11,8	10–12	0·32
Pantomime of object use	9,4	0–12	10,0	5–12	0·48
Apraxia score	49,2	20–62	55,0	44–62	0·0003

lapsed to "figure assembly" and "finger and hand positions" respectively.

Comparison between groups

The mean results of Parkinson's disease patients were worse than those of controls in all tests, but the differences reached the 0·001 level of significance only in whole limb movement sequences and the total apraxia score (table 1).

Correlation within groups

Only in the control group did females score significantly lower than males in WAIS picture completion (p = 0·0012), and judgement of line orientation (p = 0·0012). In the patient group there were no significant correlations between test scores and duration of illness, nor were there any significant differences pertaining to lateralisation of motor symptoms, presence of marked tremor, or medication with anticholinergic drugs.

In both groups there were significant correlations between picture completion and the BNT on the one hand and tests of visuospatial abilities on the other (table 2). These correlations were somewhat stronger

Table 2 *Relationship of visuospatial abilities to age, motor symptoms, and intellectual decline*

	Line orientation	Mental rotation	Figure assembly
	Parkinsonian patients		
Age	−0·32	−0·17	−0·40
Motor disability	−0·45	−0·19	−0·44
Memory	0·42	0·54*	0·48*
Organic brain syndrome	−0·36	−0·49	−0·28
Picture completion	0·59*	0·39	0·70‡
Vocabulary (BNT)	0·54*	0·39	0·70‡
Flicker fusion	0·27	9·16	0·33
	Controls		
Age	−0·34	−0·30	−0·33
Memory	0·046	0·35	0·25
Organic brain syndrome	−0·0070	−0·13	−0·080
Picture completion	0·46	0·57*	0·57*
Vocabulary (BNT)	0·41	0·059	0·54*
Flicker fusion	0·37	0·30	0·40

Spearman correlation coefficients; *p < 0·001, †p < 0·0001, ‡p < 0·00001.

Table 3 *Relationship of general intellectual decline to age and motor symptoms*

	Memory	Organic brain syndrome	Picture completion	Vocabulary (Picture naming)	Flicker fusion
			Parkinsonian patients		
Age	−0·35	0·33	−0·20	−0·43	−0·50*
Duration of illness	−0·036	0·067	−0·093	−0·053	−0·040*
Motor disability	−0·031	0·25	−0·53*	−0·27	−0·0053
			Controls		
Age	−0·49*	0·14	−0·31	−0·18	−0·63†

Spearmen correlation coefficients; *p < 0·001, †p < 0·0001.

Table 4   *Relationship of Apraxia scores to age, motor symptoms and intellectual decline*

	Finger & hand positions	Finger tapping sequence	Movement sequence	Symbolic gestures	Pantomime of object use	Apraxia score
			Parkinsonian patients			
Age	−0·16	−0·33	−0·19	−0·033	−0·21	−0·36
Motor disability	−0·27	−0·36	−0·21	−0·24	−0·12	−0·32
Memory	0·37	0·55*	0·23	0·013	0·17	0·46
Psychoorganic syndrome	0·23	0·40	0·14	0·12	0·13	0·32
Picture completion	0·48*	0·50*	0·21	0·32	0·22	0·51*
Vocabulary (BNT)	0·48*	0·50*	0·39	0·17	0·45	0·50*
Flicker fusion	0·22	0·23	0·22	0·17	0·35	0·31
			Controls			
Age	−0·16	−0·16	−0·37	−0·30	−0·21	−0·29
Memory	0·30	0·053	0·54*	·0·085	0·42	0·54*
Psychoorganic syndrome	0·23	0·012	0·099	0·32	0·17	0·080
Picture completion	0·40*	0·37	0·59*	0·045	0·26	0·58*
Vocabulary (BNT)	0·31	0·33	0·37	0·078	0·23	0·51*
Flicker fusion	0·15	0·11	0·48	0·33	0·24	0·30

Spearman correlation coefficients; *p < 0·001.

Table 5   *Relationship between Apraxia scores and visuospatial abilities*

	Finger & hand positions	Finger tapping sequence	Movement sequence	Symbolic gestures	Pantomime of object use	Apraxia score
			Parkinsonian patients			
Line orientation	0·64	0·39*	0·49*	0·27	0·47*	0·62‡
Controlling for Age	0·63	0·32	0·46*	0·29	0·43*	0·56†
Memory	0·57**	0·22	0·45*	0·30	0·45*	0·52†
Picture completion	0·51**	0·14	0·46*	0·10	0·43*	0·45*
Vocabulary (BNT)	0·51**	0·16	0·36	0·21	0·30	0·47*
Age, memory, picture completion and vocabulary	0·45*	−0·057	0·40*	0·16	0·37	0·35
Mental rotation	0·52†	0·48*	0·42*	0·23	0·47*	0·62‡
Controlling for Age	0·51*	0·46*	0·40*	0·24	0·12	0·54†
Memory	0·42*	0·27	0·36	0·28	0·071	0·41*
Picture completion	0·42*	0·36	0·37*	0·12	0·068	0·45*
Vocabulary (BNT)	0·42*	0·36	0·31	0·18	−0·034	0·45*
Age, memory, picture completion and vocabulary	0·34	0·18	0·33	0·20	−0·0059	0·35
Figure assembly	0·68‡	0·64‡	0·48*	0·31	0·43*	0·72‡
Controlling for Age	0·68‡	0·59‡	0·44*	0·35	0·38*	0·67‡
Memory	0·62‡	0·52†	0·43*	0·36	0·40*	0·64‡
Picture completion	0·54†	0·48*	0·47*	0·14	0·39*	0·59†
Vocabulary (BNT)	0·54†	0·48*	0·31	0·28	0·17	0·60‡
Age, memory, picture completion and vocabulary	0·48*	0·32	0·37*	0·22	0·25	0·48*
			Controls			
Line orientation	0·14	0·093	0·34	−0·083	0·37	0·28
Controlling for Age	0·095	0·041	0·24	0·018	0·32	0·20
Memory	0·14	0·090	0·37	−0·088	0·39*	0·30
Picture completion	−0·054	−0·095	0·90	−0·12	0·29	0·012
Vocabulary (BNT)	0·016	−0·049	0·22	−0·13	0·31	0·087
Age, memory, picture completion and vocabulary	−0·50	−0·22	0·15	0·017	0·40*	0·043
Mental rotation	0·088	0·25	0·52†	0·046	−0·018	0·20
Controlling for Age	0·041	0·21	0·46*	0·15	−0·089	0·12
Memory	−0·018	0·25	0·42*	0·017	−0·19	0·013
Picture completion	−0·19	0·052	0·29	0·024	−0·21	−0·19
Vocabulary (BNT)	0·073	0·24	0·54†	0·04	−0·032	0·20
Age, memory, picture completion and vocabulary	−0·20	0·15	0·26	0·078	−0·29	−0·22
Figure assembly	0·25	0·30	0·47*	0·050	0·16	0·43*
Controlling for Age	0·21	0·26	0·40*	0·17	0·094	0·37
Memory	0·19	0·29	0·41*	0·030	0·059	0·37
Picture completion	0·026	0·11	0·21	0·030	0·012	0·15
Vocabulary (BNT)	0·10	0·15	0·35	0·97	0·041	0·22
Age, memory, picture completion and vocabulary	−0·012	0·034	0·20	0·11	−0·029	0·080

Partial correlations computed from correlation matrix of Spearman correlation coefficients; *p < 0·01, †p < 0·001, ‡p < 0·0001.

in the Parkinson's disease group, and only in Parkinson's disease patients did the relationship between memory and visuospatial tests reach significance as well. Most likely these results indicate an influence of general intellectual decline on visuospatial abilities.

In Parkinson's disease only picture completion showed a significant correlation to motor disability (table 3), but the respective correlations of line orientation and figure assembly were close to the chosen significance level (table 2). Picture completion requires the evaluation of line drawings and may be sensitive to visuoperceptive impairment. The correlations may thus be interpreted as being a partial confirmation of previous findings of a progressive decline of visuospatial and visuoperceptive abilities with advancing stages of motor disability.[16-19]

The impairment of Parkinson's disease patients in apraxia testing showed no significant correlation with the disability score (table 4). In both, Parkinsonians and controls, there were significant correlations between verbal memory, picture completion and the BNT on the one hand, and apraxia scores on the other. General intellectual decline seems to have a deleterious influence on performance in apraxia testing.

In Parkinson's disease patients all visuospatial tests correlated strongly with the total apraxia score and with finger and hand positions, and figure assembly showed a significant correlation with the finger tapping sequence as well. In contrast, in controls only the correlation between mental rotation and the whole limb movement sequence reached significance (table 5).

The correlation between visuospatial tests and apraxia scores could have been simulated by a common influence of general intellectual deterioration or age upon both kinds of measures. Therefore, partial correlations were calculated controlling for the respective items and for their possible combinations (table 5). In both groups, a decrease of more than 0·1 in at least one correlation coefficient was obtained by controlling for age, memory, picture completion, and the BNT respectively. Control for motor disability did not affect correlation coefficients in Parkinsonians and control for the results of the Rorschach test and for critical flicker fusion had no distinct effect in either group. In both groups control for the combination of memory, picture completion, the BNT, and age turned out to be most effective in lowering the strength of correlations between visuospatial tests and apraxia scores: In the control group the correlations with the total apraxia score fell to values about zero, and only the correlation between judgement of line orientation and pantomime of object use maintained significance.

In contrast, in the Parkinson's disease group a 0·01

level of significance was maintained in the relationship of figure assembly to the total apraxia score, to finger and hand positions, and to movement sequences, as well as in the correlations of line orientation to finger and hand positions and to movement sequences. The correlations between mental rotation and apraxia scores lost significance. Apparently the relationship between visuospatial abilities and apraxia scores pertains to the exact perception of complex visuospatial information rather than to "spatial thinking".[13] However, this relationship appears to be in part independent of intellectual deterioration and age.

Discussion

In testing for apraxia we took great care to consider only failures that could not be ascribed to akinesia, hypomotility or tremor, and there was no difference in the types of errors committed by patients with Parkinson's disease and by controls. Whereas picture completion which requires no motor responses showed a significant correlation with the disability score, scores in apraxia testing did not. Therefore, the demonstrated impairment in apraxia testing cannot be convincingly explained as being a sequel of insufficient motor execution due to akinesia, hypomotility, or tremor.

With the exception of a few hand positions which required placing of the hand under the chin or beside the head, all movements could be monitored visually by the subject himself, and there were no time constraints on performance. It thus seems improbable that the impairment of Parkinson's disease patients was due to the deficit in "open loop" control of voluntary movements that has been repeatedly described as a sequel of basal ganglia damage.[4 6 20]

The mean scores of Parkinson's disease patients were lower than those of controls in all tests presumed to assess general intellectual deterioration and dementia, but these differences did not reach statistical significance. Keeping in mind that significant declines of memory and vocabulary are already found in mild dementia of the Alzheimer type,[21] one cannot reasonably consider the impairment in apraxia testing of our Parkinson's disease patients as being a symptom of concomitant dementia of the Alzheimer type.

Whether the failure in apraxia testing could be conceived of as being a sequel of defective visuospatial perception seems to be a more difficult question to answer: in accordance with the findings of Shape *et al*[28] we found Parkinson's disease patients to be significantly worse than controls in imitation of movements but not in performance of movements on verbal command. Apraxia scores of Parkinson's disease patients showed strong correlations to the results

of tests that measured visuospatial abilities but did not require a skilled motor response. From these findings it could be presumed that defective imitation of movement resulted from defective perception of the exact spatial features of the demonstated movement. We think, however, that another possible relation between visuospatial deficit and impaired imitation of movement should be considered: patients were allowed to start performance only after demonstration was terminated. Thus, short term memory for movement intervened between perception and performance. When movements are to be memorised they are encoded in terms of their visuospatial features rather than in patterns of motor innervation.[22] Consequently, if there is a deficit in central processing of visuospatial information it should affect memory for movements. A deficit in memory for movement could account for the greater impairment in imitating movement sequences than in imitating single movements, because the total amount of information that has to be stored to memorise a movement sequence is obviously greater than that necessary to memorise a single movement.

Increased sensitivity to interference between successive components of the movement sequences might have added to the deleterious effect of impaired visuospatial encoding on memory for movements in patients with Parkinson's disease. The basal ganglia function in close interaction with the frontal lobes,[6 23] and there is evidence that patients with even mild Parkinson's disease exhibit psychological deficits which resemble those of patients with frontal lobe damage.[24–26] Increased vulnerability to interference between successively presented items of memory tasks has been documented in human patients and in animals with frontal lobe damage.[14 27 28]

Considering the correlations between the verbal memory task and apraxia subtests one notes a great difference between Parkinsonians and controls with regard to the finger tapping sequence: in Parkinsonians there was a significant correlation between memory and finger tapping, whereas in controls there was hardly any correlation at all. The amount of information that has to be memorised for repeating the finger tapping sequence is rather small, consisting of a simple 1–1, 2–1–1, 2 rhythm and the demand to move one finger after the other. On the other hand, interference in this task could be very strong, because a sequence of alternating commands concerns "semantically" closely related body parts, the finger of one hand.

The demonstration of a deficit in apraxia testing of Parkinson's disease patients that cannot be reduced to being a sequel of faulty execution resulting from akinesia, hypomotility, or tremor leads to the question whether basal ganglia damage of other origin might also have a deleterious effect on the central planning and control of intentional movements. Ideomotor apraxia with exclusively subcortical lesions of vascular origin is not frequent but has repeatedly been reported.[29–31] Because of the anatomy of vascular supply, however, ischaemic lesions most oftenly encroach upon cortical as well as subcortical regions. Purely cortical lesions of the left parietal lobe following surgical excision lead only to a mild impairment in copying of complex arm movements that is distinctly less severe than ideomotor apraxia caused by stroke.[32] Kertesz and Ferro[31] looked for patients who had moderate to severe apraxia as a sequel of stroke and in whom CT displayed only small lesions. They found that seven out of nine had subcortical frontal lobe lesions. Lesions in this location may lead to apraxia because they damage both the white matter connecting the occipital with the frontal lobes and the anterior callosal fibres connecting the premotor areas of both hemispheres. They are, however, very likely to encroach upon the anterior basal ganglia as well. The effects of basal ganglia damage may thus contribute to the clinical picture of ideomotor apraxia in patients with more extensive left brain damage.

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