Impairment in visual–spatial function in catatonia: a neuropsychological investigation

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Abstract

Catatonia is a psychomotor syndrome with motor and behavioral abnormalities which may be due to alterations in fronto-parietal cortical function. We therefore investigated neuropsychological tasks (attention, executive, visual–spatial, working memory) associated with frontal and parietal cortical function. Thirteen catatonic patients, diagnosed as catatonic according to criteria by Rosebush and Bush, were compared with 13 psychiatric non-catatonic controls (matched with regard to underlying psychiatric diagnosis, age, sex, and medication), and 13 age- and sex-matched healthy controls. Catatonics showed significantly poorer performances and different neuropsychological intercorrelation patterns in visual–spatial object perception (VOSPobject) than psychiatric and healthy controls. In addition, we found significant correlations between catatonic symptoms, visual–spatial abilities, and attentional measures (i.e., d2, CWI). Catatonia was characterized by specific visual–spatial deficits which are related to attentional abilities and right parietal cortical function. The data suggest attentional–motor and fronto-parietal dysfunction in catatonia, a conclusion which should be considered as preliminary, however, due to the small sample size. © 1999 Elsevier Science B.V. All rights reserved.

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1. Introduction

Catatonia is a psychomotor syndrome with motor and behavioral abnormalities (Kahlbaum, 1874; Taylor, 1990; Fink et al., 1993; Bush et al., 1996; Northoff, 1997) such as complete akinesia, posturing, and other voluntary movement disturbances (Northoff et al., 1995a,b). Unlike Parkinsonian patients, catatonics are not fully aware of their movement disturbances, which are probably related to neuropsychological alterations in attentional–motor interactions (Northoff et al., 1998a). Studies of regional cerebral blood flow (r-CBF) reported reductions in right prefronto-parietal r-CBF in catatonia (Satoh et al., 1993; Galynker et al., 1997; Northoff et al., 1998a,b,c,d) which could account for attentional–motor disturbances. The cortical motor function showed no major abnormalities (Northoff et al., 1998a,b,c,d), whereas attentional abilities have not been investigated yet. We therefore tested attentional abilities (d2, CWI) and further neuropsychological tasks

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associated with fronto-parietal function in 13 cata-
tonic patients, comparing them with 13 non-cata-
tonic psychiatric and 13 healthy controls. Frontal
tasks included various tests for executive function
(trail making test, verbal fluency, Kramer test,
5-point test, box piling) and working memory
(progressive arithmetics). Right and left parietal
measures included various tests for visual–spatial
abilities (observation subtest from the Wilde test,
subtests 7 and 9 from the performance investiga-
tion system); right parietal function, in particular,
was measured using the visual object and space
perception test (i.e. VOSP).

2. Methods

2.1. Subjects

2.1.1. Catatonic patients

We investigated 13 catatonic patients (9 women,
4 men; age: 43.4 ± 13.1 years; means $\pm$ SD; see
Table 1 and 2 for exact details). They were selected
from all admitted inpatients (who were all screened
for catatonic syndrome using criteria by Rosebush)
at the psychiatric university clinic in Magdeburg
and at psychiatric clinics in Haldensleben and
Blankenburg between July 1996 and January 1998

(incidence, calculated in relation to all admitted
inpatients: 2.6%, all showing at least 4 from 12
symptoms according to Rosebush's criteria).

On admission, three patients were neuroleptic-
naive (i.e., they had never received any neuro-
leptics), six were neuroleptic-untreated [i.e., no
neuroleptics in the previous 6 months; treatment
before that with haloperidol (dose range: 5–20 mg)
for an average duration of 1.1 ± 0.4 years], and
one patient received clozapine (3×100 mg). No
significant differences in psychopathological and
neuropsychological measurements were found
between neuroleptically medicated and neuro-
leptically unmedicated (neuroleptic-naive and
neuroleptic-untreated patients) catatonic patients.
In addition, six patients took antidepressants (ami-
triptylin 50–200 mg), two patients received lithium
(serum concentration: 0.9 mmol/l) and one
received carbamazepine (serum concentration
8 µg/ml). None of the patients had taken benzo-
diazepines in the 6 months prior to admission;
those who had (measurement of serum concen-
tration of lorazepam on days 0 and 14 according
to the method by Greenblatt et al., 1978) were
excluded from the study ($n = 3$), since benzodiaz-
epines by themselves can lead to alterations in
neuropsychological functions. Patients with
chronic neurological or other physical illness, alco-

Table 1
Clinical and psychopathological data in individual catatonic patients

Pers	Sex	Age	Educ.	DI	Hosp	AO	TAO	DT	CPZ	AC	GAS	PANSS	HAS	HDS	DSM-IV
1	F	27	11	1	1	26	8	4	80	+	13	73	15	5	296.4c
2	F	27	10	2	2	26	5	2	460	−	17	80	24	13	295.20
3	M	28	10	4	2	24	6	4	280	−	12	103	24	8	295.20
4	M	29	11	11	10	18	8	11	330	+	17	140	19	11	295.20
5	F	38	9	5	4	33	3	3	0	+	16	95	22	14	296.44c
6	F	45	10	11	3	34	4	7	120	+	18	80	24	20	296.54c
7	M	45	13	16	2	29	3	6	100	+	8	38	24	26	295.54c
8	F	47	10	9	3	38	6	9	0	+	17	102	25	10	296.44c
9	F	48	8	13	3	35	2	5	200	+	13	67	14	24	296.54c
10	F	48	10	2	3	46	3	2	95	+	14	53	15	19	296.34c
11	F	54	8	16	6	38	4	16	70	+	10	125	24	13	296.54c
12	M	59	8	16	4	43	4	8	480	−	14	112	14	16	295.20
13	F	69	10	4	2	65	4	1	0	+	19	54	18	15	296.54

DI = duration of illness (years); Hosp = No. of hospitalizations; AO = age of onset; TAO = time since actual onset (weeks); DT =
duration of treatment (years); CPZ = neuroleptics in chlorpromazine equivalents (mg); AC = anticholinergics; GAS = global assessment
scale; PANSS = positive and negative symptom scale; HAS = Hamilton anxiety scale; HDS = Hamilton depression scale.

hol and/or substance abuse, electroconvulsive therapy, unconscious episodes, hyperkinesias and/or dyskinesias as assessed by AIMS (>2; Guy, 1976), and/or neuroleptic-induced movement disorders as assessed by SEPS (>3; Simpson and Angus, 1970) were excluded from the study.

Psychopathological assessments were made using the global assessment scale (GAS; Endicott et al., 1976), the positive and negative symptom scale (PANSS; Kay et al., 1987), the Hamilton anxiety scale (HAM-A; Hamilton, 1959), and the Hamilton depression scale (HAM-D; Hamilton, 1960) on day 0 (before initial treatment) and on day 10 (the week of neuropsychological investigation). All patients were right-handed according to the Edinburgh Inventory of Handedness (Oldfield, 1971). Co-morbid diagnoses were made according to DSM-IV (American Psychiatric Association, 1994) on discharge by two independent psychiatrists with a semi-structured clinical interview.

Catatonic syndrome was diagnosed according to criteria by Lohr (Lohr and Wisniewski, 1987) (3 of 11 symptoms), Rosebush (Rosebush et al., 1990) (4 of 12 symptoms), the Bush–Francis catatonia rating scale (BFCRS) (Bush et al., 1996) and the Northoff catatonia scale (NCS) (Northoff et al., 1998b). These scales use a rather strict definition of catatonia by relying on a cluster of symptoms, as recommended by Gelenberg (1976, 1977). Catatonic symptoms had to be manifest on the day of admission, in the presence of both examiners (G.N., P.D.). Furthermore, patients had to show complete akinesia (no voluntary movements at all) and concomitant posturing (positioning of limbs against gravity) for at least 30 min in the presence of the examiners (see Table 1 for number and scores of catatonic symptoms), so that catatonic patients with either hyperkinesias (n=3) or no concomitant akinesia and posturing (n=4) were excluded [because pathophysiological mechanisms may differ between hypo- and hyperkinetic catatonia (Northoff et al., 1995a, 1998a,b)]. All patients had to be classified as akinetic catatonic according to all four criteria lists, with agreement on every symptom by two independent psychiatrists (G.N., P.D.) who rated the same patients successively within 1 h on day 0 (before

initial medication with lorazepam), day 1 (24 h after admission) and day 14.

On admission, all catatonic patients received only a single dose of intravenous lorazepam (2 mg). According to the clinical response to lorazepam in the first 24 h, judged by the abovementioned criteria (Rosebush, Lohr, BFCRS, NCS), we distinguished between short-term responders [n=13; non-catatonic on day 1 (24 h after admission) according to Rosebush, Lohr, BFCRS, and NCS] and non-responders [n=3; still catatonic on day 1 (24 h after admission) according to Rosebush, Lohr, BFCRS, and NCS], from which only the former but not the latter were included in the study, since responders and non-responders might show distinct underlying pathophysiological mechanisms (Northoff et al., 1995a, 1998a,b). The present study therefore uses subsampling of the 'total' catatonic population, selecting only those with akinesia, response, and response to lorazepam (n=13), whereas catatonic patients with hyperkinesias (n=3), no concomitant akinesia and posturing (n=4), and non-response to lorazepam (n=3) were excluded.

After the full resolution of catatonic syndrome on day 1, lorazepam was withdrawn completely and patients received either antidepressants (n= 10) and/or neuroleptics (n=10) in the next 2 weeks until neuropsychological investigation which took place in the second week after admission.

2.1.2. Control groups

We investigated two control groups: psychiatric and healthy controls. The age- and sex-matched psychiatric control group [age: 46.5±9.2 years (mean±SD); all right-handed] included patients with similar diagnosis according to DSM-IV (American Psychiatric Association, 1994), similar duration of illness and similar medication as catatonic patients (see Table 2 for details). These patients were diagnosed according to DSM-IV by an independent psychiatrist with a semistructured interview, and underwent similar psychopathological assessment as catatonic patients (see above). Subsequently, age, sex, diagnosis, illness duration, and medication were matched between the catatonic group and the psychiatric control group, so that the only difference between the two psychiatric

Table 2

Demographic and clinical data [means (SD)] in catatonic and psychiatric control patients

Demographic and clinical variables	Catatonics ($n=13$)		Psychiatric controls ($n=13$)	
Age	43.4	(13.1)	46.5	(9.2)
Years of education	9.8	(1.4)	10.2	(1.8)
Duration of illness (years)	8.5	(5.7)	7.5	(5.0)
No. of hospitalizations	3.5	(2.3)	3.6	(2.0)
Age at onset	35.0	(12.0)	39.0	(8.9)
Time since actual onset (weeks)	4.6	(1.9)	5.7	(1.3)
Duration of treatment (years)	5.7	(4.5)	5.7	(3.5)
Neuroleptics (CPZ) (mg)	170.4	(167.5)	155.0	(136.1)
Anticholinergics (No. of treated patients)	10		10	
Global assessment scale (GAS)	14.5	(3.3)	20.2	(2.5)
Positive and negative symptom scale (PANSS)	86.3	(29.9)	80.2	(30.1)
Hamilton				
anxiety scale (HAM-A)	20.2	(4.4)	19.4	(2.6)
Depression scale (HAM-D)	14.9	(6.1)	19.4	3.8
No. of catatonic episodes	3.1	(1.8)	—	
Days of catatonic symptoms	15.3	(7.6)	—	
Rosebush scale (average No. of symptoms)				
Day 0	9.8	(1.8)	—	
Day 1	2.0	(1.2)	—	
Bush–Francis catatonia rating scale				
Day 0	26.2	(6.9)	—	
Day 1	2.8	(1.1)	—	
Northoff catatonia scale (NCS) (Day 0)				
NCS_{MOT} (motor)	20.3	(3.4)	—	
NCS_{AFF} (affective)	22.7	(2.8)	—	
NCS_{BEHAV} (behavioral)	19.5	(8.8)	—	
NCS_{TOT} (total)	62.5	(12.5)	—	

groups was the presence or absence of catatonic syndrome. Psychiatric patients with hypo-($SEPS > 3$) and hyperkinetic ($AIMS > 2$) neuroleptic-induced side effects, with catatonic symptoms/episodes on previous hospitalizations, electroconvulsive therapy, unconscious states, with alcohol/substance abuse, benzodiazepine medication in the last 6 months, and/or neurological/physical illness, were excluded from the study. Initially they all received a single injection of lorazepam in the same doses as catatonic patients (see above), were treated with similar medications, and were neuropsychologically investigated also in the third week after admission.

The healthy control group [age: 50.4 ± 15.3 years (mean $\pm$ SD); all right-handed] included 13 healthy subjects matched for age and sex to the catatonic group. Subjects with a history of psychiatric, neurological, or other serious physical illness, drug or alcohol abuse, and those with first-degree relatives with a history of major psychiatric or neurological disorders, were excluded.

2.2. Neuropsychological assessment

Standardized, commonly used tests were selected to generate a battery that would assess a number of neuropsychological abilities which are presumed to depend on frontal (executive functions, attention, working memory) and parietal (visual–spatial abilities) cortical function.

2.2.1. General intellectual functioning

2.2.1.1. Standard progressive matrices (SPM) (Raven, 1976). This was administered using the standard procedure. The test consists of 30 items of collections of figures, with one missing figure,

respectively. This missing figure has to be completed according to the logically correct solution by selection from various alternatives. The number of correctly completed figures reflects the verbally independent age-specific IQ.

2.2.1.2. Multiple vocabulary test-B (MWT-B) (Lehrl, 1995). The MWT-B consists of 37 items, each showing different words from which only one is meaningful (i.e., the other words are nonsense). This one meaningful word has to be recognized, and from the sum of recognized words an age-corrected norm value (and the respective percentile) can be calculated. These values represent the age-specific verbal IQ, as well as the assessment of general premorbid intelligence, showing high correlations with more complex tests of intelligence such as the Wechsler adult intelligence scale (which, due to reasons of time and stress, was not applied here).

2.2.2. Attention

2.2.2.1. d2 attention test (Brickenkamp, 1994). One sheet of paper with 14 rows, each containing 47 signs among which particular signs (i.e., $>d<$) should be specifically marked and selected within 20 s for each row, measuring 'attention to details'. Measures include the percentage of mistakes (F%), concentration performance (CP) as the number of correct selected signs minus the number of mistakes, and total performance (TP) as the mistake-corrected total number of all signs which were included in performance.

2.2.2.2. Colour–Word Interference Test (CWI) (Bäumler, 1985). First, a table with black-written colour names is presented, which should be read as fast as possible. Time (i.e., time for reading) and performance (correct words) are measured and transformed into general age- and education-specific norm values (i.e., FWL-T), reflecting attention and the speed of general information processing.

Second, a table with short lines in different colours is presented, from which the subject should name the respective colour. A general age- and education-corrected score (i.e. NOM-T) can be calculated, reflecting the ability of nomination.

Third, a table with coloured words for colours (green, red, etc.) is presented, where the colour indicated in the meaning, which has to be named, is not identical with the colour presented. A general age- and education-corrected score (i.e., SEL-T) can be calculated, measuring the ability of attention and selection (i.e., the interference).

2.2.3. Visual–spatial abilities

2.2.3.1. Observation from the Wilde test (BO-WIT) (Jäger and Althoff, 1994). The observation subtest is part of the Wilde intelligence test and consists of 42 items, each showing three faces of which two are identical and one altered in a small detail. The number of correctly recognized altered faces within a given time limit can be calculated as an age-specific value measuring 'visuo-perceptual speed'.

2.2.3.2. Visual object and space perception test (VOSP) (Warrington and James, 1991). Various cards with particular objects (i.e., bizarre figures as shades of objects) (i.e., VOSPobject) or silhouettes (VOSPsilh) are presented, from which a correct one has to be identified. The number of correctly identified solutions measures visual–spatial abilities specifically associated with right parietal function, in contrast to left parietal function (Warrington and James, 1991).

2.2.3.3. Subtest 7 and 9 from the performance investigation system (LPS) (Horn, 1983). The LPS is a battery for the measurement of various dimensions of the intellectual function. Subtest 7 consists of different signs which are rotated, but only one, the one to be recognized, is a reflected image. It measures the ability of mental rotation. Subtest 9 consists of three-dimensional figures for which the correct number of sides should be determined, reflecting visual–spatial abilities.

2.2.4. Executive functions

2.2.4.1. Trail making test (TMT) (Oswald and Roth, 1987). We applied a German version of the trail making test, the Zahlenverbindingstest, where

the numbers from 1 to 90 are presented in a diffuse order. The task is to connect the numbers in the correct order as quickly as possible, by drawing a connecting line with a pencil. The time needed can be calculated as an age-specific norm value measuring general information-processing speed.

2.2.4.2. Verbal fluency (Benton et al., 1983). According to the procedures by Benton et al. (1983), the subject was required to say as many words as possible beginning with a given letter (excluding numbers and proper nouns). For this study, the letters A, F, and S, as well as a semantic category (i.e., animals), were used. The F-, A-, S-, and semantic subscore, as well as the total scores (sum of all subscores), was corrected for age, sex, and education according to the existing norms, reflecting the ability of verbal fluency.

2.2.4.3. Two-group colour test according to Kramer (Goldstein and Scheerer, 1941). Eight cards with different motives must be classified into two groups of four cards, according to their respective concepts or categories (colour, letter, etc.). The total number of points is considered as the performance score, reflecting the ability of mental shifting and categorization.

2.2.4.4. 5-point test (Regard, 1991). Within 35 rectangular fields each containing 5 points, as many figures as possible between the points shall be drawn. Scores include the absolute number of figures as total points and the perseveration index as the number of perseverations in relation to total number of figures.

2.2.4.5. Box-piling test (Shallice, 1982). The box-piling test is a German version of the Tower of London test, measuring executive functions, planning capabilities and practical abilities. Following certain rules, the subject works out the least number of transports of single, numbered boxes which are necessary in order to get the order of boxes from an initial to a goal position. This is performed 10 times with different orders of boxes in the initial and goal position, respectively. The number of correct solutions and a total score (total number of points minus the deviations from the

correct solutions) are calculated as performance scores.

2.2.5. Working memory

2.2.5.1. Progressive arithmetics (Claros-Salinas and von Cramon, 1987). In the first calculation task, subjects had to subtract 9 from 100 continuously, each time adding 3 ($100 - 9 + 3$; $94 - 9 + 3$; etc.); they should name the respective provisional results. In the second calculation, subjects had to subtract 7 from 100 continuously ($100 - 7$, $93 - 7$, etc). Both procedures were repeated once. Mean time and mean errors were calculated as performance scores. Such calculations require extensive involvement of working memory abilities, because rules and results have to be memorized permanently during performance of progressive calculations.

2.3. Procedure

The tests were administered in two sessions on two successive days within 1 week, by a trained psychologist (D.N.) who was blind to the diagnosis. Patients were selected independently by G.N. Quality control procedures included double scoring of all test data and periodic review regarding reliability of test administration and scoring. Avoiding early exhaustion and/or monotony and providing alternations between tests with and without time limits, the tests were administered in the following order. Day 1: MWT-B, TMT, LPS-7, LPS-9, verbal fluency, SPM, d2, box piling; Day 2: CWI, Kramer, BO-WIT, path crosses, arithmetics, VOSP, 5-point test.

2.4. Statistical analysis

The two patient groups were compared on demographic data (age, age at onset, duration of illness, number of hospitalizations, neuroleptics) and psychopathological scores (GAS, PANSS, HAM-A, HAM-D) using two-tailed independent *t*-tests.

Neuropsychological test differences between the three groups were tested using analysis of variance

with repeated measures and post-hoc *t*-tests with Bonferroni correction for multiple comparisons.

Correlations between demographic, psychopathological and neuropsychological variables were calculated using Pearson product moment correlation analysis. Interrelationships between a reduced number (see below) of neuropsychological measures were calculated using bivariate Pearson product moment correlation. In addition, we performed partial correlations to control for effects of age, illness duration, and neuroleptic medication on those tests for which correlations were significant. Partial correlations were considered as significant at $p < 0.05$ (two-tailed). Only those relationships which correlated significantly in both kinds of correlation analyses were considered as neuropsychologically relevant and are mentioned in Section 3.

3. Results

3.1. Clinical and demographic data

Demographic data showed no significant differences between catatonics and psychiatric controls. Age, age at onset, duration of illness and treatment, number of psychiatric hospitalizations, and neuroleptic dosage (in chlorpromazine equivalents) did not differ significantly between catatonic and non-catatonic psychiatric control patients (see Table 2).

Psychopathological scores of HAM-A, HAM-D, and PANSS did not differ significantly between both groups (see Table 2). Only in the GAS did catatonics show significantly lower ($p < 0.000$) scores than psychiatric controls (see Table 2), indicating a poorer global state in catatonia (which is probably due to catatonic symptoms such as akinesia and mutism).

3.2. Neuropsychological measures

3.2.1. General intellectual function
No significant differences in tests of general intellectual function (SPM, MWT-B) were found between catatonics and psychiatric controls.

3.2.2. Attention
Catatonics did not differ significantly from psychiatric controls (see Table 3) either in the d2 test (CP, TP) or in the colour–word interference test (FWL-T, NOM-T, SEL-T). However, both psychiatric groups showed significantly lower scores in d2-CL, d2-TP, and CWI-FWL-T than healthy controls, whereas no significant differences between all three groups were found in the other subtests (NOM-T, SEL-T) of the colour–word interference test (see Table 3). In summary, catatonic patients showed no specific deficits in measures of attentional function.

3.2.3. Visual–spatial abilities
Catatonics did not differ significantly from psychiatric controls either in the observation subtest of the Wilde test (BO-WIT) or in subtests 7 and 9 of the performance investigation system. Both psychiatric groups showed significantly lower scores in both tests than healthy controls (see Table 3).

Catatonics showed significantly lower scores in VOSPobject than psychiatric ($p = 0.017$) and healthy controls, whereas no significant difference was found between psychiatric and healthy controls (see Table 3). No significant differences between all three groups were found in VOSPsilhouettes (see Table 3). In summary, catatonic patients show selective deficits in those visual–spatial abilities which are assumed to be closely related to right parietal cortical function (Warrington and James, 1991).

3.2.4. Executive function
Results of the trail making test (time), the two-group test, the verbal fluency, and box piling revealed no significant differences between catatonics and psychiatric controls, whereas in all these tests both groups showed significantly lower scores than healthy controls (see Table 3). The 5-point test revealed no significant differences between the three groups. In summary, catatonics showed no specific deficits in executive functions.

3.2.5. Working memory
Catatonics showed significantly higher scores (mean error) in one arithmetic task than psychiat-

Table 3
Neuropsychological performances in catatonics and psychiatric and healthy controls

Cognitive function	Neuropsychol. test	Catatonics		Psychiatric controls		Healthy controls		ANOVA			t-Test
		Mean	SD	Mean	SD	Mean	SD	df	F	p	
Attention	Test d2										
	TP	306.5	70.6	272.4	70.3	415.5	73.3	35	13.7	0.000	***[b], ***[c]
	CP	117.3	32.3	105.6	33.5	165.9	35.8	35	11.1	0.000	***[b], ***[c]
	CWI										
	FWL-T	47.5	6.9	44.8	6.1	53.8	6.4	37	6.4	0.004	**[b], ***[c]
	NOM-T	51.5	12.3	52.5	11.3	55.2	6.8	37	0.4	0.651	
	SEL-T	52.3	10.1	51.2	7.7	52.1	5.2	37	0.1	0.931	
Visual-spatial abilities	WIT: BO (RS)	17.9	7.9	17.6	6.0	27.6	7.1	36	8.3	0.001	***[b], ***[c]
	VOSP										
	Silhouettes	18.7	5.1	21.3	3.4	21.8	4.4	37	1.9	0.166	
	Objects	14.2	3.8	17.0	2.0	17.1	1.8	37	4.6	0.017	**[a], **[b]
	LPS-7	13.7	3.1	13.9	4.7	19.5	6.6	37	5.4	0.009	**[b], **[c]
	LPS-9	18.4	7.4	18.0	7.4	25.2	6.7	37	4.1	0.025	**[b], **[c]
Executive function	Trail Making Test										
	Mean time	110.8	32.1	129.9	48.0	68.4	17.0	38	10.7	0.000	***[b], ***[c]
	Verbal Fluency										
	FAS points	34.5	14.7	36.4	12.2	53.6	15.6	38	7.2	0.002	***[b], ***[c]
	Animal points	12.2	3.2	11.2	3.5	15.1	2.5	38	5.4	0.009	**[b], ***[c]
	Total points	46.6	17.2	47.6	14.7	68.7	16.4	38	7.8	0.002	***[b], ***[c]
	5-Point Test										
	Total points	24.7	8.1	23.7	8.1	31.8	19.2	37	1.5	0.245	
	Persev. index	1.0	0.1	0.2	0.2	0.1	0.1	37	1.6	0.208	
	2-Group Test										
	Total points	2.9	1.3	3.3	1.4	4.4	1.1	37	4.7	0.015	***[b], **[c]
	Box Piling points	90.2	5.3	90.6	6.3	96.5	3.5	31	5.6	0.009	***[b], ***[c]
	Solutions	5.9	2.3	4.9	2.7	7.8	2.4	31	4.1	0.027	**[c]
Working memory	Progr. Arithm.										
	>100−9+3<										
	Mean time	212.4	76.0	148.1	40.5	71.9	18.7	23	22.9	0.000	***[b], ***[c]
	Mean error	2.0	1.6	2.3	1.8	1.3	1.0	23	1.2	0.308	
	>100−7<										
	Mean time	113.7	51.6	76.2	24.9	36.5	14.6	23	14.8	0.000	***[b], ***[c]
	Mean error	1.6	1.1	0.3	0.5	0.4	0.6	23	5.3	0.014	**[a], **[b]

**$p \leq 0.05$; ***$p \leq 0.01$.
[a]Significant group differences between catatonics and psychiatric controls.
[b]Significant group differences between catatonics and healthy controls.
[c]Significant group differences between psychiatric and healthy controls.

ric and healthy controls (see Table 3). Though not reaching a level of statistical significance (probably due to high standard deviations), catatonics nevertheless showed higher scores in mean time than psychiatric and healthy controls (see Table 3). Both catatonics and psychiatric controls differed significantly from healthy controls (see Table 3).

In summary, catatonic patients showed poor performance in progressive arithmetics strongly involving abilities of working memory.

3.2.6. Comparison of nosological groups

Psychiatric groups were first classified syndromatically (i.e., according to the presence of catatonic syndrome) into catatonic and non-catatonic patients, independent of underlying psychiatric

disease (though the latter was matched between catatonic and non-catatonic patients). However, psychiatric patients may also be classified according to their underlying psychiatric disease as either schizophrenic or affective psychosis. In a second step, we therefore compared affective and schizophrenic patients within the catatonic (9 affective and 4 schizophrenic catatonics) and the total psychiatric (catatonics and psychiatric controls with 18 affective and 8 schizophrenic patients) sample. Analysis revealed no significant differences between affective and schizophrenic patients in both samples. Even those measures (VOSPobject), which differed significantly between catatonics and psychiatric controls, revealed no significant differences between both nosological groups (i.e., schizophrenic versus affective psychosis).

3.3. Correlations

3.3.1. Intercorrelations between neuropsychological measures

To examine relationships between neuropsychological functions, bivariate Pearson product moment correlations were calculated separately for each group (see Table 4). To reduce the number of variables in the analysis, only the tests which showed marked differences between catatonics and psychiatric controls (VOSPobject, Progressive arithmetics), were included into correlation analysis (with all other neuropsychological measures).

Catatonics showed significant positive correlations between VOSPobject and d2-CP and d2-TP which were present in neither psychiatric nor healthy controls (see Table 4). Furthermore, VOSPobject significantly correlated with CWI-FWL-T and CWI-NOM-T, as well as with mistakes in the calculation task in catatonia, but in neither psychiatric nor healthy controls. There were significant correlations between VOSPobject and the two-group test in psychiatric and healthy controls, but not in catatonics.

Measures in calculation tasks significantly correlated with performance in box piling in catatonics and healthy controls, but not in psychiatric controls (see Table 4). Catatonics showed significant

correlations between calculation tasks and attentional measures (d2, CWI-FWL-T) (see Table 4).

In summary, catatonic patients showed significant correlations between visuo-spatial and attentional measures which were present in neither psychiatric nor healthy controls.

3.3.2. Correlations between neuropsychological measures and clinico-demographic data

Neither catatonics nor psychiatric controls showed any significant correlations between neuroleptic dosage (in chlorpromazine equivalents), neuropsychological measures, and psychopathology. Only mistakes in calculation correlated significantly with duration of illness ($r = 0.932$; $p = 0.002$) and age at onset ($r = 0.949$; $p = 0.014$) in catatonic but not in psychiatric control patients. In summary, neuropsychological measures in catatonic and psychiatric control patients showed no relationships with neuroleptic treatment and other clinical and demographic variables (except the relationship between illness duration and arithmetic abilities in catatonia).

3.3.3. Correlations between psychopathological and neuropsychological measures

Significant correlations ($r = -0.668$; $p = 0.028$) between catatonic motor symptoms (i.e., NCS_{MOT}) and d2-TP were found in catatonics. Furthermore catatonic symptoms (NCS_{MOT}: $r = 0.673$, $p = 0.012$; NCS_{BEHAV}: $r = 0.603$, $p = 0.029$; NCS_{TOT}: $r = 0.668$, $p = 0.012$) significantly correlated with HAM-A. HAM-D significantly correlated with calculation time ($100 - 7$ time) in catatonics ($r = -0.889$; $p = 0.044$) and HAM-A with calculation errors ($100 - 9 + 3$) in psychiatric controls ($r = -0.852$; $p = 0.031$). In summary, catatonic symptoms showed a relationship with attentional and affective measures.

4. Discussion

The main findings in the present neuropychological study on catatonia are the following: (i) selective deficits in visual object perception associated with right parietal function; (ii) significant correlations between visuo-spatial and attentional mea-

Table 4
Significant intercorrelations between neuropsychological measures [$r(p)$] in catatonics and psychiatric and healthy controls

Cognitive function	Test	Group	Visual–spatial VOSP Objects	Progressive arithmetics >100−9+3< Time mean	>100−9+3< Error mean	>100−7 Time mean	>100−7 Error mean
	d2-TP	Catatonic	0.792 (0.004)	−0.983 (0.003)			
		Psychiatric controls					
		Healthy controls	0.578 (0.039)				
	d2-CP	Catatonic	0.887 (0.000)				
		Psychiatric controls					
Attention		Healthy controls					
	CWI: FWL-T	Catatonic	0.628 (0.022)			−0.942 (0.017)	
		Psychiatric controls					
		Healthy controls					
	NOM-T	Catatonic	−0.536 (0.059)				
		Psychiatric controls					
		Healthy controls					
	SEL-T	Catatonic					
		Psychiatric controls					
		Healthy controls					
Visual–Spatial abilities	WIT-BO	Catatonic					
		Psychiatric controls			−0.907 (0.013)		
		Healthy controls					
	VOSPsilhou	Catatonic	0.656 (0.051)				
		Psychiatric controls	0.672 (0.017)				
		Healthy controls					
	VOSPobject	Catatonic					−0.995 (0.011)
		Psychiatric controls					
		Healthy controls					
	LPS-7	Catatonic					
		Psychiatric controls					
		Healthy controls					
	LPS-9	Catatonic			−0.934 (0.018)		
		Psychiatric controls					−0.852 (0.031)
		Healthy controls	0.573 (0.041)				
	Trail Making	Catatonic					
		Psychiatric controls					
		Healthy controls		0.570 (0.042)	0.565 (0.044)	0.058 (0.034)	
	Verb. F1: FAS	Catatonic					
		Psychiatric controls			0.849 (0.033)		
		Healthy controls	0.557 (0.048)				
	Animals	Catatonic					
		Psychiatric controls					
		Healthy controls					
	Total points	Catatonic					
		Psychiatric controls			0.887 (0.019)		
		Healthy controls					

Table 4 (*continued*)
Significant intercorrelations between neuropsychological measures [$r(p)$] in catatonics and psychiatric and healthy controls

Cognitive function	Test	Group	Visual–spatial VOSP Objects	Progressive arithmetics $>100-9+3<$ Time mean	Error mean	$>100-7$ Time mean	Error mean
Executive function	5-Point Test	Catatonic					
		Psychiatric controls		0.821 (0.045)			
		Healthy controls					
	Pers. Index	Catatonic					
		Psychiatric controls				−0.931 (0.007)	
		Healthy controls					
	2-Group Test	Catatonic					
		Psychiatric controls	0.681 (0.015)			0.875 (0.023)	
		Healthy controls	0.577 (0.039)				
	Box Piling	Catatonic			−0.998 (0.045)	0.999 (0.025)	
		Psychiatric controls					
		Healthy controls		−0.591 (0.033)	−0.559 (0.047)	−0.599 (0.030)	−0.731 (0.005)
	Solutions	Catatonic					
		Psychiatric controls					
		Healthy controls					−0.760 (0.003)

sures; (iii) significant correlations between catatonic symptoms and attentional/affective measures; (iv) significant differences between catatonics and healthy controls in almost all attentional and executive tests associated with frontal function.

Although many neuropsychological studies have been conducted on schizophrenia and depression (see Zalewski et al., 1998 for an overview), no neuropsychological investigations on catatonia have been reported as yet. Instead of comparison with other studies, the discussion will thus focus on the importance of visual–spatial and attentional alterations in combination with fronto-parietal dysfunction in catatonia.

4.1. Catatonia, visual–spatial abilities, and right parietal cortical function

Catatonic patients showed significantly poorer performance in object perception in VOSP than psychiatric and healthy controls. The specificity of this finding for catatonia is further underlined by the following results: (i) no significant difference in VOSPobject between psychiatric and healthy controls (see Section 3.2.3); (ii) nosological com-

parisons between affective and schizophrenic patients revealed no significant differences, either in VOSPobject or in other visual–spatial measures (see Section 3.2.6); (iii) significant correlations between VOSPobject and attentional measures only in catatonic patients, but in neither psychiatric nor healthy controls (see Section 3.3.1).

The pathophysiological importance of such a specific visual–spatial deficit in catatonia remains unclear. The VOSP was developed for the differentiation between right and left parietal function, and is particularly sensitive to right posterior parietal cortical lesions (Warrington and James, 1991). Visual–spatial deficits in catatonia may therefore be related to right parietal cortical dysfunction. Such an assumption would be supported by findings of decreased right parietal r-CBF (Satoh et al., 1993; Galynker et al., 1997; Northoff et al., 1998d), as well as by occurrence of posturing in patients with isolated right parietal lesions (Saver et al., 1993; Fukutake et al., 1993).

In addition to visual–spatial deficits, catatonics showed significant correlations between VOSPobject, attentional tests (d2, CWI), and working memory tasks (progressive arithmetics),

measures which were present in neither psychiatric nor healthy controls (see Section 3.3.1). These results suggest a specific relationship of visual–spatial deficits with attentional and working memory abilities; i.e., between right parietal and frontal cortical function in catatonia which would be further supported by the following observations: (i) significant correlations between VOSPobject and the two-group test (as a measure of frontal function) in psychiatric and healthy controls, but not in catatonia (see Table 4); (ii) poorer arithmetic performances in catatonics compared to psychiatric and healthy controls, indicating specific alterations in working memory and frontal cortical function; (iii) significantly poorer performance in almost all executive and attentional measures in catatonia than in healthy controls, indicating a general disturbance in frontal cortical function. The functional importance of fronto-parietal dysfunction is further underlined by findings of significantly reduced r-CBF in right fronto-parietal cortex in catatonic patients compared to non-catatonic psychiatric and healthy controls (Satoh et al., 1993; Galynker et al., 1997; Northoff et al., 1998d). Right fronto-parietal dysfunction may be closely related to attentional–motor disturbances as observed in the present investigation. Studies in healthy controls performing a task specifically requiring attention to movements induced right fronto-parietal cortical activation (Deiber et al., 1996; Gitelmann et al., 1996; Jueptner et al., 1997). Concomitant dysfunction in right parietal and frontal cortex may thus account for the present findings of significant correlations between attentional measures (d2, CWI), motor symptoms and visual–spatial abilities in catatonia. In addition, significant correlations between catatonic symptoms and attentional/affective measures could well be related to peculiarities in subjective experiences. Catatonic patients often experience intense and uncontrollable anxieties whereas, unlike parkinsonian patients, they remain almost entirely unaware of their movement disturbances such as akinesia and posturing (Northoff et al., 1998a). Such a dissociation between emotional and movement awareness could be related to a shift in attention, focusing exclusively on emotions but no longer on movements, which may be reflected in unawareness

of movements and right fronto-parietal dysfunction. However, specific attentional–motor activation studies in PET/fMRI would be necessary to further support our assumption of attentional–motor and right fronto-parietal dysfunction in catatonia.

4.2. Methodological limitations

First, we were unable to account entirely for the influence of neuroleptics on neuropsychological measures, since healthy controls were unmedicated. However, differences between catatonics and psychiatric controls cannot be due to medication, because both groups were treated similarly (see Methods) and, in addition, no significant correlations were found between neuroleptics and neuropsychological measures (see Section 3.3.2). Other confounding variables, such as age, illness duration, general intellectual functioning, etc. (see Methods and Results) did not differ significantly between groups, supporting our belief that our findings cannot be interpreted as resulting from lower premorbid cognitive ability or educational attainment, thus representing genuine disease-related deficits.

Second, our findings could be related rather to the underlying disease (either schizophrenic or affective psychosis) than with catatonic syndrome itself. However, such an assumption would not be supported by our present findings. Nosological classification of catatonic and psychiatric control patients into two groups according to the underlying disease (i.e., either affective or schizophrenic psychosis) did not reveal significant differences in any of the neuropsychological measures (see Section 3.2.6). Differences between catatonics and psychiatric controls may thus be regarded as specific for catatonic syndrome itself, rather than for the underlying psychiatric disease.

Third, we did not investigate psychomotor function directly; instead, we indirectly measured such abilities in various neuropsychological tasks requiring psychomotor speed. Catatonic patients showed no major deficits in tasks requiring psychomotor speed compared to psychiatric and healthy controls (see also Section 3.2, Section 4.1).

Fourth, only working memory was investigated,

whereas other forms and processes of memory were not considered (see for example, Nathaniel-James et al., 1996; Rossell and David, 1997). Thus we cannot decide whether catatonic patients suffer from general alterations in memory or rather from specific working-memory deficits.

Fifth, the sample size of 13 catatonic patients in the present study is very small. However, considering that catatonia is quite rare (incidence in relation to all admitted inpatients of 2.6%), as well as the careful definition and selection of the present sample according to established criteria (see Section 2), the small sample size may, at least partially, be justified. Nevertheless, the present data should be considered as exploratory and preliminary; further investigations are required on larger samples.

Sixth, although intercorrelation analyses may reveal some functional relationships between neuropsychological measures involving different focal brain areas, specific 'functional connectivity', as, for example, between frontal and parietal cortex, cannot be investigated directly in neuro-psychological studies. Therefore, our assumption of fronto-parietal dysfunction in catatonia should be regarded as a provisional hypothesis awaiting further empirical support.

5. Conclusions

Catatonia is a psychomotor syndrome characterized by motor and behavioral anomalies which may be closely related to attentional–motor and fronto-parietal dysfunction. We therefore tested attentional abilities and other neuropsychological tasks associated with frontal and parietal cortical function (i.e., executive, visual–spatial, working memory). We investigated catatonic patients and compared them with non-catatonic psychiatric and healthy controls. Catatonic patients showed significantly poorer performances and different correlation patterns in visual–spatial abilities. In addition, we found significant correlations between catatonic motor symptoms, visuo-spatial abilities, and attentional measures. Though of preliminary nature given the small sample size, catatonics showed visual–spatial deficits which were closely related to attentional measures and catatonic symptoms suggesting attentional–motor and fronto-parietal dysfunction in catatonia.

Carter et al., 1998; Cohen et al., 1994; Cohen et al., 1997; Jahanshahi et al., 1995; Kohler et al., 1998; Leschinger et al., 1998; Liddle, 1994; Liddle and Morris, 1991; Norman et al., 1997; Pantelis et al., 1997; and Sturm et al., 1993 not cited in text.

References

American Psychiatric Association, 1994. Diagnostic Manual of Mental Disorders, 4rd ed. American Psychiatric Press, Washington, DC.

Bäumler, G., 1985. FWIT: Farbe-Wort-Interferenz Test. Hogrefe, Göttingen.

Benton, A.L., Hamsher, K., Varney, N.R., Spreen, O., 1983. Contributions to Neuropsychological Assessment. Oxford University Press, New York.

Brickenkamp, R., 1994. Test d2: Aufmerksamkeits-Belastungstest, 2nd ed. Hogrefe, Göttingen.

Bush, G., Fink, M., Petrides, G., Dowling, F., Francis, A., 1996. Catatonia. Rating scale and standardized examination. Acta Psychiatr. Scand. 93, 129–143.

Carter, C.S., Braver, T.S., Barch, D.M., Botvinick, M.M., Noll, D., Cohen, J.D., 1998. Anterior cingulate cortex, error detection, and the online monitoring of performance. Science 280, 747–750.

Claros Salinas, D., von Cramon, D., 1987. Diagnostik von Störungen im Umgang mit Zahlen (Akalkulie). Fortschr. Neurol. Psychiatr. 55, 239–248.

Cohen, J.D., Forman, S.D., Braver, T.S., Casey, B.J., Servan-Schreiber, D., Noll, D.C., 1994. Activation of the prefrontal cortex in a nonspatial working memory task with functional MRI. Hum. Brain Mapp. 1, 293–304.

Cohen, J.D., Perlstein, W.M., Braver, T., Nystrom, L.E., Noll, D.C., Jonides, J., Smith, E.E., 1997. Temporal dynamics of brain activation during a working memory task. Nature 386, 604–608.

Deiber, M.P., Ibanez, V., Sadato, N., Hallett, M., 1996. Cerebral structures in motor preparation in humans: a PET study. J. Neurophysiol. 75, 233–243.

Endicott, J., Spitzer, R., Flies, J. et al., 1976. The Global Assessment Scale. Arch. Gen. Psychiatry 33, 766–771.

Fink, M., Bush, G., Francis, A., 1993. Catatonia: a treatable disorder occasionally recognized. Direct. Psychiatry 13, 1–7.

Fukutake, T., Hirayama, K., Komatsu, T., 1993. Transient unilateral catalepsy and right parietal damage. Jpn. J. Psychiatry 47, 647–650.

Galynker, I., Weiss, J., Ongseng, F., Finestone, H., 1997. ECT treatment and cerebral perfusion in catatonia. J. Nucl. Med. 38, 251–254.

Gelenberg, A.J., 1977. Criteria for the diagnosis of catatonia. Am. J. Psychiatry 134, 462–463.

Gelenberg, A.J., 1976. The catatonic syndrome. Lancet 2, 1339–1341.

Gitelmann, D., Alpert, N., Kosslyn, S., Daffner, K., Scinto, L., Thompson, W., Mesulam, M., 1996. Functional imaging of human right hemispheric activation for exploratory movements. Ann. Neurol. 39, 174–179.

Goldstein, K., Scheerer, M., 1941. Abstract abd concrete behavior: an experimental study with special tests. Psychol. Monogr. 53, 2

Greenblatt, D., Franke, K., Shader, R., 1978. Analysis of lorazepam and its glucuronide metabolite by electron-capture gas–liquid chromatography. J. Chromatogr. 146, 311–320.

Guy, W., 1976. Assessment Manual for Psychopharmacology. NIH, Washington, DC.

Hamilton, M., 1959. The assessment of anxiety states by rating. Br. J. Med. Psychol. 32, 50–55.

Hamilton, M., 1960. A rating scale for depression. J. Neurol. Neurosurg. Psychiatry 23, 56–62.

Horn, W., 1983. LPS: Leistungsprüfsystem, 2nd ed. Hogrefe, Göttingen.

Jäger, A.O., Althoff, K., 1994. WIT: Der Wilde Intelligenz Test. Hogrefe, Göttingen.

Jahanshahi, M., Jenkins, H., Brown, R.G., Marsden, C.D., 1995. Self-initiated versus externally triggered movements. Brain 118, 913–933.

Jueptner, M., Stephan, K.M., Frith, C.D., Brooks, D., Frackowiak, R., Passingham, R.E., 1997. Anatomy of motor learning. Frontal cortex and attention to action. J. Neurophysiol. 77, 1313–1324.

Kahlbaum, K., 1874. Die Katatonie oder das Spannungsirresein. Eine Form psychischer Krankheit. Hirschwald, Berlin.

Kay, S.R., Opler, L.A., Fiszbein, A., 1987. Positive and Negative Syndrome Scale (PANSS) Rating Manual. Social and Behavioral Sciences Documents, San Rafael, CA.

Kohler, C., Gur, R.C., Swanson, C.L., Petty, R., Gur, R.E., 1998. Depression in schizophrenia: I. Association with neuropsychological deficits. Biol. Psychiatry 43, 165–172.

Lehrl, S., 1995. MWT-B. 3rd ed. Hogrefe, Göttingen.

Leschinger, A., Baumgart, F., Gaschler, B., Northoff, G., Scheich, H., Bogerts, B., 1998. Effect of training and experience on task performance and prefrontal cortical activation during an auditory working memory task. Hum. Brain Mapp., in press.

Liddle, P.F., Morris, D.L., 1991. Schizophrenic syndromes and frontal lobe performance. Br. J. Psychiatry 158, 340–345.

Liddle, P.F., 1994. Volition and schizophrenia. In: David, A., Cutting, J. (Ed.), The Neuropsychology of Schizophrenia. Erlbaum, Hillsdale, pp. 39–49.

Lohr, J.B., Wisniewski, A.A., 1987. Movement Disorders: A Neuropsychiatric Approach. Guilford Press, New York.

Nathaniel-James, D.A., Brown, R., Ron, M.A., 1996. Memory impairment in schizophrenia: its relationship to executive function. Schizophr. Res. 21, 85–96.

Norman, R.M., Malla, A.K., Morrison-Stewart, S.L., Helmes, E., Williamson, P.C., Thomas, J., Cortese, L., 1997. Neuropsychological correlates of syndromes in schizophrenia. Br. J. Psychiatry 170, 134–139.

Northoff, G., Wenke, J., Demisch, L., Pflug, B., 1995a. Catatonia: short-term response to lorazepam and dopaminergic metabolism. Psychopharmacology 122, 82–186.

Northoff, G., Wenke, J., Krill, W., Pflug, B., 1995b. Ball-experiments in 32 acute akinetic catatonic patients: deficits of internal initiation and generation of movements. Movement Dis. 10, 589–595.

Northoff, G., 1997. Katatonie. Einführung in die Phänomenologie, Klinik und Pathophysiologie eines psychomotorischen Syndroms. Enke, Stuttgart.

Northoff, G., Krill, W., Eckert, J., Russ, M., Pflug, B., 1998a. Major differences in subjective experience of akinetic states in catatonic and parkinsonic patients. Cogn. Neuropsychiatry 3 (3), 161–178.

Northoff, G., Koch, A., Wenke, J., Eckert, J., Pflug, B., Bogerts, B., 1998b. Catatonia as a psychomotor disease: a rating scale, subtypes and extrapyramidal motor symptoms. Movement Dis., in press.

Northoff, G., Braus, D., Sartorius, A., Khoram-Sefat, D., Russ, M., Eckert, J., Herrig, M., Leschinger, A., Bogerts, B., Henn, F.A., 1998c. Reduced activation and altered laterality in two neuroleptic-naive catatonic patients during a motor task in functional MRI. Psychol. Med., in press.

Northoff, G., Steinke, R., Czerwenka, C., Danos, P., Kropf, S., Otto, H.J., Bogerts, B., 1998d. Right fronto-parietal dysfunction in akinetic catatonia: investigation of regional cerebral blood flow. Submitted.

Oldfield, R.C., 1971. The assessment and analysis of handedness: the Edinburgh Inventory. Neuropsychologia 9, 97–113.

Oswald, W.D., Roth, E., 1987. ZVT: Zahlenverbindungstest. Hogrefe, Göttingen.

Pantelis, C., Barnes, T., Nelson, H.E., Tanner, S., Weatherly, L., Owen, A., Robbins, T., 1997. Fronto-striatal cognitive deficits in patients with schizophrenia. Brain 120, 1823–1843.

Raven, J., 1976. Manual for Raven's Progressive Matrices. H.K. Lewis, London.

Regard, M., 1991. The perception and control of emotions: hemispheric differences and the role of the frontal lobes. PhD thesis, University of Zürich.

Rosebush, P., Furlong, B., Mazurek, M., 1990. Catatonic syndrome in a general psychiatric inpatient population: frequency, clinical presentation an response to lorazepam. J. Clin. Psychiatry 51, 357–362.

Rossell, S.L., David, A.S., 1997. The neuropsychology of schizophrenia: recent trends. Curr. Opin. Psychiatry 10, 26–29.

Satoh, K., Suzuki, T., Narita, M., Kato, T., Ohnishis, H., Morita, R., 1993. Regional cerebral blood flow in catatonic schizophrenia. Psychiatry Res.: Neuroimaging 50, 203–216.

Saver, J.L., Greenstein, P., Ronthal, M., Mesulam, M., 1993. Asymmetric catalepsy after right hemispheric stroke. Movement Dis. 8, 69–73.

Shallice, T., 1982. Specific impairments of planing. Phil. Trans. R. Soc. Lond. 298, 199–209.

Simpson, G.M., Angus, J.W.S., 1970. A rating scale for extrapyramidal side effects. Acta Psychiatr. Scand. 212, 11–19.

Sturm, W., Willmes, K., Horn, W., 1993. LPS: Leistungsprüfsystem. Hogrefe, Göttingen.

Taylor, M.A., 1990. Catatonia. A review of the behavioral neurologic syndrome. Neuropsychiatr. Neuropsychol. Behav. Neurol. 3, 48–72.

Warrington, EK., James, M., 1991. Visual Object and Space Perception Battery. Thamas Vallery Test Company, Suffolk.

Zalewski, C., Johnson-Selfridge, M.T., Ohriner, S., Zarrella, K., Seltzer, J.C., 1998. A review of neuropsychological differences between paranoid and nonparanoid schizophrenia patients. Schizophr. Bull. 24 (1), 127–145.