Eur Arch Psychiatr Neurol Sci (1986) 235:301–308

European Archives of Psychiatry and Neurological Sciences

Psychomotor Disturbances in Psychiatric Patients as a Possible Basis for New Attempts at Differential Diagnosis and Therapy

II. Cross Validation Study on Schizophrenic Patients: Persistance of a "Psychotic Motor Syndrome" as Possible Evidence of an Independent Biological Marker Syndrome for Schizophrenia*

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Summary. This study investigates the existence and course of psychomotor symptoms in schizophrenic patients ($n=57$, both treated and untreated with antipsychotic drugs) as compared to 25 healthy controls. Previous psychometric studies had suggested the existence of a "psychotic motor syndrome" (PMS) both in (untreated) schizophrenic and endogenous depressed patients, consisting of disturbances of lip and tongue movements, fine and gross movements of the dominant right hand and impaired complex motor coordination of the extremities. We confirmed the existence of the PMS in this study. There was no correlation of the PMS with the psychopathological status of the patients, or with extrapyramidal side-effects of the drugs used, perhaps indicating an independent "basic syndrome" ("Basisstörung"). Factorial analyses revealed similar structures both in schizophrenic and healthy persons; the differences in motor performance may be due to an impairment of the first factor "general motor ability" in schizophrenic patients. The PMS did not disappear parallel to the psychopathological improvement of the patients, nor in the symptom-free remission interval. The role of the PMS as possible independent biological marker syndrome for schizophrenia can consequently be further supported, with its implications towards the differential diagnostic and therapeutical values of this syndrome.

Key words: Motor activity – Motor skills – Psychotic motor syndrome – Psychomotor dysfunction – Neurophysiological disorder

Introduction

Motor deficiencies in schizophrenic persons are a well-documented fact (see e.g. Manschreck et al. 1981; Mather and Putchat 1984, for a review). They range from alterations in the partial-field optokinetic nystagmus (Latham et al. 1981) and slow pursuit eye movements (Iacono et al. 1981) to differences in musical tempo in the reproduction of a simple melody (Steinberg and Raith 1985) to a broad "psychotic motor syndrome" (PMS) (Guenther and Gruber 1983), consisting of disturbances of movements of the lips, the tongue, the dominant right hand and the complex coordination of the extremities. This PMS has however been found to exist both in schizophrenic (untreated by antipsychotic drugs) and (mono- or bipolar) endogenous depressed persons.

The sources of these psychomotor disturbances are not yet clearly understood either in schizophrenics, or in endogenous depressed persons, for whom less information seems to be available.

However, a similar syndrome existing both in schizophrenia and endogenous depression might be useful in providing information on the possible underlying central functional impairments, and merits further investigation.

We conducted two cross-validation studies on this PMS. On schizophrenic persons ($n=57$) we tried to reproduce the evidence of such a syndrome and to evaluate the relations to effects caused by antipsychotic drug treatment and the course of PMS in the course of the illness. In the second study we examined endogenous depressed persons ($n=57$)(mono- and bipolar) with regard to the same questions and the results will be reported in this journal later on.

Special attention was given to the problem of matching suitable control persons, resulting in two independent studies in schizophrenics and endogenous depressed patients (with independent control groups) to carefully establish drug-associated motor side-effects.

Method

Subjects were inpatients of the Psychiatric University Hospital links der Isar, Nussbaumstrasse, Munich ($n=41$) and 16 outpatients of a private practice, situated in the center of Munich[1].

Table 1 summarizes biographical and psychiatric data of the patients and the control group. Control persons were members of the staff of the above hospital, with no psychiatric

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* Supported in part by the Deutsche Forschungsgemeinschaft (DFG Gu 207/2-1).

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Table 1. Subjects. NAT = *n*ot *a*ctually *t*reated at the time of the admission; TCL = *t*reated with neuroleptics at admission and then by the *cl*inician; TST = *t*reated with neuroleptics at admission and then by *st*udy neuroleptic drugs; INT = patients in a symptomfree *int*erval, not hospitalised; CNT = *cont*rol persons. i = initial, f = final

$n = 82$		NAT $n = 16$	TCL $n = 13$	TST $n = 12$	INT $n = 16$	CNT $n = 25$
Mean age (years)		28.8	26.4	28.3	31.6	30.9
Range		17–49	18–58	19–50	25–57	20–46
Male/female		15/1	8/5	8/4	13/3	16/9
Duration of the illness (years)		6.6	7.8	7.5	8.1	–
Neuroleptic treatment (Chlorpromazine equivalents)	– i. examination	–	540	570	480	–
	– f. examination	580	570	540	–	–
Diagnoses (DSM-III, ICD-9)	295.1	5	4	5	5	
	295.3	5	5	4	6	
	295.6	4	3	3	4	
other	295.	2	1	0	1	

and/or medical history, not taking medication, drugs or alcohol regularly nor at the time of the investigations, and medical students complying with the same criteria ($n = 25$).

As can be seen from Table 1, there was a predominance of males in the patients, which had to be matched in the control group. Neither in our previous study of psychomotor symptoms in schizophrenic persons (Guenther and Gruber 1983) nor in other reports (e.g. Yates 1973; Manschreck et al. 1981) were differences of motor symptoms described with regard to sex. However, careful consideration will be given to the correlations of motor performance with the sex variable in the results section.

In the group "not actually treated" at least 1 week of "wash-out" was required, although the majority of patients ($n = 10$) had not received any antipsychotic medication prior to the examination (being first manifestations of the illness). Diagnosis was reached independently by the senior clinical psychiatrist and the authors after a modified interview of Endicott and Spitzer (1978), fulfilling both DSM-III (APA 1980), RDC (Spitzer et al. 1977) and ICD-9 (Degkwitz et al. 1980) criteria. Patients whose diagnosis was the same in both rater groups were briefed on the purpose of the study. If their informed consent was obtained, they participated in the study.

Criteria for selection of the patients were: 16–60 years of age, right-handed (assessed using the Edinburgh inventory; Oldfield 1971), no history of organic brain damage, EEG and (obtained in many cases) cranial computed tomography normal (as evaluated by the usual clinical procedures). All examinations were carried out in a special room, with light and noise surroundings as constant as possible, at the same time of the day (afternoon).

Psychomotor Variables

We used a motor test battery, consisting of the "Motorische Leistungsserie" (Schoppe 1974), the Lincoln-Oseretzky Motor Development Scale (Oseretzky 1931; Reinert 1966; modified by Guenther 1980) and the Motor Subtest of Luria-Nebraska Neuropsychological Battery (Luria 1970; Golden 1979), which have been described in detail previously (Guenther and Gruber 1983).

Each patient was examined twice with this battery on the day after admission and on the day before discharge (some rare exceptions from this rule being inevitable for clinical reasons) – except for the interval patients, who could only be examined once in the private practice.

Table 2. Average ratings on BPRS factor scores in the patient groups. NAT = not actually treated at the time of the admission; TCL = treated with neuroleptics at admission and then by the clinician; TST = treated with neuroleptics at admission and then by study neuroleptic drugs; INT = interval patients in a symptom-free interval, not hospitalised. ANDP = anxiety/depression, ANER = anergia, THOT = thought disturbance, ACTV = activation, HOST = hostile/suspiciousness

	NAT	TCL	TST	INT
Initial examination				
ANDP	12.5 ± 4.5	11.0 ± 5.5	14.9 ± 5.1	7.8 ± 3.1
ANER	12.9 ± 4.8	12.5 ± 3.9	11.1 ± 3.0	8.3 ± 4.4
THOT	15.1 ± 3.7	12.2 ± 4.4	14.8 ± 6.3	7.7 ± 4.8
ACTV	13.4 ± 4.9	13.4 ± 3.8	12.2 ± 4.6	6.8 ± 3.0
HOST	10.8 ± 4.6	10.2 ± 5.1	9.2 ± 4.0	5.8 ± 3.1
Final examination				
ANDP	8.3 ± 3.0	9.5 ± 4.5	7.0 ± 3.9	
ANER	9.5 ± 4.0	9.5 ± 4.8	6.5 ± 2.3	
THOT	9.1 ± 4.1	6.9 ± 2.5	6.2 ± 2.6	
ACTV	5.6 ± 2.1	7.1 ± 3.5	5.5 ± 2.1	
HOST	5.4 ± 1.7	5.2 ± 2.3	5.0 ± 2.5	

Psychopathology Variables

The Brief Psychiatric Rating Scale (BPRS) was carried out in order to screen the psychopathological status of the patients at the time of the initial and final motor examination; in the interval group only one BPRS rating along with the single motor examination was performed.

Table 2 shows the scores on the five factors within the BPRS (Overall and Gorham 1976). As can be seen, there were similar factorial scores in the initial examination in the inpatient groups, which showed parallel improvement towards the final examination before discharge. The interval group corresponded well to the values obtained in these final

Table 3. Significant differences of means obtained with the Motorische Leistungsserie in the initial and final examinations. Analyses of variance were calculated for each item, separately for the initial and final examination, and subsequent multiple pair comparisons by Duncan tests were performed. NAT = not actually treated at the time of the admission; TCL = treated with neuroleptics at admission and then by the clinician; TST = treated with neuroleptics at the time of admission and subsequently by study neuroleptic drug; INT = interval patients in a relatively symptom-free period, not hospitalised; CNT = control persons. F = faults, Fd = duration of faults, CR = correct responses, Td = total duration, P = part of the task. LS = long sticks, SS = short sticks, LF = line following, PR = pursuit rotor. i = initial, f = final examination, * = $P<5\%$, ** = $P<1\%$

		NAT/CNT		TCL/CNT		TST/CNT		INT/CNT
Aiming	F							
	Fd							
	CR							
	Td	15.00*		20.00*	20.50*	21.10**	19.90*	21.00**
LS	Td	86.50*	74.90*	107.70**	85.60**	87.30*	72.90**	134.00**
Steadiness	F	22.70*		52.40**	35.70*	74.90**		
	Fd				10.70*			
LF	F							
	Fd			11.70*				24.90*
	Td							
Tapping 1.p.				14.20*			12.90*	43.10**
2.p.				18.00*	15.40*			27.20**
SS	Td	116.70**	112.00**	95.70**	76.60**	145.60**	166.80**	125.30**
PR 1.p.	F	4.10*	4.30*	6.40**	4.70*	6.30*	4.70**	7.10**
	Fd	33.60*	25.10**	33.40**	41.70**	41.80**	44.50**	40.20**
2.p.	F	6.30*	6.20*	8.40*	4.50*	7.60*	4.80**	6.70**
	Fd	45.90*	37.30**	47.20**	45.60**	45.00**	45.70**	41.10**
		i	f	i	f	i	f	i

psychopathology ratings, but showed no further improvement. However, most of these patients showed good social adjustment (working, living with their families), which is not well documented in the BPRS.

Therefore, again careful consideration will be given to the correlations of motor performance with psychopathological variables. Additionally the need to perform a longitudinal prospective study is suggested (in preparation, including topographic EEG and regional cerebral blood flow measurements during motor performance in these patients and screening effects of different therapeutical efforts).

Extrapyramidal Side-Effects

The rating scales of Webster (1968) and Simpson and Angus (1970) were performed in all patient groups at every motor examination in order to screen for correlations between extrapyramidal side-effects of the antipsychotic drugs administered and motor symptoms observed.

Other Variables

In order to screen for correlations of motor performance with intellectual and concentration abilities we performed the Hamburg-Wechsler-Intelligenztest in a shortened version (WIP; Dahl 1972) and the Revisions-Test (Stender and Marschner 1972).

Only the first examination date (as soon as possible after admission) was used to carry out these tests; we did not do re-tests at the time of discharge, because there might be serious test-retest biases.

Since we did not find significant correlations between these variables and the motor deficits in our previous investigation, we did not force reluctant patients to perform these tests, in order not to endanger the motor follow-up. Thus we were able to collect these data in 41 (of 57) patients only and we will have to restrain statements to correlational interpretation only.

Results

Results Obtained with the Motorische Leistungsserie (MLS)

For each item of the MLS we performed analyses of variance, tested the homogeneity of variances using Bartlett tests and subsequently did multiple pair comparisons by Duncan tests, thus applying the statistical procedures described previously (Guenther and Gruber 1983).

Only significant ($P<5\%$) differences of group means are displayed in Table 3 (initial and final examination), and as can be seen we reproduced our previous findings of motor performance deficits, occurring in every schizophrenic patient group including the interval patients.

These performance deficits showed equally in all schizophrenic groups in the test items aiming (duration of the task), short and long sticks (duration of the task), and the pursuit rotor task variables (number and duration of errors in both halves of the task).

Interestingly, these motor performance deficits shown with the MLS were similar at the final examination. Also similar MLS items separated every schizophrenic patient group from the control group (Table 3).

Table 4. Differences of means of the sum scores of motor performance quality obtained with the Lincoln-Oseretzky Scale, initial and final examinations. Analyses of variance were performed over the sum scores with subsequent multiple pair comparisons by Duncan tests. NAT = not actually treated at the time of the admission; TCL = treated with neuroleptics at admission and then by the clinician; TST = treated with neuroleptics at the time of admission and subsequently by study neuroleptic drugs; INT = interval patients in a relatively symptom-free period, not hospitalised; CNT = control persons. i = initial, f = final examination, N.S. = not significant, * = $P<5\%$, ** = $P<1\%$

		NAT	TCL	TST	INT	CNT
NAT	i	–				
	f	–				
TCL	i	0.15 N.S.	–			
	f	1.06*	–			
TST	i	0.42 N.S.	0.27 N.S.	–		
	f	1.14**	0.20 N.S.	–		
INT	i	0.47 N.S.	0.62 N.S.	0.89 N.S.	–	
CNT	i	1.52**	1.67**	1.94**	1.05*	–
	f	1.92**	1.02*	0.88*	–	–

Table 5. Differences of means of the sum scores of motor performance quality obtained with the motor subtest of the Luria-Nebraska Neuropsychological Battery, initial and final examinations. Analyses of variance were performed over the sum scores with subsequent multiple pair comparisons by Duncan tests. NAT = not actually treated at the time of admission; TCL = treated with neuroleptics at admission and then by the clinician; TST = treated with neuroleptics at the time of admission and subsequently by study neuroleptic drugs; INT = interval patients in a relatively symptom-free period, not hospitalised; CNT = control persons. i = initial, f = final examination, N.S. = not significant, * = $P<5\%$, ** = $P<1\%$

		NAT	TCL	TST	INT	CNT
NAT	i	–				
	f	–				
TCL	i	4.86 N.S.	–			
	f	2.26 N.S.	–			
TST	i	1.94 N.S.	2.92 N.S.	–		
	f	1.11 N.S.	1.15 N.S.	–		
INT	i	5.00 N.S.	0.14 N.S.	3.06 N.S.	–	
CNT	i	14.38**	9.52**	12.44**	9.38**	–
	f	9.14**	6.88**	6.88**	–	–

Thus, our findings with the MLS are possible evidence that motor deficits (1) are observed already in untreated (first manifestation) patients, (2) tend to persist toward the end of an acute phase of schizophrenic illness in the same patients (and presumably even longer:), and (3) exist in socially well-adapted interval patients (without productive symptoms).

Results Obtained with the Modified Lincoln-Osteretzky Scale (LOS)

According to the construction of this subtest (detailed previously, Guenther and Gruber 1983) our chosen five test items have had factorial loadings similar in factor analysis studies (Thams 1955). Thus, we felt justified in calculating an ordinal-scaled sum score of motor performance over these items. Then, for the initial and final examination separately, we performed analyses of variance over these sum scores, and did multiple group comparisons with Duncan tests, as described above (for the MLS results) and previously (Guenther and Gruber 1983).

Only the differences of group means of these sum scores are shown in Table 4, and again there were motor performance deficits in untreated and neuroleptic-treated inpatient schizophrenics and in the interval patients as compared to normal subjects. This was found both in the initial and the final motor examination with the LOS. No significant differences were obtained within the schizophrenic patient groups, either at the initial or final examination.

Results Obtained with the Motor Subtest of the Luria-Nebraska Neuropsychological Battery (LNB)

According to the test construction, the items of the motor subtest are again supposed to load highly on a "general motor ability" factor (Luria 1970, Golden 1979), thus allowing the calculation of ordinal-scale sum scores in order to screen motor performance quality. These sum scores were analyzed statistically by analyses of variance and subsequent multiple pair comparisons by Duncan tests, as described above for the LOS items and detailed previously (Guenther and Gruber 1983). These analyses were done for the initial and final examination separately.

Only the differences of group means of these sum scores are shown in Table 5; there were clear motor performance deficits in untreated and neuroleptic-treated schizophrenic patients and interval patients as compared to normal control persons. The results were very similar to those obtained with the LOS (Table 4) thus possibly supporting each other.

These differences of motor performance were again shown both for the initial and the final examination between schizophrenic and normal persons; no differences were obtained between the different groups of schizophrenic patients.

Thus, our findings obtained with the LOS and the LNB are additional (to the findings of the MLS) evidence that motor performance deficits (1) already exist in untreated schizophrenic persons, (2) have the tendency to persist to the end of an acute phase of schizophrenic illness (i.e., were observed in the final examination as well as the initial one), (3) exist in socially well-adapted and not productively psychotic schizophrenics in the remission interval, and (4) do not exist in age and sex matched control persons.

Results of the Correlation Statistics

Correlations between the Motor Performance and Extrapyramidal Motor System (EPMS) Side-Effects of the Antipsychotic Drugs Aministered. We calculated, independently for the initial and final examinations, rank correlation coefficients R for every motor test item to the sum score of the Webster (1968) and Simpson and Angus (1970) rating scales of EPMS side-effects. The correlation coefficients for the initial examination were based upon the data of 57 schizophrenic patients, those for the final examination upon 41 (without the interval patients, who were examined only once).

The 76 total coefficients ranged from -0.3640 ($P=0.0348$) (tapping, 2. part to sum score of the Simpson scale) to 0.4433

($P=0.0091$) (long sticks, total duration to sum score of the Webster scale). There were 40 positive and 36 negative correlations to the quality of motor performance. Only 6 coefficients had $P<5\%$, not exceeding the above ranges and thus not accounting for more than a maximum of 20% common variance.

Our results do not support a significant covariation of motor performance quality with possible EPMS side-effects of the antipsychotic drugs administered. This finding might be further supported by the reported existence of a PMS in untreated schizophrenics.

Correlations between Motor Performance and Psychopathological Symptoms. In an analogous manner to that described above, we calculated 380 Rs of the motor test items to the five factors of the BPRS (Overall and Gorham 1976) (Table 2).

These coefficients ranged from 0.7015 ($P<0.001$) (line following, total duration to BPRS thought disturbance) to -0.5190 ($P=0.0016$) (LNS total score to BPRS thought disturbance).

Only 31 (of 380; 8%) of the Rs calculated had $P<5\%$, which is not very different from the number of significant Rs to be expected by chance. There were 56% positive and 44% negative correlations of the motor performance quality to the psychopathological variables. Again, our results do not support a close covariance of motor performance quality with variables of the psychopathology, as they had seen screened by the BPRS. Thus, the PMS does not seem to be a phenomenon secondary to other psychopathological symptoms, but seems to exist independently ("Basisstörung", Süllwold 1977).

Correlations between Motor Performance and Person-Related (Concentration and Intellectual Abilities) and Biographical (Age, Sex) Data. In contrast to the procedures described above, we calculated the rank correlations only for the initial examination, since this seemed sufficient for the variables age and sex necessary for the psychometric test variables, in order to avoid test-retest biases.

For the concentration (revision test, Stender and Marschner 1972) and intellectual investigations (WIP, Dahl 1972) we collected the data of 38 patients (3 of the inpatients refused the psychological testing; no such examination possible on the interval patients). Thus, our results might contain selection biases and are to be interpreted with caution.

Of 38 calculated Rs (of the 19 motor items to the global IQ and to the fault-stanine value of the Revisions test 12 (32%) were significant. They ranged from -0.5881 ($P<0.001$) (pursuit rotor, fault duration, 2. part to IQ) to 0.5717 ($P=0.001$) (tapping, 2. part to IQ). Thus, for the variables "concentration and intellectual abilities" there was some evidence for low to medium covariation with motor performance quality.

This covariation did not exceed 35% common variance, and only 33% of the correlation coefficients calculated were significant.

Thus, there was at the most a low to medium covariation of motor performance quality with concentration and intellectual abilities. This may indicate involvement of a "global motor factor" into the PMS, possibly covarying with other more "global" functions, as reflected in the total IQ and the performance quality in a concentration test. More evidence for such an involvement is discussed below.

For the variable age we found only 2 significant (of 19) Rs, the highest being 0.3643. For the variable sex no significant rank correlation was found.

Thus, we did not find any evidence in the age range of the patients examined (i.e. from 17 to 58 years), of any covariance with age. This is another possible clue indicating an independent PMS, which does not alter with increasing age.

This motor syndrome in schizophrenic patients did not show covariation with sex supporting the assumption that the slight sex differences in our schizophrenic patient groups did not affect our results.

Results of the Factorial Analyses

Due to space limitations it is not possible to provide full details of the methodology and results of the factorial analyses, and they will be reported in full separately (Guenther and Guenther, submitted). However, the results are sufficiently important to be outlined in this paper.

The database for the factorial analyses was provided by 57 schizophrenic patients (initial examination only) with regard to 26 motor variables (17 MLS variables, 5 LOS variables, 4 LNB variables); note that no sum scores of LOS and LNB entered the analyses, but the detailed results of single items resp. item groups, described previously (Guenther and Gruber 1983). For the controls the database consisted of 40 persons, 25 were the controls from this study, and 15 were the controls from our previous study, well comparable for age (30.9 years vs 30.5 years) and sex distribution (male/female 16/9 vs 10/5). Statistical preevaluation was performed, in order to screen the homogeneity of the two groups (e.g., testing the homogeneity of variances by Bartlett tests, Clauss and Ebner 1974). The motor items involved were as described for the schizophrenic persons.

We initially performed main axis factorial analyses, orthogonally, exit criterion being Eigenwert above 1.0; these analyses were performed independently for the 57 schizophrenics and the 40 controls and for the whole sample of 97. Table 6 shows the extracted factors and their explained variance. There were a similar number of extractable factors both in schizophrenics and normals, which did not change between the two groups. Additionally, it should be noted, that in all three groups, there was a similar "explained" variance (71.3% versus 82.1% versus 71.1.%). The configuration of items and

Table 6. Variances explained by unrotated factors (Eigenwerte above 1) based on the intercorrelations of 26 motor variables in 3 independent factorial analyses (on 57 schizophrenic (SCH) and 40 normal (CNT) persons, and the total sample of 97 (TOT) persons). Percentage values

Eigenwert Nr.	SCH	CNT	TOT
1	18.2	31.6	32.1
2	12.8	13.3	9.7
3	10.2	10.6	8.6
4	9.5	8.6	7.4
5	6.1	6.5	5.0
6	5.5	6.1	4.2
7	4.9	5.4	4.0
8	4.0		
Sum of variance explained	71.3	82.1	71.1

Table 7. Structure of the first orthogonal factor "general motor ability". Factor loadings of the 26 motor items on this factor in schizophrenic (SCH) and control (CNT) persons. + labels the variables, which showed loadings above 0.30, considered as "marker variables" for this factor. In the last two columns are the "Trennschärfe" coefficients (item-test correlation r_{it}) for SCH and CNT. Abbreviations: LOS 1–5 = single items of the LOS, LNB 1–4 subgroups of motor items of the LNB (Guenther and Gruber 1983); all other abbreviations cf. Tables 4 or 5

Item		SCH	CNT	+	SCH (r_{it})	CNT (r_{it})
Aiming	F	0.24	−0.20			
	Fd	0.25	0.03			
	CR	0.10	0.15			
	Td	0.52	0.67	+	0.38	0.12
LS	Td	0.58	0.88	+	0.54	0.41
Steadiness	F	0.61	0.68	+	0.61	0.30
	Fd	0.49	0.69	+	0.53	0.33
Lf	F	0.26	0.84			
	Fd	0.25	0.84			
	Td	0.05	0.43			
Tapping	1.P.	−0.51	0.02			
	2.P.	−0.57	−0.09			
SS	Td	0.44	0.64	+	0.42	0.14
PR 1.P.	F	0.58	0.58	+	0.32	0.68
	Fd	0.61	0.85	+	0.51	0.64
2.P.	F	0.58	0.71	+	0.32	0.76
	Fd	0.60	0.87	+	0.30	0.77
LOS 1	F	0.10	0.00			
LOS 2	F	0.25	0.00			
LOS 3	F	−0.11	−0.05			
LOS 4	F	0.02	−0.11			
LOS 5	F	0.30	0.00			
LNB 1	F	0.48	0.34	+	0.24	0.27
LNB 2	F	0.49	0.08			
LNB 3	F	0.13	0.53			
LNB 4	F	0.32	0.33	+	0.28	0.42

loading on each factor will be mentioned briefly, except for the reults for the first factor which are given in full detail. Our factor 2 seems to correspond to the "Fleishman" factor (Fleishman 1954; Schoppe 1974; Guenther and Gruber 1983) "speed of precise arm-hand movements", factor 3 to "arm-hand steadiness", factor 4 to "precision of arm-hand movements" and factor 5 to "wrist-finger speed".

Our factor 6, only partly homogenous, seems to be represented by the LOS items and factor 7 by parts of the LNB. Although, in our analyses, LOS and LNB did not contain items which load highly on our first "general motor ability" factor, they seemed to be nonetheless rather homogenous (representing factors 6 and 7), allowing retrospectively calculation of sum scores (relying on the reported test construction).

To evaluate the structure of our first "general motor ability" factor, the first column in Table 7 lists the loadings of the 26 motor items of the schizophrenics, and column 2 those of the controls. Those motor variables, which loaded in both groups higher than 0.30, were considered as being "marker variables" on this first factor. Column 3 labels these 11 "together" variables and columns 4 and 5 list the "Trennschärfe" coefficients (i.e. item-test correlation r_{it}) for the schizophrenic (column 4) and control (column 5) groups.

If the hypothesis that these items play a special role in separating schizophrenic and control groups is correct, there should be a predominance of these items amongst the significantly poorer performed motor test items.

If we examine Tables 3, 4, and 5 for the variables which consistently separated the patient groups from the control groups we find the following variables: aiming total duration, long sticks total duration, short sticks total duration, pursuit rotor-all four variables, LOS sum score, LNB sum score.

Comparing these items to those being evaluated as "marker variables" for the first factor "general motor ability", there was a striking congruence: all items (except the LOS items), separating the schizophrenics from the controls were "marker variables" on the first factor. This striking finding was statistically significant (e.g. r (pbis) = 0.65, $df = 24$, $P < 0.001$).

In conclusion, these results of our factorial analyses reveal evidence that the factorial structure of motor performance, as measured by our test battery is similar in schizophrenic and normal control persons, and that performance deficits shown in schizophrenics (the PMS) might be due to an impairment of a first factor, a "general motor ability" factor.

Discussion

Previous findings obtained with our motor test battery (Guenther and Gruber 1983) suggested motor deficiencies in schizophrenic and endogenous depressed patients, consisting of impaired fine movements of the dominant right hand, lips, and tongue, and deficiencies of complex motor coordination of the extremities.

In order to reevaluate these findings we conducted two cross-validation studies on 57 schizophrenic and 57 endogenous depressed patients. In this paper we report the results of the study on the 57 schizophrenic persons as compared to 25 age and sex matched control persons.

We reestablished findings suggesting the above described motor symptoms in schizophrenic patients and proposed the "psychotic motor syndrome (PMS)" (we have reproduced such a syndrome in endogenous depressed patients too – to be reported separately).

The results presented in this study produced some further support that PMS in schizophrenic patients is not simply secondary to treatment with antipsychotic drugs, nor to extrapyramidal motor side-effects of such treatment. We tried to examine this prospectively by examining the same patients before and after drug treatment.

As is suggested by our results yielding only low to medium correlations between the PMS and EPMS side-effects as reflected by the Webster and Simpson and Angus rating scales (Webster 1968; Simpson and Angus 1970), there were no closer correlations between motor performance parameters of our PMS and EPMS side-effects of the drugs used for treatment.

As the PMS existed similarly both before and after treatment it cannot be considered as being directly related to drug treatment itself.

These findings might appear surprising and reflect that voluntary movements (as measured by our battery) may be impaired in patients with or without drug treatment (and their possible side-effects on the EPMS).

Supporting evidence for this can be obtained from studies establishing motor deficiencies in endogenous psychotic patients long before psychopharmacological drugs had been introduced (Wulfeck 1941).

Thus we have reproduced evidence of motor deficiencies in psychotic patients as an independent "basic syndrome", already existing in untreated patients as well as during drug treatment, and existing independently of the actual psychopathological status (signs of a trait marker): the PMS exists similarly in serverely ill patients at the time of admission, as well as − in the same patients − at the time of discharge, when the psychopathology has improved. Consequently there were only low to medium correlations with psychopathological status as reflected by the five factors of the BPRS, the PMS was demonstrable in our interval patient group as well, who did not show signs of active psychosis.

However, some critical methodological remarks should be noted. The interval patients were not identical with the inpatients. Only the inpatient groups, both treated and untreated at the time of admission were followed prospectively until the time of discharge with regard to motor symptoms, drug treatment, side-effects, and psychopathology.

Clearly, a prospective follow-up of the PMS and the other variables mentioned for longer time periods after discharge is needed, but this had to be postponed, in order to clarify which parameters (by which methods) merited follow-up in such a time consuming and costly study.

We extended our search for changes underlying a possible PMS to neurophysiological methods like the topographical EEG and the regional cerebral blood flow (rCBF; Xenon-133 inhalation method). These studies can only be outlined here, but they do provide some possible further support for the existence of the PMS and differential diagnostic and therapeutic values possibly attached to it. First, we conducted two studies with a 16-channel topographic EEG in 20 productive (type I, Crow et al. 1981) schizophrenic patients as compared to 20 age and sex matched controls) Guenther and Breitling 1985, Guenther et al. 1986). We investigated EEG correlates of central cortical activation pattern during simple to complex motor activity in the dominant right hand.

In both studies we found functional abnormalities as reflected by the topographical EEG in schizophrenic patients during motor activity: signs of left hemisphere hypofunction, and signs of possible concomitant "compensatory?" right hemisphere overactivation.

In a third study (not yet published) with the same methodology, we investigated 10 "negative" (type II, Crow et al. 1981) schizophrenic patients as compared to 10 matched controls, and found signs of a diffuse bilateral "hyporeactivity" (hypofunction) both during motor function of the dominant right and the nondominant left hand. In all studies, the healthy control persons showed clear "lateralisation of hemispheric function" to the contralateral cortex during simple hand and finger movements, and a bilateral activation pattern in complex ones, thus being different from schizophrenic patients both of type I and II.

Consequently in our subsequent rCBF investigations we concentrated on simple motor activity of the dominant right hand in both type I and type II schizophrenic patients as compared to severely and almost recovered endogenous depressed and healthy persons ($n = 8$ in each of all groups mentioned; Guenther et al. in press).

Again, both schizophrenic subgroups showed circulatory activation patterns which were clearly different from those of normal controls: the type I patients showed bilateral hyperfunction in contrast to the type II patients who showed bilateral hyporeactivity. In normal persons, we found a clear restricted flow increase in the contralateral primary motor region as has been reported by others (Lauritzen et al. 1981).

Thus, virtually all our neurophysiological studies have so far revealed evidence of a disturbed "laterality" of hemispheric function during motor performance in schizophrenic patients supporting theories known from the literature (Flor-Henry 1976; Gruzelier and Hammond 1976).

Further support for a possible impaired "laterality of function" comes from other working groups, using different "functional windows": e.g., Gur et al (1983, 1985) performed rCBF studies in both unmedicated and medicated schizophrenic patients, using a cortical activation schedule of spatial imagination and verbal tasks. Whereas normal control persons showed predominantly right hemispheric flow increases during the spatial imagination, schizophrenics showed them in the left hemisphere. During verbal stimulation, the control persons demonstrated predominant left hemispheric increases, whereas in schizophrenic persons such a "laterality" was absent (diffuse bilateral activation). Neuroleptic medication tended to change these functional alteration towards "normal lateralisation" (Gur et al. 1985).

During an eye-tracking task with positron emission tomography using 18 F-deoxyglucose as tracer, Brodie et al. (1985) found both an "underreactivity" of glucose metabolism predominantly in type II schizophrenics and a left hemisphere basal ganglia hyperfunction, already existing during resting conditions.

Since there are inhibitory connections from the basal ganglia to the motor cortex (amongst others), there might be a tempting, but speculative, relation to our left hemispheric cortical hypofunction during motor function, which merits further investigation.

Thus our psychometric and neurophysiological evidence of a "general motor ability" impairment in schizophrenics, demonstrating itself by our reestablished PMS and signs of disturbed hemispheric laterality of motor function might possibly provide some further information that (1) a wide variety of motor functions are affected in schizophrenics (from eye movements to the complex motor coordination of the extremities), (2) perhaps motor stimulation might be of some further use as a "functional window" in studies using PET, rCBF, and quantitative EEG facilities, (3) the differential diagnostic value of motor symptoms in psychiatric patients might be further supported, and (4) it might be of some clinical benefit, to use such a PMS as a "target syndrome" for motor training programs as an additional treatment (Guenther 1984).

Acknowledgement. We thank G. Niller and B. Müller for their valuable assistance.

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Received November 18, 1985