Eur Arch Psychiatr Neurol Sci (1988) 237:65–73

European Archives of Psychiatry and Neurological Sciences

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

III. Cross Validation Study on Depressed Patients: The Psychotic Motor Syndrome as a Possible State Marker for Endogenous Depression*

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Summary. This study investigates the presence and course of motor symptoms in endogenous ($n = 42$) and non-endogenous ($n = 15$) depressed patients (both medicated and unmedicated) in comparison to 15 healthy control persons. As in our previous studies on schizophrenic and depressed patients, we used a motor test battery, which consisted of the Motorische Leistungsserie, a modified version of the Lincoln Oseretzky Motor Development Scale and the motor subtest of the Luria Nebraska Neuropsychological Battery. Previous findings had suggested the existence of a psychotic motor syndrome (PMS) in endogenous depressed patients (and schizophrenics), involving disturbances of the lips and tongue, fine and gross movements of the dominant right hand and the complex motor coordination of the extremities. We re-confirmed the PMS in acute endogenous depressed patients, both medicated and unmedicated. Such a motor syndrome did not exist in healthy controls, or in non-endogenous depressed patients, irrespective of the severity of the depressive syndrome. This PMS showed a clear improvement with the amelioration of the depressive symptoms in endogenous depressed patients towards the end of the hospital treatment period and disappeared entirely in patients in a symptomfree interval. This may be suggestive of a possible role for the PMS as a state marker for endogenous depression, in contrast to the persistence of the PMS in schizophrenics (trait marker), described previously. The results of factorial analyses on motor performance did not reveal differences in the factorial structure between depressives, schizophrenics and normals. However, they indicated disturbances on a general motor factor, which may account for the performance deficits in psychotic patients. This may be in accordance with signs of diffuse alterations of brain function during analogous motor activation in these patients demonstrated recently with neuro-imaging methods.

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

Introduction

Motor symptoms in endogenous psychotic patients were described long before antipsychotic medication was introduced into the treatment of those patients. In 1911 Bleuler described motor abnormalities in schizophrenic and endogenous depressed patients and proposed a schizophrenic and manic-depressive catatonia. He used the term catatonia in order to describe a close connection between motor and psychopathological symptoms, which had been suggested by Kahlbaum as early as 1874. Kleist (1908), in continuation of the work of Wernicke concluded from the similarity of clinically determined motor symptoms

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

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Abbreviations: Bf-S Befindlichkeits-Skala; CNT Control Persons; DEP Depressive Patients; ED Endogenous Depressed Patients; EDT Endogenous Depressed Patients, treated; EDU Endogenous Depressed Patients, untreated; HAMD Hamilton Depression Scale; INT Interval Patients; LNB Luria-Nebraska-Battery; LOS Lincoln-Oseretzky-Scale; MLS Motorische Leistungsserie; NED Non-Endogenous Depressed Patients; PD-S Paranoid-Depressivitäts-Skala; PMS Psychotic Motor Syndrome; rCBF regional Cerebral Blood Flow, SCH Schizophrenic Patients

in cerebral organic and schizophrenic/affective psychotic illness, that there should be a cerebral-organic aetiology for endogenous psychoses too. Kraepelin (1896) and Hoch (e.g. 1904 for review) investigated the writing balance in psychiatric patients and found evidence in endogenous depressed patients, that there "exists not so much a motor excitement as an increased motor excitability" in these patients. In 1940 Wulfeck reported impairments of precision in voluntary hand movements, slowing in rhythm and increases of the reaction time in endogenous depressed (and schizophrenic) patients, as compared to neurotics and normal control subjects.

Later psychometric studies (Greden and Carroll 1981) seemed to confirm a wide variety of motor deficiencies in affective disorders, both in medicated and unmedicated patients. They ranged from impairments of psychomotor expression of emotions in the facial musculature (Whatmore and Ellis 1959; Schwartz et al. 1976a, b; Oliveau and Willmuth 1979), increased speech pause time (Pope et al. 1970; Szabadi et al. 1976; Greden and Carroll 1980), to alterations in gross motor activity (Kupfer and Foster 1973; Kupfer et al. 1974). Various findings in fine motor functions concerned predominantly retardation of simple and complex motor tasks in endogenous depressed patients (Haase and Krantz-Gross 1956; Hartwich 1970; Yates 1973), although some authors (e.g. Blackburn 1975) found differences between monopolar and bipolar patients.

Concerning gross motor activity, Kupfer and associates (1973, 1974) measured whole body motility with specially designed motor-activated electronic equipment and reported as major findings: unipolar depressed patients exhibited significantly more motor activity before treatment than bipolar depressives; when the depressive syndrome improved, this difference disappeared; there were diurnal variations of whole body activity in depressed patients, who showed a higher percentage of 24-h activity during the night time as compared to normal subjects.

Previous own findings have been indicative of a psychotic motor syndrome (PMS) existing similarly in (treated and untreated) schizophrenic (SCH) and (monopolar and bipolar) endogenous depressed (ED) patients (Günther and Gruber 1983; Günther et al. 1986a). This syndrome consisted of disturbances of the lips, tongue and mouth, fine and gross movements of the dominant right hand as well as the complex co-ordination of the extremities. It had shown evidence of a trait marker in schizophrenic patients, i.e. it persisted independently of the acute psychotic state in these persons.

Therefore, in this study, we attempted to check the existence of such a PMS in ED (monopolar and bipolar) patients, in comparison to non-endogenous depressed (NED) and healthy control (CNT) persons. Special attention was given to the possible role of anti-depressant medication and the course of the PMS with respect to the psychopathological status.

Method

Subjects were in-patients of the Psychiatric University Hospital links der Isar, Nussbaumstrasse, Munich ($n = 45$) and out-patients of a private practice ($n = 12$)[1]. Table 1 summarizes biographical and diagnostic data of the patients and the CNT subjects. Control persons were members of the staff (doctors, nurses, technical personal/labourers, medical students) of the above hospital with no history of psychiatric, neurological and/or medical illness, not taking any medication, drugs or alcohol regularly at the time of the investigation. As can be seen from Table 1, the average age of the non-endogenous depressed patients (NED; $n = 15$) was lower, than that of the ED, both untreated (EDU; $n = 15$) and treated (EDT; $n = 15$). Additionally, there was a predominance of females in the endogenous patients groups, which was reversed in the non-endogenous group.

The age and gender distribution of the CNT persons was matched as carefully as possible to the ED patients, thus allowing some differences in these variables in comparison to the NED group. Although in our previous investigations (Günther and Gruber 1983; Günther et al. 1986a), there were no correlations of the motor performance with gender or age (within the above age range) either in normals or patients with depressive symptoms, this issue will be given special attention in the *Results* section.

The NED and EDU patients were not being treated with antipsychotic medication at the time of the investigation. However, whereas in the NED patients ⅔ had never received such medication (as far as this can be explored reliably), most of the EDU had had a drug-free period of between 1 week and 1 month. Although there may be biases due to this, there was no way of avoiding this problem, since EDU patients are very rare in our hospital. The diagnostic procedures involving ICD-9 and DSM-III criteria, the establishment of right-handedness, exclusion of organic brain disease and the examination situation were analogous to those in our previous investigations on schizophrenics (Günther et al. 1986a).

Psychomotor variables. We used a motor test battery, consisting of the Motorische Leistungsserie (MLS, Schoppe 1974), the Lincoln Oseretzky Motor Development Scale (LOS, Reinert 1966; Günther 1980) and the Motor Subtest of the Luria Nebraska Neuropsychological Battery (LNB, Luria 1970; Golden 1979). Every patient was examined twice (on the day after admission and before discharge, always at the same time of the day, as far as possible), except for the interval (INT) patients, who could be studied only once. The investigators of the psychomotor variables were unaware of the diagnoses of the patients and did not participate in the evaluation of psychopathological status.

Psychopathology variables. The Hamilton Depression Scale (HAMD, Hamilton 1960), the Paranoid-Depressivitäts-Skala (PD-S) and the Befindlichkeits-Skala (Bf-S) (von Zerssen 1973) were used for psychopathological assessment in all pa-

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Table 1. Subjects

$n = 72$	EDU ($n = 15$)	EDT ($n = 15$)	NED ($n = 15$)	INT ($n = 12$)	CNT ($n = 15$)
Mean age (years)	42.3	49.8	29.4	47.5	41.3
Range	25–62	35–61	20–46	27–59	29–53
Male/female	3/12	7/8	12/3	4/8	5/10
Mean duration of illness (years)	6.63	8.07	1.33	5.83	–
Diagnoses (ICD-9; DSM III)	296.1 = 10	296.1 = 13	300.4 = 8	296.1 = 8	–
	296.3 = 5	296.3 = 2	309.0 = 7	296.3 = 4	–
Last treatment with psychotropic drugs					
Never before	1		10		
More than 1 year ago			1		
More than 1 month ago	2		1		
More than 1 week ago	12		3		
Patients treated with					
Antidepressants at admission		14		10	
Antidepressants and/or neuroleptics		3		2	
Antidepressants and/or tranquilizers		3		3	
Antidepressants and/or lithium		4		3	

EDU = endogenous depressed patients, untreated at the time of admission; EDT = endogenous depressed patients treated at admission and afterwards; NED = non-endogenous depressed patients; INT = patients in a symptom-free interval, not hospitalised; CNT = control persons

Table 2. Psychopathological status of the patients

$n = 57$			EDU ($n = 15$)	EDT ($n = 15$)	NED ($n = 15$)	INT ($n = 12$)
Hamilton Depression scale (mean of total score)		At admission	25.5	24.4	16.1	9.1
		At discharge	10.7	13.2	9.2	
Paranoid-Depressive scale (Zerssen 1976)	D score	At admission	25.9	25.4	16.2	8.3
		At discharge	10.1	14.1	11.7	
	P score	At admission	8.1	7.7	3.9	0.5
		At discharge	2.3	3.6	2.2	
Befindlichkeitsskala BfS (Zerssen 1976)	Total score	At admission	39.1	36.9	29.9	9.9
		At discharge	12.3	11.5	11.0	

Abbreviations see Table 1

tient groups, both for the initial and final examination. Table 2 shows the scores on these scales.

As can be seen from this Table, the ED in-patients showed a distinct depressive symptomatology on all scales at admission, which improved considerably towards discharge (at which time there were no significant differences present between EDU, EDT, NED and INT patients). However, there was a lesser degree of severity of the depressive symptoms on all scales in NED patients at admission.

Other variables. In order to screen for correlations of the 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 initial examination date (as soon as possible after the motor test) was involved in these tests; re-tests at the time of discharge were omitted because of possible test-retest biases. Additionally, in order not to endanger our motor re-examination, we did not force reluctant patients to perform this type of psychometric tests. Thus we were able to collect these data in only 38 (of 57) patients.

Data base for factorial analyses. The data base for the intercorrelation matrices consisted of 26 motor variables (17 MLS, 5 LOS, 4 LNB; no sum scores entering the analyses) of $n = 113$ depressive patients ($n = 57$ from this study, $n = 30$ from the 1983 study and $n = 26$ endogenous depressive patients from a study, which attempted to establish preliminary evidence of the value of motor training programmes in the treatment of psychotic patients, first reported by Streck 1986, the complete data will be reported as final part of our series later on).

Results

Results Obtained with the MLS

For each item of the MLS we performed the non-parametric H-test of Kruskal-Wallis, followed by

Table 3. Significant differences of means obtained with the Motorische Leistungsserie in the INITIAL EXAMINATION. H-tests of Kruskal and Wallis were calculated over each item, with subsequent multiple pair test analysis by Dunns tests

a		EDU/CNT	EDT/CNT	NED/CNT	INT/CNT	EDU/EDT	EDU/NED	EDT/NED	EDU/INT	EDT/INT
Aiming	F									
	Fd									
	CR									
	Td	36.90**	59.90**				26.20*	49.90*	27.60*	50.60*
LS	Td	68.00**	91.60**				57.20*	80.80*	64.30*	87.90*
Steadiness	F	37.90**	37.50**				33.20*	32.80*		
	Fd	13.00**	14.60**		2.90*		13.00*	14.60*		
LF	F									
	Fd									
	Td									
Tapping	1. P.	23.10**	23.80**				16.00*	16.70*	28.20**	28.90**
	2. P.	18.40**	15.10**				16.70**	13.40*	15.10*	11.80*
SS	Td	57.80**	110.70**				34.90*	108.90**		
PR 1. P.	F	11.20**	10.00**				5.70*	4.70*	10.01**	8.90*
	Fd	48.40**	53.00**				26.50*	31.10**	38.30**	42.90**
2. P.	F	11.60**	9.40**				6.60*	4.40*	9.50*	7.30**
	Fd	42.00**	50.40**				24.80*	29.20*	34.70**	39.10**

b		EDU/EDT	EDU/NED	EDT/NED	EDU/CNT	EDT/CNT	FINAL EXAMINATION
Aiming	F						
	Fd						
	CR						
	Td		16.60*	20.50*	24.90*	28.80*	
LS	Td			68.50*			
Steadiness	F		33.30*	26.20*	28.30*	21.20*	
	Fd		11.10*	6.80*	9.10*		
LF	F		15.60*	30.00**			
	Fd						
	Td						
Tapping	1. P.			15.40*		19.40*	
	2. P.			11.80*		15.80*	
SS	Td			87.00*		74.50*	
PR 1. P.	F						
	Fd						
2. P.	F						
	Fd						

EDU = endogenous depressed patients untreated at the time of admission; EDT = endogenous depressed patients treated at admission and afterwards; NED = non-endogenous depressed patients; INT = patients in a symptom-free interval, 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. * = P below 5%, ** below 1%

multiple pair test comparisons by Dunns tests (Clauss and Ebner 1974; Römig 1984). Thus we followed the statistical procedure detailed previously (Günther and Gruber 1983), with the difference, that we used non-parametrical H-tests (instead of analyses of variance) and multiple pair test methods (Dunn's tests instead of Duncan tests), since the initially performed Bartlett tests indicated inhomogeneity of variances in some items of the MLS (and the other subtests).

Only significant ($P<0.05$) differences of groups means are displayed in Table 3a for the initial (day

Table 4. Significant differences of means of ratings of performance quality for every item of the Lincoln-Oseretzky Scale, initial and final examinations. Kruskal-Wallis tests were performed over every item with subsequent multiple pair comparisons by Dunns tests

Item	EDU/EDT	EDU/NED	EDT/NED	EDU/INT	EDT/INT	EDU/CNT	EDT/CNT	NED/CNT	INT/CNT	NED/INT
Initial examination										
LOS 1										
LOS 2										
LOS 3		0.47*	0.54**	0.55**	0.62**	0.60**	0.67**			
LOS 4		0.67**	0.60**	0.63**	0.56**	0.67**	0.60**			
LOS 5				0.60**	0.67**	0.60**	0.67**			
Final examination										
LOS 1										
LOS 2										
LOS 3										
LOS 4							0.60**			
LOS 5						0.60**	0.47*			

Abbreviations see Table 3
* = P below 5%, ** = below 1%

after admission) and Table 3b for the final examination (day before discharge). As can be seen from Table 3a, both in EDU and EDT patients, we reproduced our previous evidence of motor abnormalities. These disturbances were not found in the NED patients, nor in the (ED) patients in a symptom-free interval. Statistical screening did not yield evidence of differences between monopolar (296.1) and bipolar (296.3) patients. Thus, there may be some preliminary evidence for the possible role of the PMS as a state marker for endogenous depression.

Table 3b indicates additional evidence for this state marker quality, since there was considerable improvement of motor disturbances in the ED patients before their discharge, as compared to the day after admission (along with the considerable improvement of the depressive syndrome indicated in Table 2).

Results Obtained with the Modified LOS

We used the performanced scores on each of the five items as a basis for H-tests of Kruskal-Wallis and subsequent multiple pair test comparisons using Dunns tests, thus following the procedure for the MLS items. Table 4 shows the results, both for the initial and final examinations. The items LOS 3 (rhythmical alternating tapping with the feet and simultaneously making circles with both hands), LOS 4 (rhythmical alternating movements with the feet and tapping with both hands simultaneously with the right foot), LOS 5 (balance on tiptoes with the knees bent) separated the hospitalised ED patients (both treated and untreated) from all other groups (INT, NED and CNT) *in the initial examination.* However, in the final examination, there were no differences between any of the groups (Table 4). Statistical screening did not reveal differences between monopolar and bipolar patients in motor performance as measured by LOS items. Thus, our results obtained with the LOS also seem to indicate that distinct motor deficiencies in ED patients seem to have a state marker quality. These motor symptoms seemed to disappear in the symptom-free interval and were not found in NED or in control persons.

Results Obtained with the Motor Subtest of the LNB

We used the item groups:
LNB 1 (items 1–4) simple repetitive movements with the dominant or both hands
LNB 2 (items 21–23) alternating rhythmical hand movements with both hands
LNB 3 (items 32–33) movements with lips and tongue
LNB 4 (items (36–47) drawings of simple geometrical figures
Further details of the item groups and their selection criteria can be found in Günther et al. (1986a), Rödel (1987) and Gruber (1982). Analogously to our statistical procedures used in the MLS and LOS, we performed Kruskal-Wallis H-tests for each item group with subsequent multiple pair test comparisons using Dunns tests. The results are listed in Table 5. There

Table 5. Significant differences of means of ratings of motor performance quality for item groups of the Luria-Nebrasky-Battery. LNB 1 (item 1–4): simple repetitive movements with the dominant or both hands; LNB 2 (item 21–23): alternating rhythmical movements with both hands; LNB 3 (item 32–33): movements with lips and tongue; LNB 4 (item 36–47): drawings of simple geometrical figures. Kruskal-Wallis H-tests were calculated over each item group with subsequent multiple pair test comparisons with Dunns test

Item	EDU/ EDT	EDU/ NED	EDT/ NED	EDU/ INT	EDT/ INT	EDU/ CNT	EDT/ CNT	NED/ CNT	INT/ CNT	NED/ INT
Initial examination										
LNB 1 (1–4)		0.70*	1.30**	0.98*	1.25**	1.13*	1.40**			
LNB 2 (21–23)		1.54**	1.00**	1.37**	0.83*	1.47**	0.93**			
LNB 3 (32–33)										
LNB 4 (36–47)						2.40**	1.80**	1.80**	3.52**	
Final examination										
LNB 1		0.93*	0.93*			1.07**	1.97**			
LNB 2										
LNB 3										
LNB 4						4.40**	4.47**			

Abbreviations see Table 3

* = P below 5%, ** = P below 1%

were signs of motor impairment in the acute ED patients, both treated and untreated, in comparison to INT, NED and CNT groups *in the initial examination*. These differences seemed to diminish considerably towards the end of the hospitalisation period. Again, our statistical screening did not yield differences of motor performance between monopolar and bipolar patients in the LNB items.

Thus, well in accordance with our results obtained with MLS and LOS methods, we found evidence in favour of a state marker for endogenous depression, fading away towards the end of a depressive phase, not existing in the interval or in NED or CNT persons.

Results of the Correlation Statistics

Due to practical reasons (some patients cooperated only in the motor part of this study and not in the psychological part) we were able to collect a complete set of data only in 38 of 57 depressed patients (14 EDU, 13 EDT, 6 NED, 5 INT). This means, that the correlation coefficients may contain selection biases and the eventual results must be interpreted with caution.

We calculated rank correlation coefficients between the motor (MLS, LOS, LNB) and psychopathological (HAMD, Bf-S and PD-S) variables. There were fewer significant coefficients than would be expected by chance, not exceeding 0.55 in absolute value for the MLS, and 0.37 for LOS and LNB. Recalculation of the correlation coefficients, removing the 6 NED and 5 INT patients did not change the results, i.e. the number of significant correlations between motor performance and psychopathology remained below that which would be expected by coincidence. No coefficient exceeded 0.39 in absolute value. In view of the evidence of a state marker quality for endogenous depression from the motor subtests, this may indicate that there are no systematical covariations between certain aspects of the psychopathology and certain aspects of motor impairment, indicating already a more general and loose covariation between motor and psychopatholgoical symptoms.

Additionally, this may indicate that the motor symptoms disappear along a time dimension in endogenous depression rather than along a psychopathology dimension. Additionally, this lack of close correlation supports the validity of our findings in the NED patients (who showed some lower degree of psychopathology), in accordance with analogous findings in the Günther and Gruber 1983 study. Similarly to our previous results, we found no evidence of significant covariations of the motor symptoms with the variables age, gender, concentration and intellectual abilities (as measured by WIP and Revisionstest). For the MLS no rank correlation coefficient exceeded 0.40; for LOS and LNB there were only 3 coefficients significant with values between 0.46 and 0.61, no more than would be expected by chance. Again these findings may support the validity of our findings of motor performance differences between groups, who show a different age distribution (ED vs NED patients).

Table 6. Variances explained by unrotated factors (Eigenwerte above 1) based on the inter-correlation of 26 motor variables in 3 separate factorial analyses (on 113 depressive, 57 schizophrenic and 40 control persons) all values in percent

Eigenwert number	DEP	SCH	CNT
1	28.3	18.2	31.6
2	11.5	12.8	13.3
3	7.3	10.2	10.6
4	6.9	9.5	8.6
5	5.8	6.1	6.5
6	4.8	5.5	6.1
7	4.5	4.9	5.4
8	3.7	4.0	
9	3.4		
Sum of explained variance	76.4	71.3	82.1

DEP = depressive persons; SCH = schizophrenic persons; CNT = control persons

Results of the Factorial Analyses

These results will only be outlined here, and are provided in full detail elsewhere (Guenther and Guenther in press).

We performed Bartlett tests on the total data pools (not over special subgroups of patients because of heterogeneity of variances, in order to provide the mathematical basis for the conjunction of the three pools (Clauss and Ebner 1974).

We calculated main axis factorial analyses, orthogonally, exit criterion Eigenwerte above 1.0. Thus, we used the same methodology as in Günther et al. (1986a) on SCH ($n = 57$) and normals ($n = 40$; independently calculated analyses) and will compare the present results of the factorial analysis on depressives, with the results from 1986a.

Table 6 reports the factorial structures and explained variances for depressives (DEP), SCH and CNT. First it should be noted, that the explained variance (using an identical exit criterion) was similar in all three subject groups (DEP 65.4%; SCH 71.3; CNT 82.1%).

The number of extractable factors was also very similar: 7 factors in CNT, 8 in SCH and 9 in DEP. Briefly, factor 2 seemed to correspond to the Fleishman-factor (Fleishman 1954; Schoppe 1974; Günther and Gruber 1983) speed of precise arm-hand movements, factor 3 to arm-hand steadiness, 4 to precision of arm-hand movements and 5 to wrist-finger speed. Factor 6, only partly homogeneous, seemed to be represented by the LOS items and 7 by the LNB. The last factors 8 (in SCH and DEP) and 9 (in DEP) explained only 4% of the total variance. Concerning the first factor which explained most of the variance in all groups, we attempted to test the hypothesis, whether its factorial structure, is similar in the 3 groups, using the following statistical methods: the correlation coefficient of the loading coefficients on the first orthogonal factor, which explained by far the maximal variance (DEP 28.3%; SCH 18.2%; CNT 31.6%) was 0.87 ($df = 24$, P below 0.01) for DEP/SCH and 0.61 ($df = 21$, P below 0.01) for DEP/CNT (for SCH/CNT this coefficient was 0.67, $df = 21$, P below 0.01). This can be seen as the first statistical indication of a significant similarity of the factorial structure of the first factor in all 3 groups. We compared the configuration of variables, which load highly (above 0.30) on the first factor in all groups SCH, DEP and CNT, and found that each variable loading highly on factor 1 for SCH and CNT did so for DEP. In contrast, only 7 of 12 variables not loading highly on factor 1 shwoed this quality in DEP. This configuration similarity was highly significant (e.g. test of Fisher- Yates, phi 0.50, $P < 0.01$). In conclusion, we obtained evidence from our factorial analyses, that the first unrotated factor loads highly on a wide variety of motor abilities, similarly in DEP, SCH and CNT. This factor, which explains most of the variance in all 3 groups, can be interpreted as general motor ability factor; and the item structure on this first factor is very similar in DEP, SCH and CNT groups.

Finally, it was tested, whether the motor impairments in acute ED patients show significant covariation with this general motor ability factor (Table 6). As criterion for statistical testing, we used for every motor item the algorithm $z = M(D) - M(C)/S$, where M(D) and M(C) are the means of this motor item in DEP and CNT, S the (balanced) mean of the standard deviations (thus using a criterion which is analogous to the t-value; Clauss and Ebner 1974). The correlation yielded to 0.53 ($df = 21$, P below 0.01), i.e. the motor impairments of ED persons, in comparison to controls, are significantly more marked on the variables loading highly on the first factor (only 2 of 12 low loading, but 8 of 11 high loading variables had increased z-values in DEP). This indicates that DEP show inpaired motor performance predominantly in tasks, which load highly in the first factor suggesting impairments in general motor ability in these patients.

Discussion

A wide variety of motor symptoms in depressed patients has been reported in the literature yielding evidence that motor symptoms may be found in differ-

ent motor subsystems, which seems to be suggestive of a possible general motor disability in these persons, and that several motor symptoms seem to exist independently of antipsychotic medication, suggesting a disease-related rather than a drug-related syndrome. This is supported further by the evidence of various motor symptoms reported in affective psychoses long before antipsychotic drugs were introduced into the treatment of these persons. However, "it may well be, that all of these psychomotor abnormalities are highly correlated and perhaps even due to the same central malfunction" (Greden and Carroll 1981).

In congruence with this statement we attempted in our series on psychomotor disturbances in psychiatric patients (Günther and Gruber 1983): to check on this wide variety of motor symptoms using a test battery, which integrates many dimensions of motor symptoms described in the literature (e.g. fine and gross movements of the extremities, complex motor coordination and planning, bucco-linguo-facial movements), (2) to check on evidence of a general motor disability, which might be responsible for the variety of such symptoms, (3) to check on the course of such motor dysfunction and the relationship to drug treatment, especially whether some particular motor dysfunction or a full-blown PMS exists independently from antipsychotic medication in ED patients and (4) to check on concomitants of cerebral dysfunction during disturbed motor behaviour.

In this study, we reestablished evidence for a PMS in acute ED patients consisting of disturbances of lip and tongue movements, fine and gross movements of the dominant right hand, and the complex motor coordination of the extremities. Such a PMS had been found in ED patients and schizophrenics (Günther and Gruber 1983; Günther et al. 1986a). There is no evidence, that a PMS is secondary to antipsychotic medication. This seems consistent with evidence in the literature, reporting motor dysfunction in SCH and ED subjects long before antipsychotic drugs were available.

Furthermore, there was no evidence that motor symptoms as assessed by our test battery, are significantly different in diagnostic subgroups (monopolar vs bipolar in ED patients, or diagnostic SCH subtypes).

However, the PMS seemed to persist in SCH irrespective of the actual psychopathology, even in the symptom free INT patients (signs of a trait marker). In contrast, it seemed to show a clear (though prolonged) remission in hospitalised ED patients towards the end of the inpatient period and disappeared completely in INT patients (signs of a state marker).

Both the results of our psychometric studies and the factorial analyses indicated a similar PMS both in SCH and ED patients. The factorial structure seemed to be normal in all psychotic patients and the performance deficits maximal on items which load highly on a general motor ability factor. This supports speculations on a similar aetiology for the variability of motor symptoms, as already hypothesized by Greden and Carroll (1981) and raises suspicions towards a diffuse rather than localised cerebral dysfunction accounting for such diffuse motor abnormalities. It is beyond the scope of this paper to detail the preliminary evidence on cerebral (motor and other) dysfunction in endogenous psychosis (extensive review e.g. Flor-Henry and Gruzelier 1983; Gruzelier 1985; Takahashi et al. 1987). Signs of a widespread bilateral cerebral dysfunction seem indeed to exist both in neuroleptic-treated SCH (measured be EEG mapping: Guenther and Breitling 1985; Guenther et al. 1986b, 1988) and untreated patients (measured by regional cerebral blood flow (rCBF) measurements, Guenther et al. 1986c; Gur et al. 1985). Surprisingly (drug-treated) ED patients at the beginning and the end of their hospital treatment show abnormal cerebral motor activation patterns similar to subgroups of schizophrenics (rCBF measurements, Günther et al. 1986c). The continuation of our work using EEG mapping on motor dysfunction in untreated SCH and affective psychotic persons, including the effects of medication on patterns of abnormal cerebral functioning shall be next part of this series on motor dysfunction in psychiatric patients.

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Received July 1, 1987