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ORIGINAL PAPER

W. Günther · P. Streck † · N. Müller · G. R. Mair
E. Kalischek · W. Bender · R. Günther

Psychomotor disturbances in psychiatric patients as a possible basis for new attempts at differential diagnosis and therapy

Part VI. Evaluation of psychomotor training programs in schizophrenic patients

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Abstract Parts I–III of this series established signs of disturbed motor performance – the "psychotic motor syndrome" (PMS) – in schizophrenic and endogenous depressed patients, which was not found in neurotic/reactive depressed nor healthy persons. Part IV yielded EEG signs of concomitant brain dysfunction in these patients, which were demonstrated by other (SPECT/PET) neuroimaging methods also. In part V we engaged in the development of motor-training programs applied both actively and mentally, using the PMS as target syndrome in depressed patients. We hypothesized that motor training would not only improve disturbed motor behaviour, but ameliorate other symptoms of psychopathology additionally, which was supported for these patients. Part VI is the final paper of this series demonstrating favourable results of our motor-training programs in 96 schizophrenic inpatients in two separate investigations. A general discussion to the whole series attempts to link motor symptoms to neuroimaging findings of brain dysfunction during motor challenge and to modern three- and four-factor models of schizophrenic symptomatology. A final version of our complete training programs will be published as an appendix to this paper along with information regarding the abbreviated test battery.

Key words Motor-training programs · Motor dysfunction in psychosis · Additional treatment in psychosis · Motor brain dysfunction

W. Günther (✉) · P. Streck † · N. Müller · G. R. Mair
E. Kalischek
Psychiatrische Universitätsklinik, Nussbaumstr. 7,
D-80336 München, Germany

W. Günther
Psychiatrische Klinik St. Getreu, Bamberg, Germany

W. Bender
Bezirkskrankenhaus, Haar/München, Germany

R. Günther
Psychologisches Institut, Tübingen, Germany

Introduction

The motor symptoms of psychopathology have been investigated both clinically and experimentally as long as Kahlbaum's concept of catatonia (1874) and the early interest in the experimental investigation of motor dysfunction by Kraepelin (1896), Wernicke (1900) and his pupil Kleist (1908). Decades before the introduction of neuroleptics into the treatment of psychiatric patients signs of disturbed psychomotor performance were described predominantly in schizophrenic and endogenous depressed patients, in contrast to normal persons or to patients with other psychiatric diagnoses (personality disorder, addiction, neurosis; e.g. Wulfeck 1941). Both voluntary and involuntary movements may be involved (Crider 1991; Manschreck et al. 1990) and motor symptoms may, at least in acute schizophrenic patients, be related to the severity of other psychopathology (Vrtunski et al. 1990), whereas in more chronic courses they may not (Schröder et al. 1992a; Martin et al. 1994).

In parts I–III of this series (Günther and Gruber 1983; Guenther et al. 1986; Guenther et al. 1988) we reported findings of our own psychometric research establishing a psychotic motor syndrome (PMS) in schizophrenic and endogenous depressed patients (diagnosed according to ICD-8 and ICD-9), which occurred in drug-naive patients independently of previous treatment with psychoactive drugs. It consisted of disturbances of fine movements of the tongue and lips, the fingers and hands, and a perturbation of development and execution of complex motor sequences. The motor test battery used by our group was submitted to factorial analyses (Guenther et al. 1986) and found to measure predominantly a general motor disability factor, which separated maximally schizophrenic/endogenous depressed from neurotic-reactive depressed and healthy persons. An abbreviated version of this test battery was developed including only the items with maximal loading on this general motor factor (detailed in the Appendix).

In a series of neuroimaging studies using motor activation as a challenge paradigm we established signs of con-

comitant brain dysfunction using EEG mapping (Guenther et al. 1989; Günther et al. 1993a), single photon emission computerized tomography (SPECT) and magnetic resonance imaging (MRI; Guenther et al. 1991), and positron emission tomography (PET; Günther et al. 1994). Psychomotor activation tasks have been used by other groups also exploring brain function in normal persons (Derambure et al. 1993), and by our group in other clinical samples as Gilles de la Tourette patients (Müller et al. 1992) or persons with dementia of the Alzheimer type (Günther et al. 1993b).

The signs of brain dysfunction yielded by these neuroimaging methods can only be outlined here for schizophrenics. Patients with predominant negative symptomatology showed an inability to increase focal cerebral activity (necessary presumably in order to keep up with increased demands of a complex stimulation), which was accompanied by poorer performance during such tasks (as shown by our EEG mapping, SPECT and PET studies).

In contrast, positive schizophrenics showed nearly normal activation (EEG mapping) or even a diffuse hyperactivation (SPECT) on manumotor stimulation tasks. An overview and speculative hypotheses on the underlying pathophysiology of schizophrenia are provided in our 1991 paper. Similar conclusions concerning insufficient cerebral laterality of motor functions were drawn by Gorynia et al. (1994) from the investigation of (unmedicated) children and adolescents with schizophrenic psychosis, predominantly of the catatonic subtype, and patients with motility psychosis (according to Leonhard 1986). The authors hypothesized that the observed instability of cerebral laterality in these patients may possibly be favored by prenatal and early postnatal influences as suggested by other evidence also (e.g. Okada et al. 1994; overview e.g. Crow 1993).

However, apart from such a pathophysiology aspect of the investigation of motor symptoms in endogenous psychoses, we attempted also to use the PMS as a target syndrome for treatment programs, and additional therapy for patients suffering from affective and schizophrenic psychosis. We hypothesized that such training would not only improve motor functional circuits, but other clinical symptoms of brain dysfunction also. We included modern developments of complex motor-training programs, such as alternating active and mental tasks, in order to obtain better efficacy, as known from sports-psychology studies (e.g. Ulich 1973; Günther 1980). However, we had to develop several versions of such a training program in order to make it suitable for use in psychiatric patients. We reported (Günther et al. 1992) favourable results in ICD-9 endogenous depressed patients of the monopolar (296.1) and bipolar (296.3) subtype. The results for schizophrenic patients are presented in this last paper of the series.

Table 1 Personal variables, diagnoses and psychopathology scores (pretreatment ratings), study I ($n = 32$). UPT unspecific physiotherapeutic treatment; AT active treatment programs; AMT active-mental treatment programs; Bfs "Befindlichkeitsskala", total score; HAMD Hamilton Depression Rating Scale scores, including subscores; BPRS Brief Psychiatric Rating Scale scores, including subscores; ANDP anxiety-depression; ANER anergia; THOT thought disorders; ACT Activation; HOST Hostility (BPRS subscales). Indicated are means and standard deviations (in brackets)

	UPT ($n = 9$)	AT ($n = 11$)	AMT ($n = 12$)
Personal variables			
Age (Mean in years)/range	32.1/17–53	31.2/17–46	35.0/23–49
Gender (m/f)	5/4	3/8	7/5
Handedness (r/l)	6/3	10/1	11/1
Diagnoses			
295.1	2	2	1
295.2	1		
295.3	3	5	6
295.6		1	4
295.7	3	3	1
Duration of disease (mean in years/range)	5.2/1–8	6.2/1–10	5.7/1–7
Start of motor treatment after admission (mean in days/range)	16.8/15–23	17.4/15–26	17.5/15–25
Psychopathology ratings			
Bfs	27.2 (14.0)	17.3 (12.0)	17.8 (9.5)
HAMD Retardation	1.8 (1.5)	1.8 (1.8)	1.7 (1.4)
HAMD Attention	1.6 (1.5)	1.5 (1.5)	0.7 (1.0)
HAMD Anxiety	2.8 (2.1)	2.8 (2.3)	2.9 (1.6)
HAMD Somatic score	3.3 (2.0)	2.6 (2.2)	2.6 (1.6)
HAMD Sum score	14.3 (5.8)	14.0 (5.8)	12.8 (4.3)
BPRS ANDP	12.4 (3.4)	9.8 (3.7)	9.8 (5.0)
BPRS ANER	10.8 (3.2)	10.5 (4.4)	13.3 (3.3)
BPRS THOT	9.7 (3.3)	9.3 (3.7)	10.4 (3.8)
BPRS ACT	8.9 (3.6)	7.6 (2.7)	9.3 (3.4)
BPRS HOST	8.6 (1.9)	7.9 (2.5)	7.3 (1.8)
BPRS Sum score	50.2 (9.1)	45.2 (9.5)	50.0 (10.0)

Remark to studies

In a validation/cross validation experimental design we performed two investigations in independent samples of schizophrenic patients. The results of the first investigation suggested that the application of our training programs in a purely active mode was not efficacious and inferior to the active-mental application. Therefore, in our study II, no such group was included. Both studies can only be outlined in this paper for space reasons; full details must be obtained in two (German language) doctoral theses (Kalischek 1995; Mair 1995).

The hypotheses tested were as follows:

1. The training of motor abilities exerts a positive influence on motor *and* psychopathology variables.

2. The efficacy of our motor-training programs is greater than an unspecific physiotherapeutic treatment of equal duration.

3. If there is such a clinical efficacy of our motor-training programs, there should be a further advantage of combined active-mental strategies as compared with a purely active motor treatment (The last hypothesis was not supported by our first study in schizophrenics and not carried over to study II).

Subjects and methods

Study I

The subjects were 32 inpatients of the Psychiatric University Hospital IdI, Munich. A clinical psychiatrist as well as a research psychiatrist (P.S.) reached independently of each other diagnoses of (ICD-9) schizophrenia including schizoaffective psychosis (295.7, ICD-9). Because our previous investigations (Günther and Gruber 1983; Guenther et al. 1986) had shown a PMS both in untreated and neuroleptic-treated schizophrenics in accordance with findings of other authors (Schröder et al. 1992a, 1993), and because we intended to work as closely as possible to normal clinical circumstances, drug treatment was allowed for all patients in the study. Both first-episode patients as well as those with exacerbations of chronic courses were admitted to the study (details cf. Table 1). Exclusion criteria were as follows:

1. Very acute symptoms of a schizophrenic psychosis with impairment to take contact and understand the instructions of our motor programs. This precondition yielded as a consequence that we started our training sessions not before 2 weeks after admission.

2. Age under 18 years or above 65 years.

3. Impaired intelligence (as screened by clinical examination only) preventing understanding the instructions.

4. Signs of organic brain disease (established by EEG, X-ray, CT or MRI as much as available).

5. Neurological diseases yielding motor symptoms.

6. Surgical or orthopaedical causes of motor disturbances.

7. Major disturbances of vision.

8. Insufficient understanding of the German language, which made understanding of the instructions doubtful.

A total of 32 patients fulfilling the above criteria and being consecutively admitted to the Psychiatric Hospital Munich during the index period were included in the study. They were randomly assigned to three treatment groups: (a) unspecific treatment, (b) active treatment by the training programs, and (c) active-mental application of them.

Table 1 shows the diagnoses, the personal and the psychopathology variables that were assessed following the methodology of previous work using the Hamilton Depression Scale (HAMD) (Hamilton 1960), the "Befindlichkeitsskala" (Bfs)/study I and "Paranoid-Depressivitäts-Skala" (PD-S)/study II (von Zerssen 1976), respectively, and the Brief Psychiatric Rating Scale (BPRS; Overall and Gorham 1976).

Statistical screening of all personal (except gender, handedness and diagnoses) and psychopathology variables was performed using the nonparametrical Kruskal-Wallis H-test (Claus and Ebner 1974, p 345ff). Analysis of variance was not performed, because the three groups were small and unequal ($n = 9$, $n = 11$, $n = 12$) *and* the Bartlett test indicated inhomogeneity of variances (cf. e.g. Bortz 1989, p. 347). For none of the 15 H values was the probability below 0.05, which does not support significant differences in the central tendencies in *any* variable. For the qualitative variables, gender and handedness, we performed χ^2-square tests, which were 2.72 and 2.98, respectively ($df = 2$; $P > 0.01$). Thus, we could reasonably assume that all three groups were comparable with regard to personal and psychopathology variables.

Design of study

We used for all three groups a test-intervention-test strategy. In every group eight treatment sessions, of 30 min duration each,

Table 2 Personal variables, diagnoses and psychopathology scores (pretreatment ratings), study II ($n = 64$). Indicated are means and standard deviations (in brackets)

	UPT	AMT
Personal variables		
Number	31	33
Age (mean in years/range)	31.8 18–59	35.0 20–56
Gender (m/f)	21/10	17/16
Handedness (r/l)	30/1	29/4
Diagnoses		
295.0	11	8
295.1	3	1
295.2		1
295.3	11	13
295.6	1	4
295.7	4	5
297.0	1	–
298.3		1
Duration of disease (mean in years/range)	4.8/1–7	5.5/1–8
Start of motor treatment after admission (mean in days/range)	16.3/14–21	17.4/14–25
PDS-P	13.9 (11.1)	15.4 (10.8)
PDS-D	16.7 (8.6)	20.1 (7.8)
PDS-K	10.6 (4.9)	9.5 (5.5)
BPRS ANDP	11.5 (4.2)	11.9 (4.4)
BPRS ANER	9.4 (4.0)	9.7 (4.1)
BPRS THOT	8.7 (4.9)	9.3 (5.0)
BPRS ACT	7.1 (3.3)	8.5 (3.4)
BPRS HOST	7.5 (3.5)	7.6 (3.1)
BPRS Sum score	44.2 (11.7)	46.9 (11.4)

NOTE: See Table 1 for abbreviations

were applied altogether with a frequency of three times weekly. In this naturalistic clinical approach, no attempts were made to control for the medication received during this treatment period and/or other intervening clinical variables. However, assuming that the amount of attention given to the patients in all three experimental groups would be a major source of variance, no "pure" clinical control group without such treatment sessions was included.

Study II

Subjects were 64 consecutively admitted inpatients of the Bezirkskrankenhaus Haar (a large state hospital near Munich). All diagnostic procedures were as detailed above for study I, except for the self-rating-scale BfS, which was replaced by the paranoid-depression scale (PDS; von Zerssen 1976). Table 2 shows the personal and diagnostic variables. Statistical screening of all steady personal (i.e. except gender, handedness, diagnoses) and psychopathology variables was performed using repetitive *t*-tests. After subsequent Bonferroni-Holm corrections of alpha niveau (e.g. Abt 1981), no significant differences were obtained indicating that the two groups were comparable in these variables before treatment intervention. Patients were randomly assigned to two treatment groups: (a) unspecific treatment, and (b) active-mental treatment by our training programs. The design of the study including the application of the treatment programs was identical to study I.

Results

Study I

Pretreatment findings

For the assessment of psychomotor variables, we used the (non-abbreviated) motor test battery of our previous investigations consisting of the "motorische Leistungsserie" (MLS; Schoppe 1974) the motor subtest of the Luria-Nebraska Neuropsychological Battery (LNB; Luria 1970; Golden et al. 1978), and the Lincoln-Oseretzky Motor Development Scale (LOS; Reinert 1966).

For space reasons we cannot report in detail the findings of the pretreatment measurements of the psychomotor variables in the three groups, which were comparable to our results reported in part I (1983, p. 198f). It has to be noted here, however, that statistical screening using the nonparametrical Kruskal-Wallis H-test (again, analysis of variance was not performed for reasons stated previously) yielded only 1 of 24 H values with a probability below 0.05. Because this is less than the number expected by

Table 3 Psychomotor changes (post-minus pretreatment measurements). The dimension in each variable is directed that positive values indicate improvements in posttreatment measurements. Indicated are means and standard deviations (in brackets)

	UPT	AT	AMT
Aiming			
Hits (*n*)	0.6 (1.7)	0.2 (2.4)	–0.8 (2.0)
Total duration (s)	10.0 (18.9)	12.8 (46.1)	16.8 (28.6)
Sticks long (*n*)	38.8 (66.0)	2.7 (94.9)	32.2 (79.7)
Sticks short (*n*)	111.4 (160.9)	–9.9 (116.8)	41.3 (127.0)
Errors (*n*)	36.2 (65.1)	7.1 (52.7)	7.3 (59.1)
Errors duration (s)	26.4 (64.1)	–1.6 (25.6)	27.9 (63.7)
Line pursuit			
Errors (*n*)	19.1 (22.4)	1.2 (20.2)	19.5 (34.7)
Errors duration (s)	19.2 (12.3)	–1.8 (18.8)	14.5 (31.1)
Total duration (s)	–7.6 (98.2)	24.6 (50.7)	23.0 (155.0)
Tapping			
First part (*n*)	2.8 (7.4)	4.6 (9.3)	5.5 (10.6)
Second part (*n*)	1.6 (7.3)	2.6 (9.2)	4.6 (9.0)
Total	1.2 (13.1)	7.1 (17.2)	31.4 (68.3)
Pursuit rotor			
First part: Error (*n*)	2.9 (6.2)	–1.0 (7.1)	0.4 (4.7)
Error duration (s)	–11.9 (38.3)	6.1 (39.7)	2.7 (15.6)
Second part: Error (*n*)	0.1 (4.1)	–1.3 (8.1)	–1.3 (5.8)
Error duration (s)	3.2 (47.2)	–6.2 (46.3)	6.2 (39.1)
Total error (*n*)	–2.8 (7.6)	–2.3 (12.5)	–0.8 (8.9)
Total error duration (s)	–8.7 (81.1)	–0.1 (81.9)	8.8 (49.9)
Luria Nebraska Battery			
Item 1– 4 (points)	0.8 (1.5)	0.5 (1.1)	1.1 (1.1)
Item 21–23 (points)	1.2 (1.1)	1.2 (1.1)	0.8 (1.1)
Item 32–33 (points)	0.7 (1.0)	–0.1 (0.8)	0.3 (0.9)
Item 36–47 (points)	0.3 (3.6)	1.9 (3.7)	0.3 (4.0)
Sum of points	4.6 (6.4)	5.0 (4.2)	3.6 (3.0)
Lincoln Oseretzky scale score	0.1 (1.7)	2.6 (2.5)	–0.8 (2.6)

chance, we could reasonably reject differences between the three groups in psychomotor variables before treatment.

Posttreatment minus pretreatment findings

Table 3 displays the motor variable changes (i.e. posttreatment minus pretreatment measurements – dimensionality is always directed so that positive values indicate psychomotor improvements in the second measurement). As can be seen from Table 3, all three groups improved their psychomotor performance (posttreatment minus pretreatment measurements) in most items. This is screened statistically in Table 4 following the methodology of our treatment study in depressed patients (Günther et al. 1992), which is summarized in the following. Because we intended in both investigations to work as closely as possible to "normal clinical conditions" (naturalistic design), we attempted also to choose a descriptive statistical screening suitable for this purpose. This means that we did not predominantly engage in establishing "significant" group differences, because these might be meaningless clinically for an individual patient. Instead, we attempted to assess the number of responders vs nonresponders in the three groups in those psychopathology and psychomotor variables that showed significant differences of posttreatment vs pretreatment measurements on *t*-test screening. Only in these variables did we consider an improvement of more than one standard deviation above mean of the initial measurement as responder.

Table 4 Proportion of patient responders (in the posttreatment measurement more than one SD above group mean of the pretreatment measurement)/nonresponders

	UPT	AT	AMT
Aiming			
Total duration			9/3*
Sticks short	7/2*		
Steadiness			
Errors-*(n)*	9/0**		
Errors duration	9/0**		
Line pursuit			
Errors-*(n)*	8/1**		9/3*
Errors duration	8/1**		
Tapping			
Second Part			8/4*
Total			9/3*
Pursuit rotor			
First part error-*(n)*	8/1**		
Luria Nebraska Battery			
Item 1–4			9/3*
Item 21–23	6/3*	7/4**	9/3*
Item 32–33	6/3*		
Sum of points	8/1**	9/2**	11/1**
Lincoln Oseretzky scale		11/0**	

NOTE: Only motor variables yielding significant group improvements (as screened by *t*-tests; * $P < 0.05$, ** $P < 0.01$) were submitted to this responder vs nonresponder analysis and included

Table 5 Rating scale score changes, posttreatment minus pretreatment measurements. Indicated are means and standard deviations (in bracket)

	UPT	AT	AMT
Bfs	1.6 (16.3)	1.6 (13.3)	4.3 (6.3)
HAMD Retardation	0.9 (0.9)	0.2 (1.7)	0.6 (1.2)
HAMD Attention	0.0 (2.7)	0.6 (1.4)	0.0 (0.7)
HAMD Anxiety	0.0 (2.6)	0.7 (2.2)	0.5 (1.6)
HAMD Somatic score	1.2 (2.1)	0.9 (2.0)	0.9 (1.1)
HAMD Sum score	3.9 (5.4)	3.8 (5.4)	2.6 (3.4)
BPRS ANDP	5.4 (3.7)	3.4 (2.5)	2.9 (3.1)
BPRS ANER	2.4 (2.7)	2.8 (2.4)	5.2 (2.8)
BPRS THOT	3.7 (4.2)	3.4 (3.1)	4.5 (3.3)
BPRS ACT	3.0 (2.4)	1.9 (2.1)	3.8 (3.0)
BPRS HOST	0.4 (7.4)	2.7 (3.5)	3.3 (2.1)
BPRS Sum score	19.4 (15.9)	14.3 (8.3)	19.8 (7.7)

NOTE: See Table 1 for abbreviations

As displayed in Table 4, both the unspecific physiotherapeutic treatment and the active-mental programs yielded in a similar number of items (9 vs 7) significant improvements in psychomotor variables (nonsignificant variables omitted in the table), whereas in the active treatment group there was only little improvement for the group as compared with the initial measurement. (This treatment was therefore omitted in study II.) Additionally, these group differences for the unspecific physiotherapeutic treatment and the active-mental programs seemed to be clinically meaningful as indicated by a high number of responders in both groups.

Psychopathology variable changes (posttreatment minus pretreatment measurements) are displayed in Table 5,

Table 6 Proportion of patient responders (in the posttreatment measurement more than one SD above group mean of the pretreatment measurement)/nonresponders

	UPT	AT	AMT
Bfs			10/2*
HAMD Retardation	6/3*		
HAMD Attention			
HAMD Anxiety			
HAMD Somatic score			7/5*
HAMD Sum Score	7/2*	8/3*	9/3*
BPRS ANDP	9/0**	9/2**	11/1**
BPRS ANER	6/3*	9/2**	11/1**
BPRS THOT	7/2*	9/2**	11/1**
BPRS ACT	8/1**	8/3*	11/1**
BPRS HOST		7/4*	11/1**
BPRS Sum score	9/0**	11/0**	12/0**

NOTE: Only motor variables yielding significant group improvements (as screened by *t*-tests; * $P < 0.05$, ** $P < 0.01$) were submitted to this responder vs nonresponder analysis and included

Table 7 Psychomotor changes (post- minus pretreatment measurements). The dimension in each variable is chosen so that positive values indicate improvements in posttreatment measurements Indicated are means and standard deviations (in brackets)

	UPT		AMT	
Steadiness				
Errors—*(n)*	7.1	(59.3)	4.4	(53.0)
Errors duration	−2.6	(43.1)	3.2	(29.8)
Sticks long	19.7	(46.5)	0.0	(63.2)
Sticks short	37.9	(120.8)	23.6	(108.8)
Pursuit rotor				
First part: Error-*(n)*	1.2	(5.7)	1.6	(5.6)
Error duration	3.8	(35.7)	−0.2	(42.7)
Second part: Error-*(n)*	0.6	(6.6)	−0.5	(4.9)
Error duration	1.4	(31.1)	5.2	(53.8)
Total error-*(n)*	1.2	(6.8)	−0.1	(6.3)
Total error duration	4.0	(32.3)	6.3	(38.4)
Luria Nebraska Battery				
Item 1	0.5	(0.6)	0.5	(0.6)
Item 2	0.5	(0.6)	0.5	(0.6)
Item 3	0.2	(0.7)	0.2	(0.7)
Item 4	0.0	(0.7)	0.0	(0.7)
Item 36	0.0	(0.9)	0.0	(0.9)
Item 37	0.3	(0.7)	0.3	(0.7)
Item 38	0.1	(0.7)	0.1	(0.7)
Item 39	0.9	(0.7)	0.0	(0.7)
Item 40	0.2	(0.8)	0.2	(0.8)
Item 41	0.8	(0.5)	0.0	(0.5)
Item 42	−0.1	(0.9)	−0.1	(0.9)
Item 43	0.2	(0.6)	0.2	(0.6)
Item 44	0.0	(0.7)	0.0	(0.7)
Item 45	0.1	(0.6)	0.1	(0.6)
Item 46	0.2	(0.9)	0.2	(0.9)
Item 47	0.2	(0.4)	0.2	(0.4)
Sum of points	2.4	(3.7)	2.4	(3.7)

and Table 6 provides similar information on the clinical significance of these changes.

As can be seen from Table 6, the only significant changes in the subjective (self-rating) scale Bfs were obtained by the active-mental treatment, whereas there appeared to be no obvious difference between the three groups in the other rating scales in the responder analysis.

In conclusion, whereas in psychomotor variables both unspecific and active motor training resulted in about equal improvements of psychomotor disturbances, for the more important psychopathology variables there remains a (slight) advantage for the active-mental training in the subjective feeling better. Because of these preliminary encouraging results, we decided to perform a cross-validation study in a new (bigger) sample of patients *and* another hospital. However, because the purely active motor-training programs were not as efficacious as the active-mental form, this application mode was not included in the cross-validation study.

Table 8 Proportion of patient responders (in the posttreatment measurement more than one SD above group mean of the pretreatment measurement)/nonresponders

	UPT	AMT
Sticks long	18/13*	
Sticks short		22/11**
Luria Nebraska Battery		
Item 1	19/12*	19/14*
Item 2	20/11*	20/13*
Item 37		23/10**
Item 39	20/11*	
Item 41	18/13*	
Item 43		23/10**
Item 44	19/12*	
Item 47		22/11**
Sum of points		19/14*

NOTE: Only motor variables yielding significant group improvements (as screened by *t*-tests; * $P < 0.05$, ** $P < 0.01$) were submitted to this responder vs nonresponder analysis and included

Study II

Several patients of study I had complained of the long duration of the psychomotor assessment procedures. Because the results of our factorial analyses (detailed in Günther et al. 1986, p. 306) indicated that the Lincoln-Oseretzky scale as well as the most variables of aiming, line pursuit and tapping of the MLS, did not load on the first general motor ability factor, these variables were not included in study II. Similarly, items 21–23 and items 32–33 of the LNB, which did not load on the general motor ability factor, were also excluded from the final version of our motor test battery. As detailed in the discussion to the factorial analyses in our 1986 publication, the general motor-ability factor was the one that separated schizophrenic and control persons maximally, whereas the omitted variables contributed little to this separation.

Again, for space reasons, we cannot report in detail the findings of the pretreatment measurements of the psychomotor variables in the two groups, which were again comparable to our results reported in parts I (1983) and II (1986). It is pointed out that statistical screening using the same methodology as outlined previously for the personal and psychopathology variables (repetitive *t*-tests and subsequent Bonferroni-Holm corrections of alpha niveau) again yielded no significant differences, indicating that the two groups were comparable in these variables.

Table 7 demonstrates the psychomotor changes (post-minus pretreatment measurements) after treatment interventions for both groups, both for the MLS and the LNB, along with the statistical evaluation of these changes in Table 8. As displayed in Table 8, there were no differences in the responder rate in the variables of the MLS, whereas there was a superiority of the active-mental training in the items of the more complex movement sequences of the LNB as indicated by the sum score.

Finally, Table 9 displays the changes in psychopathology variables along with the statistical screening of these

Table 9 Rating-scale score changes, posttreatment minus pretreatment measurements. Indicated are means and standard deviations (in brackets)

	UPT	AMT
PDS-P	4.5 (6.8)	4.3 (9.2)
PDS-D	3.0 (10.7)	5.3 (5.3)
PDS-K	–0.9 (4.6)	–0.8 (3.2)
BPRS ANDP	1.6 (6.2)	3.1 (4.3)
BPRS ANER	1.1 (5.1)	2.5 (4.0)
BPRS THOT	2.3 (3.4)	3.6 (4.0)
BPRS ACT	0.4 (4.1)	2.7 (3.4)
BPRS HOST	0.9 (3.4)	3.3 (2.8)
BPRS Sum score	5.6 (15.5)	15.1 (12.1)

NOTE: See Table 1 for abbreviations

Table 10 Proportion of patient responders (in the posttreatment measurement more than one SD above group mean of the pretreatment measurement)/nonresponders

	UPT	AMT
PDS-P	22/9*	23/10*
PDS-D	22/9*	26/7**
PDS-K		20/13*
BPRS ANDP		23/10*
BPRS ANER		20/13*
BPRS THOT	27/4**	29/4**
BPRS ACT		23/10*
BPRS HOST		27/6**
BPRS Sum Score	18/13*	31/2**

NOTE: Only motor variables yielding significant group improvements (as screened by *t*-tests; * $P < 0.05$, ** $P < 0.01$) were submitted to this responder vs nonresponder analysis and included

changes demonstrated in Table 10. Whereas in the subjective-scale PDS both treatment interventions yielded about equal improvements, there was again a distinct superiority of the active-mental treatment intervention in the objective rating scale BPRS, especially the sum score.

We consider this to be the major finding of these two studies, i.e. that active-mental motor-treatment programs can yield a distinct increase in the responder rate, not only in psychomotor variables, but also in objective and subjective psychopathology variables. This result is similar to favourable findings with the treatment programs in depressed patients, and seems to be of potential clinical interest, if supported further, not only by effectiveness studies (naturalistic clinical circumstances), but also by well-controlled efficacy experiments (e.g. Wilson 1995). It is discussed in more detail in the following section along with speculative hypotheses on possibly underlying brain dysfunctions and dimensions of psychopathology in schizophrenic disorders.

Discussion

Before discussing our results on the effectiveness of motor-training programs as an "add-on" therapy for schizophrenic patients, several methodological problems have to be pointed out. Psychomotor variables depend on many complex influences both in psychiatric and normal populations. However, in several investigations in our series of studies we attempted to rule out or at least control for some variables influencing psychomotor symptoms in psychiatric patients. Intelligence, handedness and variables of concentrative abilities did not significantly determine existence and degree of the symptoms of the PMS neither in depressed nor in schizophrenic patients (Günther and Gruber 1983).

In subsequent investigations we were able to further rule out age, configuration of psychopathology and subdiagnoses from influencing the psychomotor syndrome. Especially and most importantly, in our follow-up studies on schizophrenic (Guenther et al. 1986) and depressed (Guenther 1988) patients we established that the PMS seems to be independent from psychotropic medication, because both in the factorial analyses on depressed and schizophrenic patients, either drug-naive or treated with psychotropic substances, there was no difference in the constellation of the PMS. This means that the motor disturbances loading on a general motor-disability factor are responsible for most of the variance. Thus, although there is no doubt that lithium, neuroleptics and other psychotropic substances may produce motor disturbances on their own, they obviously do not load on the general motor ability and the PMS. This allowed us to use a shortened version of our motor test battery for clinical application (see Appendix).

Thus, there is additional justification to include medicated patients in our evaluation of additional treatment programs, because obviously the target syndrome seems to be independent of drug treatment. Additionally, in a naturalistic and realistic clinical approach to evaluate effectiveness (and/or efficacy) of treatment programs it seems virtually impossible to control prospectively e.g. life events and medication, just to name two possibly important variables, in experimental and control groups. Despite these inevitable biases of all naturalistic investigations, the relative time stability of the PMS syndrome – without intervention with motor treatment – may add reliability to our results. Both in schizophrenic (Günther et al. 1986) and depressed patients (Günther et al. 1988) there was evidence that psychotic motor symptoms existed in a prolonged manner after improvement (of psychopathology), thus displaying trait characteristics. Interestingly, direct intervention on the psychomotor target syndrome was able to improve the motor symptoms also, indicating that the PMS is, under direct training, less time-stable than without motor treatment.

However, another limitation in interpreting the results in this way must be pointed out. In their series of investigations on neurological soft signs, Schröder et al. (1992a, 1993) found a significant reduction of these symptoms in the course of both remitting schizophrenic and affective (depressed) patients, but not in chronic schizophrenics. Thus, although the relation of their motor soft signs to our "general motor disability" remains to be elucidated, the

possibility exists that different compositions of schizophrenic samples with regard to psychopathology (e.g. remitting vs chronic) may influence the course and stability of motor symptoms with or without motor treatment.

It is unknown in which manner *psycho*pathological and *motor*pathological improvement may interact, although recent neuropsychological results indicate that executive, memory and motor functions may be independent specific deficits (Sullivan et al. 1994) in schizophrenics. More studies are needed to elucidate further these complex issues.

With all these limitations, however, empirically and clinically our results may be of interest that motor-training programs as additional treatment in depressed and schizophrenic patients improved not only motor variables, but also psychopathology variables. Although motor therapies in clinical treatment settings are used in nearly all psychiatric hospitals, controlled studies to their effectiveness (not to speak of efficacy evaluations) are rare. Weber and coworkers (1993) studied subjective and objective psychopathology and body imaging in various psychiatric patients. They found that after 4 weeks of clinical motor training there were especially improvements in subjective psychopathological variables and body imaging in their patients. Although the authors did not evaluate psychomotor disturbance directly, their findings seem partially consistent with our results, because both in depressed and schizophrenic patients we found improvements in objective and subjective psychopathology using motor training as "add-on" therapy. In our group we evaluated additionally psychomotor performance in these patients and found improvements also.

Finally, the evaluation of concomitant signs of brain dysfunction during motor activation and their relations to dimensions of psychopathology are discussed.

Speculative hypotheses on pathophysiology/brain dysfunctions in subgroups of schizophrenic patients

Using psychomotor activation paradigms and 16- and/or 32-channel EEG mapping, we reported signs of brain dysfunction in schizophrenic patients. They indicated hypofunction in left hemisphere primary sensorimotor areas and right (compensatory?) hemisphere overactivation in patients displaying positive without (primary or secondary) negative symptoms (Guenther et al. 1989; Günther et al. 1993b).

Schizophrenic patients with predominant negative symptomatology, in contrast, displayed *bilateral* hypofunction during motor activation involving diffuse cortical areas including primary motor regions (Guenther et al. 1989). Interestingly, these "negative" schizophrenics showed a similar nonreactivity as found with the same activation paradigm and EEG mapping in mild-to-moderate Alzheimer patients (Günther et al. 1993b). Similarities between chronic and negative schizophrenics, as well as Alzheimer's patients, are further supported by findings using a different methodology. Royall et al. (1993) reported on similar signs of frontal-lobe brain damage in 40 young, chronically ill schizophrenics and demented elderly patients displaying signs of executive dyscontrol. Further evidence linking schizophrenia to dementia of the Alzheimer type is suggested by evidence of pathomorphology implicating predominantly the mediotemporal (entorhinal) cortex and the hippocampal areas (e.g. Goldsmith and Joyce 1995), although much remains to be elucidated about the etiology of such shared findings in different disorders. Using music-perception tasks as an additional challenge, similar findings to those described for motor tasks were obtained by EEG-mapping methods (Günther et al. 1993b). Furthermore, more reliable neuroimaging methods, such as SPECT (Guenther et al. 1991) and PET (Günther et al. 1994) supported signs of brain dysfunction in schizophrenia also. Schizophrenic patients with predominant positive symptomatology showed during a simple complex task of the right dominant hand a bilateral diffuse increase of the cerebral blood flow as measured by SPECT. In schizophrenic patients with predominant negative symptomatology, however, there was again (similar to the findings of the EEG-mapping studies) a complete nonreactivity upon such stimulation (Guenther et al. 1991). This latter finding was supported by our own recent PET study (Günther 1994), yielding again signs of nonreactivity of glucose uptake during a manumotor task.

In the SPECT study of 1991, we investigated additionally the area of the corpus callosum and found no difference in the schizophrenics (as a whole group) when compared with age- and gender-matched normal persons. This finding is in accordance with several reports in the literature (e.g. Colombo et al. 1994). However, when predominantly positive and negative schizophrenics were separated, significantly different midsagittal areas of the corpus callosum within the schizophrenics were observed. These findings lead us to finally postulate some speculative hypotheses on pathophysiology of schizophrenia:

Multiple predisposing influences (e.g. early neurodevelopmental disorders, intra-uterine maldevelopment, genetic influences, e.g. Bogerts 1995) may lead in some (later on schizophrenic) persons – long before the manifestation of first core symptoms – to very subtle signs of brain dysfunction, e.g. ambidexterity (Gorynia 1994) or various other "neurological soft signs" (Schröder et al. 1992a, 1993). Long-lasting compensatory efforts of the brain in an attempt to cope with these subtle brain dysfunctions may then result in a breakdown/disinhibition under multifactorial influences (e.g. life events, influences of hallucinogenic drugs or other toxic substances, other insults to the brain such as epileptic seizures or infection, endocrinopathy, etc.).

In such patients with manifest schizophrenia, those who have neuroimaging signs of disinhibited compensatory brain hyperactivation may be the patients with positive symptoms such as hallucinations and delusions. Because of unknown reasons (medication, severity of the disease, genetic or environmental influences), however, this disinhibited (originally compensatory?) hyperactiva-

tion may not be held up anymore in some of these patients and lead to a burn out of such pathological brain organization producing negative symptomatology. This change is detected by neuroimaging studies as a complete nonreactivity to various stimuli. Cerebral nonreactivity and lack of increase of focal neuronal activity to complex demands lead consecutively to an insufficient performance of difficult tasks. This may, of course, not only be seen in complex motor task, which was used in our series of investigations as "functional window to the brain", but also in other functional circuits: Impaired psychosocial functioning, loss of differentiation of personality, lack of concentration and goal-directed intention are further examples of brain nonreactivity found in chronic psychotic patients.

New atypical drug developments in combination with psychosocial and psychotherapeutic activating methods may increase the number of patients who can escape this "brain burn-out syndrome". Both our psychomotor treatment programs (as a psychotherapeutic approach in a broad sense) and our findings on pathophysiology in subgroups of schizophrenics attempt to contribute to clinical improvements for this unfortunate subgroup of patients.

Finally, apart from these speculations, it has to be admitted that only very little experimental evidence links psychopathology and neuroimaging/brain dysfunction in a sophisticated way in order to overcome e.g. the certainly simplifying positive-negative dimension. Schröder et al. (1992b) used psychopathological and morphological (CT) data in schizophrenic patients and separated by factorial analyses four subsyndromes: remitted, chronic delusional, chronic asthenic and chronic disorganized. Negative symptoms were shared by all three chronic subgroups, whereas CT alterations implicated different brain areas in the chronic subgroups and were not predominant in the remitting cluster. Similarly, Liddle and colleagues (1992) used PET for investigation of blood flow in schizophrenias and performed factorial analyses together with data of the psychopathology. They delineated three subsyndromes (psychomotor poverty, disorganization, reality distortion), which were linked to pathological brain perfusion in left and right dorsolateral prefrontal cortex, basal ganglia and medial temporal lobe, respectively.

Based on psychopathological data assessed by scales originally derived to measure negative (SANS) and positive (SAPS) dimensions, Peralta et al. (1994) proposed a four-syndrome model, which fitted best the psychopathology data of his schizophrenic sample using confirmatory factorial analysis. Thus, assessments of combinations of psychopathological and neuroimaging data may yield more sophisticated models of schizophrenic subsyndromes, which seem undoubtedly important for the clinical treatment and prediction of outcome for these patients, and may be a rewarding line for this type of research.

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Appendix

Motor Test Battery, final version:

Motorische Leistungsserie (MLS; Schoppe 1974) (available e.g. through Dr. G. Schuhfried, Hyrtlstrasse 45, A-2340 Mödling):
Steadiness (variables: Errors-number, Errors-duration)
Sticks long, sticks short (variables: total duration)
Pursuit rotor (variables: Error-number, Error-duration, both 1. and 2. part, Total Error-number, Total Error-duration)

Luria Nebraska Neuropsychological Battery (LNB; Golden, Purisch and Hammeke (1978) (available e.g. through University of Nebraska Press):
Motor part Items 1–4, 36–39, 40–47 (variables scores as defined in the manual).
These items 1–4 and 36–47 load similarly to all MLS items indicated above on a general motor ability factor. This general motor ability factor separates maximally endogenous depressed and schizophrenic from neurotic-reactive depressed and normal persons.

Motor Training Programs, version A (version B has similar tasks in another order):

Material: Chair, table, paper and pencil, matches, handle, stick.

General instruction: You sit in an upright position on a chair before a table. Your feet are standing firmly on the floor, the arms on the table. All the tasks you have learned before. At the signal you perform a task continuously at a speed of your choice until the stop signal is given. After each task you relax until the next begins.

Exercise 1: Hold your hands upwards over the table with extended arms. Open and close the hand to a first alternatively starting with the right hand. (Mental training additionally: Do not perform the movement in reality, but only in your mind. Be careful not to really move any part of your body).

Exercise 2: With your lips closed put your tongue between lower lip and teeth, then between upper lip and teeth. Then, press your tongue against the left cheek, then the right cheek.

Exercise 3: On the table in front of you there is a sheet of white paper. Take a pencil into your writing hand, holding it vertically at its tip with your fingers. Draw a big square first, and smaller ones inside, thus producing a kind of maze.

Exercise 4: Hold your hands over the table with your arms extended. First, with your writing hand bring the index finger to the thumb, then the middle, fourth and little finger. Reverse this order going back over fourth, middle and index finger. Then make the same movements with the other hand.

Exercise 5: Put 10 matches on the table parallel with a distance of about 1 cm from each other. Take the pencil with

your writing hand, holding it vertically at its tip with your fingers. Try to hit the intervals between the matches with the tip of the pencil from right to left; begin again at the right when you are ready.

Exercise 6: Stand upright beside of your chair. Put slowly the arms aside until they are in right angle to your body. Thumbs point to your front, palms downwards. Then close your eyes, go on tiptoes and bend your knees. Try to keep the balance. If you feel uncertain or sway, open your eyes and restart the exercise.

Exercise 7: Take the handle in your writing hand and begin to squeeze it regularly. Then you move your right arm from aside your body to a position rectangular to it. Continue to squeeze the handle while you are moving the arm. Then move the arm back to the body. Again, continue to squeeze the handle.

Exercise 8: On the table before you there is a sheet of white paper. Take a pencil into your writing hand, holding it vertically at its tip with your fingers. Draw a circle and a triangle inside of it, whose corners touch the triangle. Then draw again a circle. Draw a square inside it whose corners touch the circle. Continue with these two figures.

Exercise 9: Put your dominant arm rectangularly beside your body. Draw circles clockwise into the air with the hand. Continuing this movement, put the foot of that side with its tiptoes alternatively to one and the other side of your standing leg. Stop when you lose balance and restart the whole exercise.

Exercise 10: Draw with the tiptoes of your dominant foot a triangle on the floor. Touch the floor slightly with your toes at the corner of that triangle. Then draw with that same foot a circle in the air. Restart the two figures.

(Booklet with instructions and figures available from the author.)

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