Eur Arch Psychiatry Clin Neurosci (1992) 242:152–160

European Archives of Psychiatry and Clinical Neuroscience

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

V. Evaluation of Psychomotor Training Programs in Depressed Patients

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Received February 5, 1992

Summary. Parts I–III of this series used psychometric assessment of motor performance in psychiatric patients and indicated a "psychotic-motor syndrome" (PMS) in schizophrenic and affective psychoses, which was not found in "neurotic"/reactive or healthy persons. Part IV yielded signs of concomitant brain dysfunction in these patients, demonstrated by EEG mapping as well as other (SPECT/PET) neuroimaging methods. Apart from this "basic science" interest into the pathophysiology of endogenous psychoses we engaged in the development of motor training programs using the PMS as "target" syndrome. We hypothesized, that motor training would not only improve disturbed motor behaviour, but ameliorate other symptoms of psychopathology also. These assumptions were supported in the first two independent studies involving $n = 45$ and $n = 31$ ICD-9 mono- and/or bipolar endogenous depressed patients, respectively (the studies on schizophrenic patients being reported finally as part VI of this series, along with the final version of our modified motor test battery). Examples of the motor training programs are provided in this paper, although the final version of the complete programs will be published separately for space reasons and for better availability for routine clinical use.

Key words: Motor training programs – Motor dysfunction in psychosis-additional treatment in psychosis-motor brain dysfunction

Introduction

The motor aspect of psychological processes in psychiatric patients has received attention for a long time. Arising from Kahlbaum's concept of catatonia (1874), Wernicke stated 1900 that "there being nothing but motor and verbal expressions of thoughts and will, one may subsume the whole psychopathology in disturbances of motor behaviour". Following this probably too optimistic concept, many investigations have been performed to assess psychomotor disturbances in psychiatric patients since the beginning of the 20th century. Major findings yielded disturbed psychomotor performance in schizophrenic and depressive patients (of the "endogenous" subtype), in contrast to normal controls and persons with other psychiatric diagnoses (psychopathy, addiction, neuroses), as outlined in parts I–III of this series (1983, 1986a, 1988).

In these papers, we reported on psychometric research, establishing a "psychomotor syndrome" (PMS) in schizophrenic and endogenous depressed patients, which occurred in drug-naive patients independently of previous drug treatment. It consisted of disturbances of fine movements of the tongue and lips, of the fingers and hands, and a perturbation of development and execution of complex movement sequences.

Additionally, starting in 1985, we engaged in establishing correlated features of cerebral dysfunction during various psychomotor tasks using EEG mapping (part IV of the series 1989a, review of all our EEG mapping studies to this issue 1989b), regional cerebral blood flow (Günther et al. 1986b, 1991), nuclear magnetic resonance imaging (1989c, 1991) and positron emission tomography (1990, 1992) methods.

Virtually all these methods yielded signs of disturbed brain function during motor performance in schizophrenics, and also – with EEG and SPECT methods only – in ICD-9 endogenous depressed patients. Although details must be obtained in the original papers indicated above, major findings of "motor brain dysfunction" will be outlined here. – They consisted for schizo-

* Supported by Deutsche Forschungsgemeinschaft DFG Gu 207/1-1 –/5-1

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phrenic patients with predominant "negative" symptomatology in an inability to increase focal cerebral activity in order to keep up with demands of complex stimulation resulting in poorer performance during such tasks (shown by EEG mapping, SPECT and PET). In contrast, "positive" schizophrenics (untreated/drug-naive) showed "normal activation" (EEG mapping) up to "diffuse hyperactivation" (SPECT-MRI) on such manumotor stimulation tasks.

Apart from this "basic science" aspect related to the pathophysiology of endogenous psychoses we engaged in the attempt to use disturbed motor behaviour in these patients as a "target-syndrome" for the development and therapeutic evaluation of motor treatment programs.

We hypothesized, that such training with disturbed motor behaviour would not only improve the motor "functional circuits", but also other clinical manifestations of brain dysfunction in these patients – especially, of course, symptoms of psychopathology. Additionally, we hypothesized that modern developments of motor training programs should try to reach maximal efficacy of this therapeutic approach. In particular, this should involve recent findings of sports psychology, namely that for the training of complex motor sequences not only active motor training procedures should be involved, but also controlled mental imaginary of the sequences (e.g. Ulich 1973; Günther 1980). We postulated, that the combined use of active-mental motor training programs could optimize efficacy of this approach for use in psychiatric patients (especially those suffering from schizophrenic or affective psychosis).

We tested this hypothesis with specially designed motor training programs both in depressed and schizophrenic patients. This paper will report the findings on depressed patients along with examples of the training programs. Part VI will be the final paper of this series, reporting results of the training programs in schizophrenics along with examples of our motor test battery.

Both the complete motor training programs (separate publication) and the psychomotor test battery will be made available for clinical application in the future.

Material and Methods

Introductory Remark. In a validation/cross validation experimental design we performed two investigations of independent samples of depressive patients. The results of the first investigation yielded some modifications and improvements of our initial motor training programs which were than applied to the second sample of patients in part II of our studies. The improvement in the quality and acceptance of our training programs seemed to justify this deviation from a strict "test-retest" design. Both studies will be outlined in this paper, although, for space reasons, full details must be obtained in a (german language) doctoral thesis (Streck 1991).

The hypotheses to be tested in both studies were as follows:

1. The training of motor abilities exerts a positive influence on motor *and* psychopathological variables of endogenous depressed patients.
2. The efficacy of the motor training programs is greater than an "unspecific" physiotherapeutic treatment of equal duration.
3. If there is such a clinical efficacy by motor training programs, then there should be a further advantage of a combined "active-mental" strategy as compared with a purely active motor treatment.

Study I

Subjects were inpatients of the Psychiatric University Hospital ldI Munich. A clinical psychiatrist as well as the research psychiatrist had to establish independently of each other a diagnosis of ICD9-endogenous depression, monopolar (296.1) or bipolar (296.3) type. Since our previous investigation (1988) had shown no differences in motor performance for these patient groups in untreated versus treated patients and since we intended to work closely to "normal" clinical circumstances, drug treatment was allowed for all patients of the study. $N = 45$ patients fulfilling the above criteria and being consecutively admitted to this hospital were included. They were allocated by chance to three experimental groups, with $n = 15$ in each group. Table 1 shows the personal variables for the three experimental groups. There was no significant difference between age, sex or diagnoses between the three groups both on parametrical t-test and non-parametrical Mann-Whitney U-test (Bortz 1989) screening. All 45 patients were right-handed according to our (standard) criteria, 9 or 10 positive items on the shortened version of the Edinburgh-Scale (Oldfield 1971). Thus, the diagnostic procedures, the establishment of right-handedness and the exclusion of organic brain disease were analogous to our previous investigations.

Similarly, the assessment of psychopathological variables followed also the methodology of our previous work, using the Hamilton Depression Scale (HAMD) (Hamilton 1960), the Paranoid-Depressivitäts-Skala (PD-S) and the Befindlichkeitsskala (Bfs) (von Zerssen 1973) in all patients, before and after treatment intervention. The findings on these variables will be reported below in the results section.

For the assessment of psychomotor variables, we used the same motor test battery as in our previous investigations, consisting of the motorische Leistungsserie (MLS, Schoppe 1974), the motor subtest of the Luria-Nebraska-Neuropsychological-Battery (LNB, Luria 1966; Golden et al. 1978) and the Lincoln-Oseretzky-Motor-Development-Scale (LOS, Reinert 1966; Günther 1980). However, according to our findings from factorial analysis in depressive patients, both the LOS and the LNB were shorted and modified as described in 1988 (page 69f.).

Table 1. Personal variables and diagnoses

	AM	PT	CO
Age mean	50.7	50.6	45.3
Range	23–68	27–62	34–64
Sex (m/f)	3/12	7/8	2/13
Handedness (l./r.)	0/15	0/15	0/15
Diagnoses (296.1/.3)	12/3	13/2	12/3

AM = active-mental group, PT = physiotherapeutic group, CO = control group

Table 2. Results of the motor variables, pre-treatment measurement, in the 3 groups

	AM		PT		CO	
Aiming						
Errors-number	3.0	(7.0)	3.4	(6.4)	1.2	(1.5)
Errors-duration	2.6	(6.3)	2.6	(4.9)	1.8	(2.6)
Hits-number	19.3	(0.7)	19.5	(0.7)	18.1	(2.1)
Total duration	115.9	(27.7)	122.9	(34.7)	117.1	(55.7)
Sticks long	443.8	(80.8)	494.4	(107)	441.6	(108.7)
Steadiness						
Errors-number	34.3	(37.4)	36.5	(38.1)	37.4	(37.2)
Errors-duration	22.7	(33.9)	13.9	(13.9)	16.2	(28.1)
Line-pursuit						
Errors-number	67.4	(37.8)	78.7	(42.5)	59.9	(21.1)
Errors-duration	44.1	(28.3)	52.5	(27.6)	39.6	(17.0)
Total duration	181.7	(117.9)	195.1	(113.9)	166.8	(57.3)
Tapping						
1. part	91.1	(14.4)	89.4	(10.1)	88.2	(13.8)
2. part	84.7	(13.2)	81.8	(9.7)	87.7	(12.8)
Total number	175.9	(27.3)	171.2	(18.6)	175.3	(24.5)
Sticks short	520.7	(145.4)	572.2	(177.2)	483.4	(118.4)
Pursuit rotor						
1. part: error-number	15.1	(4.3)	11.2	(5.2)	15.3	(4.1)
error-duration	77.1	(33.8)	91.5	(51.2)	72.9	(23.3)
2. part: error-number	13.9	(3.9)	12.9	(5.7)	14.9	(3.7)
error-duration	85.0	(40.7)	92.3	(37.4)	73.2	(31.4)
Luria Nebraska batterie						
Item 1– 4	2.7	(1.8)	3.1	(1.8)	2.9	(2.0)
Item 21–23	2.1	(2.0)	2.3	(1.7)	2.2	(1.8)
Item 32–33	0.8	(1.1)	1.1	(1.3)	0.9	(1.1)
Item 36–47	4.4	(2.7)	5.3	(3.6)	6.7	(3.9)
Sum of points	14.8	(9.2)	20.3	(11.8)	16.5	(10.6)
Lincoln-Oseretzky-scale	16.1	(4.2)	14.5	(4.2)	16.7	(4.0)

The results on these variables before and after treatment will also be reported in the results section.

Design of the Study. We used for all three groups a test-intervention-test strategy. One group (CO-"control group") was treated with conventional clinical strategies only, in order to control for the course of the illness under "normal" clinical conditions, especially antidepressive medication and psychotherapy (in a broad sense).

A second group (PT-"physiotherapeutic group") used "unspecific" motor activation group therapy of two physiotherapists of the hospital, in order to control for unspecific effects of motor training alone.

Finally, the experimental group (AM-"active-mental group") was treated with an active-mental version of our training programs. Some information on this program will be given in the following.

Psychomotor Training Program (Version of Study I)

This motor training program involved eight tasks, which were designed according to the tasks which were most poorely performed by depressive patients in our previous investigations (1983, 1988). Each item lasted for 90 seconds followed by a 60 seconds interval. The whole treatment session in all groups lasted about 30 minutes; all 8 treatment sessions were applied during maximally 3 weeks of clinical treatment. All treatment instructions were standardized and provided by a type recorder. We followed in the experimental group an alternating procedure of active and mental performance of the tasks, always starting with the active task.

All tasks were performed by the subjects sitting on a chair with a table before them. The feet were aligned parallel on the floor, the hands and forearms lying relaxedly on the table. As an example we provide here only the instructions for item one and two (full details of the *final* – study II – training program, along with instructions to the motor test battery will be provided on an separate publication which may be used for distribution among interested patients).

Exercise 1. Hold your hands with straight arms out frontally before you above the table. Now open your right

hand until the fingers are in line with the arm and close the left hand to a tight fist simultaneously. Then, close the right hand to a tight fist, while you open the left hand until the fingers are straightly aligned with the arm. Continue with these simultaneous movements of both hands until the signal arises.

Mental version: Same instruction as above, modified by: You perform this activity now only mentally. Please watch carefully that, although you perform these movements in your imagination, you don't perform it really.

Exercise 2. Take the box of matches which lies on the table in front of you and let the matches fall out of the box onto the table. Put the box to the left of the matches. Take one match with the right hand and put it into the box, then the next one with the left hand. Continue alternatively with both hands until the signal arises.

Mental instruction as above for exercise 1.

For space reasons exercise 3 to 12 are not detailed here (Streck 1991, pp 30–34). Briefly we can indicate only that exercise 3 involves the lips and the tongue, exercise 4 stimultaneous movements of hands and feet, exercise 5 drawing figures with the dominant right hand, exercise 6 building up figures with matches, exercise 7 complex movements of opposition with the dominant and non dominant hand, exercise 8 alternative move-

Table 3. Self-rating scale scores, pretreatment measurements

	AM	PT	CO
Bfs	29.8 (14.8)	27.1 (14.1)	41.4 (9.3)
PD-S (P)	6.8 (7.5)	6.7 (10.2)	9.2 (4.3)
PD-S (D)	16.4 (9.5)	21.0 (8.2)	26.9 (9.6)
PD-S (K)	11.7 (5.6)	9.6 (5.5)	8.2 (4.9)

PD-S = Paranoid-Depression-Scale, (P) = Paranoid-score, (D) = Depression-score, (K) = Kontrollwert (control-score)

Table 4. Hamilton-Depression-Rating-Scale (HAMD) Scores, pretreatment measurements

	AM	PT	CO
HAMD	27.4 (11.0)	30.7 (10.1)	28.5 (9.4)

Table 5. Psychomotor changes (post- minus pretreatment measurements). The dimension in each variables is chosen so that positive values indicate improvements in post-treatment measurements

	AM	PT	CO
Aiming			
Errors-number	1.5 (7.1)	−0.6 (4.7)	−0.1 (2.2)
Errors-duration	1.8 (6.3)	−0.6 (1.9)	−0.9 (4.3)
Hits-number	−0.9 (2.5)	−0.9 (2.6)	2.3 (4.7)
Total duration	14.9 (21.8)	8.0 (18.9)	12.1 (33.3)
Sticks long	−3.1 (63.6)	42.0 (98.6)	0.6 (73.3)
Steadiness			
Errors-number	10.7 (25.3)	3.0 (21.9)	1.3 (32.1)
Errors-duration	10.9 (30.6)	−4.7 (23.1)	4.6 (26.7)
Line-pursuit			
Errors-number	−3.9 (45.0)	1.8 (39.4)	−4.3 (25.2)
Errors-duration	−5.6 (28.7)	0.6 (37.8)	−5.0 (19.8)
Total duration	2.5 (108.7)	15.5 (55.6)	2.3 (41.5)
Tapping			
1. part	5.9 (18.7)	−2.9 (13.5)	−3.5 (9.8)
2. part	12.8 (49.4)	−0.1 (8.3)	−4.1 (9.2)
Total number	18.7 (67.7)	−3.1 (19.8)	−7.5 (15.3)
Sticks short	−2.9 (58.6)	22.6 (153)	−11.0 (117.2)
Pursuit rotor			
1. part: error-number	1.3 (4.2)	−1.7 (3.7)	0.4 (5.5)
error-duration	−7.9 (20.7)	−0.9 (31.0)	−0.3 (12.4)
2. part: error-number	0.1 (3.5)	0.8 (4.0)	0.9 (4.3)
error-duration	−4.3 (20.0)	1.3 (20.4)	−8.5 (21.5)
Luria Nebraska batterie			
Item 1– 4	1.5 (1.5)	1.3 (1.4)	0.7 (1.4)
Item 21–23	1.8 (1.6)	1.0 (1.4)	0.3 (1.2)
Item 32–33	0.3 (0.5)	0.2 (1.2)	0.1 (1.0)
Item 36–47	0.5 (3.0)	−0.1 (3.1)	−0.1 (3.4)
Sum of points	4.3 (5.2)	4.3 (4.6)	−1.1 (5.1)
Lincoln-Oseretzky-scale	1.7 (2.2)	1.2 (2.2)	−1.4 (6.2)

ments of both hands, exercise 9 sequential movements of the dominant hand using matches and a pencil, exercise 10 continuous circular movements with the dominant hand using a ball and a wooden stick, exercise 11 balancing the body on tiptoes and with flexed knees, exercise 12 balancing a wooden stick with the dominant right hand.

Results

Table 2 displays the results of the (pretreatment) motor performance, whereas the psychopathological variables are displayed in Tables 3 and 4.

Respective statistical screening with H-test (Chi^2-tests for nominally-scaled variables) *and* subsequent Bonferroni corrections did not yield significant differences in *any* variable.

Motor variables *changes* (posttreatment minus pretreatment measurements; the dimensionality is always directed that positive values indicate improvements in the second measurement) are displayed in Tables 5 and 6.

Table 5 shows the results of psychomotor variables, post- minus pre-measurements for the 3 groups and Table 6 shows the statistical significance for the changes in psychomotor variables as compared to pretreatment measurements. As can be seen from this table both experimental groups showed more improvements in motor variables than the control group, which did not receive any motor treatment. Psychopathology variables/changes (posttreatment measurements) are displayed in Tables 7–10.

Table 7 shows the results of Bfs and PD-S changes, posttreatment minus pretreatment and Table 8 the significance of these changes. Table 9 shows analogously the changes for the Hamilton Depression Scale and Table 10 the significance of these changes. As can be seen in the psychopathology tables all three groups improved both in selfrating-scales and Hamilton Depression Scale in the posttreatment versus pretreatment measurement. Whereas in the selfrating-scales there are no differences, we found a tendency to a greater improvement in the absolute ratings on the Hamilton-Scores in the active-mental and physiotherapeutic group which would be a very interesting result and was therefore reinvestigated in a cross-validation study, to be reported in the following.

Table 6. Proportion of patients with significant improvements in motor variables (*t*-test and subsequent chi-square-tests)

	AM	PT	CO
Aiming			
Total duration	10/5*	11/4*	
Sticks long		10/5*	
Steadiness			
Errors-duration	11/4*		
Tapping			
1. part	11/4*		
Total number			4/11*
Pursuit rotor			
1. part: error-number		3/11*	
2. part: error-duration		9/6*	
Luria Nebraska batterie			
Item 1– 4	8/0**	7/1*	7/1*
Item 21–23	8/0**	5/1*	
Item 32–33	4/0*		
Sum of points	12/1**	13/2**	
Lincoln-Oseretzky-scale	9/0**	8/3*	

Table 7. Self-rating scale score changes, post- minus pretreatment measurements

	AM	PT	CO
Bfs	7.4 (15.0)	8.5 (9.6)	6.9 (11.8)
PD-S (P)	−0.8 (8.6)	−0.3 (6.1)	3.5 (6.8)
PD-S (D)	1.6 (11.1)	3.4 (10.2)	6.3 (11.0)
PD-S (K)	0.5 (6.6)	−0.4 (8.4)	0.4 (3.5)

PD-S = Paranoid-Depression-Scale, (P) = Paranoid-score, (D) = Depression-score, (K) = Kontrollwert (control-score)

Table 8. Proportion of patients with significant improvements in self-rating scale variables (U-tests and subsequent chi-square-tests)

	AM	PT	CO
Bfs	9/3*	12/2*	11/4*
PD-S (P)			11/3*
PD-S (D)			12/2*

Table 9. Hamilton-Depression-Rating-Scale score changes (post-minus pretreatment)

	AM	PT	CO
HAMD	17.7 (10.8)	11.9 (10.1)	7.5 (7.1)

Table 10. Proportion of patients with significant improvements in HAMD rating scores (U-tests and subsequent chi-square-tests)

	AM	PT	CO
HAMD	14/0***	12/2***	14/1***

Study II

Since in the first investigation in nearly all motor and several psychopathology variables there was a clear advantage for both motor training groups as compared with the purely clinically treated group, we included into this cross validation study no such control group. Thus in study II, the group AM comprised 15 patients and the group PT 16, which is further detailed on Table 11, along with the medication in both groups at the beginning of

Table 11. Personal variables, diagnoses and medication at the beginning of the investigation, study II

	AM	PT
Age (mean)	50.7	36.6
Age (range)	22–75	19–63
Sex (m/f)	2/13	1/15
Handedness (l./b./r.)	0/1/14	2/0/14
Diagnoses (296.1/.3)	12/3	13/3
Tricyclic antidepressants (amitriptyline, -oxid, imipramine, doxepine, dibenzepine, clomipramine)	9/15	15/16
Tetracyclic antidepressants (mianserine, maprotiline)	4/15	0/16
Monoamineoxidyseinhibitors (tranylcypromine/or combination with trifluoperazine)	2/15	2/16
Aminprecursor (oxitriptan)	1/15	0/16
High potent neuroleptics (haloperidol, flupentixol, sulpirid)	7/15	8/16
Middle potent neuroleptics (thioridazine)	1/15	0/16
Low potent neuroleptics (promethazine, levomepromazine)	1/15	1/16
Benzodiazepines (lorazepam, diazepam, flurazepam, lormetazepam, dipotassium-chlordiazepoxid)	5/15	5/16
Lithium salts (carbonate)	2/15	3/16
Carbamazepine	0/15	2/16
Internal medicine drugs	6/15	8/16

AM = active-mental group, PT = physiotherapeutic group

the treatment. Although there were no obvious medication differences in both groups, it should be noted, however, that we performed a "naturalistic" clinical study as already stated for study I above. This means that there was no possibility to exclude drugs, so that during our treatment sessions the medication may not have been constant. However, this design seems suitable for evaluating additional (to conventional) treatment programs in a first approach study. Furthermore, there was no statistical difference in any of the personal variables between the two groups on *t*-test screening, although it should be noted, that two left-handed patients were included and that the AM group was older (although this was not statistically significant). All other inclusion criteria and diagnostic procedures were as detailed above for study I.

However, the psychomotor training program (version II) was improved versus that of study I in the following respects:

1. The number of exercises was increased (20 tasks of 45 seconds duration each, separated all by a 30 second intervall, so that it was possible to use on subsequent days two alternate parts of the program in order to avoid habituation and boredom of the patients.
2. Active and mental training were performed on the same day, so that the difficult motivation for patients to engage in the mental training could be overcome.
3. The total duration of the training programs was decreased (from 30 minutes in study I to 15 in study II) in order to comply with the reduced concentration and performance capacities of our patients.

Finally we gave up the close relation of our training program items to the test battery items in order to improve the practicability of our treatment (which was not optimally accepted in the simple version of study I).

The 20 items of version II trained fine and gross motor movements of the dominant half of the body, the regions of tongue, mouth and lips and the complex coordination of the extremities. A handout of all 20 tasks along with the instructions was given to the patients so that they were able to perform independent training sessions on their own.

Both the active and the mental part of each of the 20 exercises lasted 45 seconds followed by a 30 seconds interval. Only half of the tasks were administered on one training day, both in active and mental instruction. Thus, the training sessions did not exceed 15 minutes in duration; all 8 sessions in experimental and control groups had to be performed within 3 weeks of clinical treatment. All training program tasks will be published in a separate publication; only two examples can be demonstrated here for space reasons.

Exercise 6. Please stand upright aside of your chair. Then put slowly your arms aside of your body until they are rectangular to both sides. The thumbs point in frontal, the palms in inferior direction. Close the eyes now

and go to your tiptoes, meanwhile somewhat bending the knees. Try now to keep the balance in this position until the signal arises. Should you lose balance you just retry. After the signal please sit back down again.

Mental instruction: Same instruction as above modified by: You perform this activity now only mentally. Please watch carefully that although you perform these movements in your imagination you don't perform it really.

Exercise 7. Take the handle in your writing hand and straighten up the arm horizontally in front of you. Doing this you press the handle regularly with the frequency of 1/s. Then you move the arm parallel aside of your body and subsequently rectangularly to the side. During the whole exercise you continue pressing the handle with a frequency of 1/s. When you have finished this complex movements you start again and continue until the signal arises.

Mental instruction: As above for exercise 6.

Results

Introductory remarks: Comparing the subjects of study II with those of study I the following deviations have to be noted: In the active-mental group of study II we had a higher average age and more females in both groups (corresponding to the distribution of gender in the patients of our hospital). As pointed out above there were 2 ambidextrous patients in the active-mental group and two lefthanders in the physiotherapy group (which differences, however, did not reach significance on statisticals screening). The medication in both studies included tricyclic antidepressants, atypical antidepressants, MAO inhibitors, neuroleptics, benzodiazepines, lithium and carbamazepin and internistic medication. In contrast to study I, all patients were inpatients at the Bezirkskrankenhaus Haar bei München and all ratings and treatments were performed there.

Table 12. Results of the motor variables (pretreatment measurements); shortened motor test battery of study II

	AM		PT	
Steadiness				
Errors-number	66.7	(93.5)	55.3	(44.3)
Errors-duration	41.5	(75.2)	22.3	(27.4)
Sticks long	468.0	(100.9)	464.8	(116.5)
Sticks short	490.7	(104.4)	475.5	(150.1)
Pursuit rotor				
1. part: error-number	14.4	(4.6)	12.4	(5.3)
error-duration	114.1	(36.6)	125.2	(25.7)
2. part: error-number	12.6	(4.5)	13.1	(6.4)
error-duration	107.2	(40.7)	122.4	(34.1)
Luria Nebraska batterie				
Item 1– 4	2.7	(2.2)	2.1	(1.2)
Item 36–47	8.4	(4.0)	5.7	(3.7)
Sum of points	11.1	(5.7)	7.8	(4.2)

Table 13. Self-rating scale scores (pretreatment measurements); study II

	AM		PT	
PD-S (P)	6.7	(9.1)	6.3	(8.4)
PD-S (D)	23.1	(12.0)	21.6	(11.4)
PD-S (K)	8.8	(6.0)	11.6	(5.1)

PD-S = Paranoid-Depression-Scale, (P) = Paranoid-score, (D) = Depression-score, (K) = Kontrollwert (control-score)

Table 14. Hamilton-Depression-Rating-Scale (HAMD) Scores, pretreatment measurements; study II

	AM		PT	
HAMD	18.5	(9.3)	15.2	(6.2)

Table 15. Psychomotor changes (post- minus pretreatment measurements). The dimension in each variables is chosen so that positive values indicate improvements in posttreatment measurements; study II

	AM		PT	
Steadiness				
Errors-number	12.9	(87.3)	5.5	(46.8)
Errors-duration	21.3	(70.9)	3.3	(30.2)
Sticks long	23.5	(86.2)	27.6	(66.4)
Sticks short	24.0	(114.7)	36.9	(51.0)
Pursuit rotor				
1. part: error-number	1.1	(6.5)	−1.5	(7.8)
error-duration	8.2	(25.4)	35.9	(42.8)
2. part: error-number	−3.5	(4.7)	−0.4	(9.1)
error-duration	4.1	(26.3)	33.1	(57.8)
Luria Nebraska batterie				
Item 1– 4	0.9	(1.5)	0.1	(1.4)
Item 36–47	2.0	(3.9)	−0.7	(3.9)
Sum of points	2.9	(4.2)	−0.6	(4.8)

Design of the Study. As in study I, care was taken that the training sessions took place at the same time of the day (late afternoon). 8 treatment sessions were performed in both groups, as in study I.

In contrast to our study I version of the training programs we had no complaints or drop outs from malacceptance of the training program. The motor test battery, finally, was shortened according to the results of our factorial analyses to items measuring a "general motor ability" factor, as detailed in 1988 (p. 69f.).

Table 12 displays the motor performance variables of the pretreatment measurements (shortened test battery), Tables 13 and 14 the pretreatment measurements of the psychopathology scales. Statistical screening (using U-tests) did not show significant differences for any of the variables.

Motor variables changes (posttreatment minus pretreatment measurements; positive values indicate improvement) are displayed in Tables 15 and 16. Table 15

Table 16. Proportion of patients with significant improvements in motor variables (*t*-test and subsequent chi-square-tests); study II

	AM	PT
Luria Nebraska batterie		
Item 1– 4	10/1*	
Item 36–47	8/6*	
Sum of points	8/4*	
Sticks long		13/3*
Sticks short		11/5*
Pursuit rotor		
1. part: error-duration		13/3*
2. part: error-number	3/11**	
error-duration		12/3*

Table 17. Self-rating scale score changes, post- minus pretreatment measurements

	AM	PT
PD-S (P)	3.5 (6.9)	0.4 (3.8)
PD-S (D)	5.1 (10.0)	4.0 (6.5)
PD-S (K)	−1.3 (3.9)	−1.0 (2.8)

PD-S = Paranoid-Depression-Scale, (P) = Paranoid-score, (D) = Depression-score, (K) = Kontrollwert (control-score); study II

Table 18. Proportion of patients with significant improvements in self-rating scale variables (U-tests and subsequent chi-square-tests); study II

	AM	PT
PD-S (P)	8/1*	
PD-S (D)	10/5*	13/2**

Table 19. Hamilton-Depression-Rating-Scale score changes (post-minus pretreatment); study II

	AM	PT
HAMD	7.20 (7.19)	2.63 (6.49)

Table 20. Proportion of patients with significant improvements in HAMD rating scores (U-tests and subsequent chi-square-tests); study II

	AM	PT
HAMD	11/3**	

shows the absolute differences for the motor variables along with the statistical screening, displayed in Table 16. As can be seen from these tables, both groups improved their motor performance without obvious differences between them. Psychopathology variables changes (post- minus pre-measurements) are displayed in Tables 17 to 20. Table 17 shows the changes of the selfrating scales in both groups along with the statistical screening (Table 18). Whereas both groups improved in their self-rating of depressive symptomatology significantly, it has to be pointed out, that only the active-mental group improved significantly on the paranoid scale.

The advantages of the active-mental group become even more marked, considering the results using the Hamilton Depression Rating Scale. Table 19 shows the absolute values of changes in both groups and Table 20 the statistical screening. Only in the active-mental group there was a significant improvement in Hamilton Depression Rating Scale scores.

In summary, in study II as already in study I there were signs of an advantage of the active-mental therapeutic group as compared to the physiotherapeutic group, especially in the improvement of psychopathological variables. Both groups were superior to no treatment (study I), whereas both treatment procedures seemed to be equally effective on the motor symptoms themselves.

The finding that active-mental treatment programs can improve significantly the psychopathology of depressive patients is of potential clincial interest and shall be discussed further in the following section.

Discussion

At the beginning it has to be pointed out, that psychomotor variables depend upon so many complex influences, that it is really impossible to control for them all. As has been shown in former parts of this series, however, we have been able to a rule out a greater influence of intelligence, handedness, and variables of concentrative abilities on the existence and degree of a PMS in psychotic patients. Therefore, we were able to omit these variables in this paper on our first results on treatment effects.

Furthermore, neither age (Gruber 1982), nor configuration of psychopathology were significantly correlated with the psychotic motor syndrome (PMS) (Rödel 1987). Finally, since the PMS existed independently of antidepressant and/or antipsychotic medication in our previous investigations (1983, 1986a, 1988) we were able to include drug-treated patients into this motor treatment study. This is especially important, since this represents the normal clinical situation, under which additional treatment programs should be evaluated. However, it should be noted that independence of the PMS from drug treatment does not imply necessarily, that the application and possible utility of our training programs is independent of this variable also. This issue cannot be addressed in this first step evaluation but needs own clinical study later.

With these precautions only we discuss several of our findings in the following section. Especially interesting appear the findings of this paper in respect to subjective ratings scales of depressive patients. In the first study we found for all three treatment groups a significant improvement of the scores of the Befindlichkeitsskala, which no differences between the groups. Similarly, in study II the scores of PD-S were significantly improved

in the posttreatment, compared with the pretreatment measurements. Again, there was no difference between the two treatment groups. This seems to indicate that subjectively all treatment efforts were accepted by the patients in a similar manner, giving rise, along with the course of the illness under clinical treatment, to equal subjective improvements of the patients.

Contrasting to this, somewhat unexpectedly, we found advantages in both studies, when the "objective" psychopathology was considered. In a first and possibly minor rewarding respect this concerned the variables of the psychotic motor syndrome. Both active-mental treatment programs and conventional physiotherapy as an additional treatment for depressive patients yielded in significant improvements of psychomotor variables in depressive patients. Both treatment strategies were in this respect better than no motor training, which seems less surprising though.

However, it is evident that the major goal of any treatment of psychiatric patients should result in improvements in their psychopathological symptoms.

Here, in an important clinical field, seems to be a real value of our treatment programs. In both studies the active-mental training programs yielded significantly greater improvements of the objective psychopathology in depressed patients (as measured by Hamilton Depression Rating Scale). This effect seems not to be secondary to improvements in the motor field, but an effect on its own and merits further evaluation in our opinion. The better acceptance of the final version of our treatment programs by psychiatric patients was a necessary precondition for such further clinical evaluation.

Finally, the separate publication of our motor training programs along with the results of there evaluation in *schizophrenic* patients is another necessary precondition for the greater clinical applicability of these training programs and will finish this series of papers, along with a final general discussion.

References

Bortz J (1989) Statistik für Sozialwissenschaftler. Springer, Berlin Heidelberg, pp 178–183

Golden CJ, Hammeke T, Purisch A (1978) Diagnostic validity of a standardized version of Luria's neuropsychological investigation. J Consult Clin Psychol 46(6):1258–1265

Gruber H (1982) Psychomotorische Untersuchungen bei schizophrenen Psychosen, endogenen und neurotischen Depressionen. Diss, München

Günther W (1980) Untersuchungen zur Wirksamkeit mentaler Trainingsverfahren bei grobmotorischen Bewegungsstörungen. Diss, Tübingen

Günther W, Gruber H (1983) Psychomotorische Störungen bei psychiatrischen Patienten als mögliche Grundlage neuer Ansätze in Differentialdiagnose und Therapie. I. Ergebnisse erster Untersuchungen an depressiven und schizophrenen Kranken. Arch Psychiatr Nervenkr 233:187–209

Günther W, Breitling D (1985) Predominant sensorimotor area left hemisphere dysfunction in schizophrenia measured by brain electrical activity mapping. Biol Psychiatry 20:515–532

Günther W, Günther R, Eich FX, Eben E (1986a) 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. Eur Arch Psychiatr Neurol Sci 235:301–308

Guenther W, Moser E, Müller-Spahn F, Oefele K v, Buell U, Hippius H (1986b) Pathological cerebral blood flow during motor function in schizophrenic and endogenous depressed patients. Biol Psychiatry 21:889–899

Guenther W, Guenther R, Streck P, Römig H, Rödel A (1988) 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. Eur Arch Psychiatr Neurol Sci 237:65–73

Guenther W, Streck P, Steinberg R, Guenther R, Raith L, Backmund M (1989a) Psychomotor disturbances in psychiatric patients as a possible basis for new attempts at differential diagnosis and therapy. IV: Brain dysfunction during motor activation measured by EEG mapping. Eur Arch Psychiatr Neurol Sci 239:194–209

Guenther W, Steinberg R, Petsch R, Streck P, Kugler J (1989b) EEG mapping in psychiatry: studies on type I/II schizophrenia using motor activation. In: Maurer K (ed) Topographic brain mapping of EEG and evoked potentials. Springer, Berlin Heidelberg, pp 438–450

Guenther W, Moser E, Petsch R, Brodie JD, Steinberg R, Streck P (1989c) Pathological cerebral blood flow and corpus callosum abnormalities in schizophrenia: relations to EEG mapping and PET data. Psych Res 29:453–455

Guenther W (1990) MRI-SPECT and PET-EEG findings on brain dysfuction in schizophrenia. In: Yamashita I, Toru M, Coppen AJ (eds) Clinical neuropharmacology, 13 suppl 2. Raven Press, New York, pp 170–171

Guenther W (1992) MRI-SPECT and PET-EEG findings on brain dysfunction in schizophrenia. Prog Neuropsychopharmacol & Biol Psychiatry 16:445–462

Guenther W, Petsch R, Steinberg R, Moser E, Streck P, Heller HJ, Kurtz G, Hippius H (1991) Brain dysfunction during motor activation and corpus callosum alterations in schizophrenia measured by cerebral blood flow and magnetic resonance imaging. Biol Psych 29:535–555

Hamilton M (1960) A rating scale for depression. J Neurol Neurosurg Psychiatr 23:56–62

Kahlbaum KL (1874) Die Katatonie oder das Spannungsirresein. Hirschnakl, Berlin

Luria AR (1966) Higher cognitive functions in man. New York

Oldfield RC (1971) The assessment and analysis of handedness: the Edinburgh inventory. Neuropsychologia 9:97–113

Reinert G (1966) Entwicklungstest. In: Handbuch der Psychologie. Hogrefe, Göttingen, pp 315–321

Rödel A (1987) Psychomotorische Störungen bei Erkrankungen des depressiven Formenkreises. Diss, München

Schoppe KJ (1974) Das MLS-Gerät. Ein neuer Testapparat zur Messung feinmotorischer Leitungen. Diagnostica 20(1):43–46

Streck P (1991) Klinisch-Experimentelle Untersuchungen über die Anwendung aktiv-mentaler Trainingsmethoden zur unterstützenden Therapie der endogenen Depression. Diss, München

Ulich E (1973) Beiträge zum mentalen Training, Schriftenreihe Training und Beanspruchung. Frankfurt am Main

Wernicke K (1900) Grundriß der Psychiatrie in klinischen Vorlesungen. Thieme, Leipzig

Zerssen D v (1973) Beschwerdeskala bei Depressionen. Therapiewoche 46:4426–4440