Eur Arch Psychiatry Clin Neurosci (1994) 244:59–64


ORIGINAL PAPER

Peter Martin · Michaela Tewesmeier · Matthias Albers
Gary Schmid · Christian Scharfetter

# Investigation of gestural and pantomime performance in chronic schizophrenic inpatients

Received: 20 September 1993 / Accepted: 27 April 1994

**Abstract** The present study compared the performance on apraxia tests and additional motor tasks between medicated chronic schizophrenic inpatients ($n$ = 21) and healthy subjects ($n$ = 21). Praxis testing did not reveal a clear-cut apractic syndrome in the patient group. The most striking difference between patients and healthy controls was the more frequent use of body parts as object (BPO) by schizophrenic patients ($P < 0.0003$). There was no significant difference in psychopathology between subgroups classified according to their BPO performance, although a tendency towards a difference in duration of illness and the actual hospitalization period existed between BPO subgroups. Schizophrenic patients performed more poorly than controls in frontal motor tasks: sequencing, including oral sequential movements, and reciprocal innervation. Frontal motor task performance tended to be related to the negative dimension of schizophrenia in accordance with previous studies. The data do not support the assumption that BPO performance is part of a multidimensional concrete attitude in schizophrenic patients. Nevertheless, this peculiarity in motor behaviour might be a link between neurophysiology and psychopathological phenomenology in schizophrenia. We therefore suggest further investigation focusing the performance of object-related pantomime movements in schizophrenic patients.

**Key words** Apraxia · Body part as object · Schizophrenia

## Introduction

Recently, Walker and Lewine (1990) reported on a preliminary study in which they investigated developmental precursors of schizophrenia using home movies that showed healthy children together with their siblings who developed schizophrenia in later life. The results suggest that viewers blind to the identity and psychiatric status of the subjects in the films are able to differentiate individuals who later become schizophrenic patients from their unaffected siblings before the age of 8 years. The judgement was made according to the viewers, own criteria, which included neuromotor characteristics of the children.

Earlier in the 20th century Bleuler (1911) mentioned movement disturbances in a group of schizophrenics. While pointing out the peculiarities of gait in schizophrenic patients he also referred to their "apraxia-like symptoms". Kraepelin (1904) described various odd characteristics of voluntary actions in schizophrenia, stressing the strange mannerisms of patients while eating, e.g. eating by hand or making use of a spoon in some extraordinary ways. In his work on psychomotor movement disorders Kleist (1908) claimed motor behaviour as an objective correlate of different psychic disorders. Accordingly, more recent investigations aimed at identifying motor or movement characteristics of psychoses as biological markers and diagnostic signs (Merriam et al. 1990; Günther et al. 1986; Manschreck 1982; Yates 1973; King 1954).

Freeman (1969) reported his findings on some chronic schizophrenic patients to whom he applied tests for disorders of motility, including serial and reciprocal movements. He stated that these patients show disturbances in posture as well as in voluntary movements, e.g. blocking, delayed initiation, incomplete movement, repetition and inability to pass from one step in a movement sequence to the next.

To investigate voluntary motor activity in schizophrenia Manschreck et al. (1982) used the techniques proposed by Freeman (1969). They found disturbances in voluntary movements in a vast majority of schizophrenic patients. Besides clumsiness and awkwardness in spontaneous movement, they especially found elicited motor disturbances, primarily abnormalities in alternating movements and motor sequencing.

P. Martin (✉)
Psychiatric Department of the University of Freiburg,
Hauptstrasse 5, D-79104 Freiburg, Germany

M. Tewesmeier · M. Albers · G. Schmid · Ch. Scharfetter
Psychiatric University Hospital Zurich,
Research Department P.O.Box 68, CH-8029 Zurich, Switzerland

In their study on relations between neurological signs and the positive-negative dimension in schizophrenia Merriam et al. (1990) found a significant association between negative symptomatology and neuropsychological signs of prefrontal impairment. They also examined praxis (buccolingual, limbs and finger) and found a positive correlation between abnormalities in praxis-testing and duration of illness, but no association with positive or negative symptoms, age or neuroleptic dose. Although the authors did not give much information about the extent and characteristics of disturbances in praxis test performances, their results might nevertheless suggest that apractic signs are schizophrenia trait-markers that appear in the later course of the disease. One could also speculate that a more elaborate and extended examination of praxis might uncover these disturbances in voluntary movement at earlier stages of the illness.

As Manschreck (1982) assumes in accordance with the early view of Kleist (1908), certain motor and thinking abnormalities in schizophrenics may have a common pathological basis. Current neuropsychological models of schizophrenia tend to integrate the neuroanatomical and neurophysiological basis of motor function and cognitive processes. Frith and Done (1988) assume that thinking is internalized action and focus on a hypothetical internal monitoring system for voluntary actions (and thoughts). They propose that failure in the internal labelling of acts, i.e. the incapacity for information about self-generated acts to reach the monitor might explain positive symptoms of schizophrenia (not recognizing the internal source of "self" of actions), whereas negative symptoms are attributed to a failure in initiating self-generated movements.

Gray et al. (1991) propose a model for integrating the neural and cognitive aspects of positive symptoms, and focus on connections between the limbic system and basal ganglia (striatum). Their model explains certain substantial aspects of motor behaviour such as the reaction to novelty and the transition from control to automatic processing, which are regarded as crucial in schizophrenic disorders.

Besides other experimental investigations of movement disturbances in schizophrenic patients, classical ideomotor-apraxia testing may serve as a means to study the drafting of motor concepts in an elaborate and partly standardized way. Particularly, the ability to plan ahead ideationally and to perform actions symbolically are assessed by testing ideomotor apraxia. Apraxias are defined as disorders of the execution of learned skilled movements not caused by paresis, akinesia, abnormality of tone or posture, interference of abnormal involuntary movements, deafferentation, incomprehension or inattention to commands (Heilman 1979; Geschwind 1975). The term ideomotor apraxia refers to a disorder limited to simple, single movements and gestures, although complex movements can indeed be correctly executed. Thus, the performance of single gestures on verbal command or by imitation is disturbed in ideomotor apraxia (Hécaen 1981), whereas spontaneous activity (object manipulation) is only rarely disturbed.

Since the pioneering work of Liepmann (1905), who was the first to study apraxia systematically, localizational neuroscience attributed ideomotor apraxia to a dysfunction of the dominant hemisphere, especially of the parietal cortex (presumed to contain the s.c. visuokinesthetic motor engrams) and the intra- and interhemispheric interconnections between the parietal lobe and other areas that are involved in the execution of learned movements (e.g. Heilman 1979).

Lesions within different brain areas and neural systems may give rise to different apractic syndromes regarding the body regions affected (orofacial, axial and limb movements; Geschwind 1975). To our knowledge a systematic investigation of apraxia in schizophrenia that compares patients with healthy subjects does not exist.

The aim of this study was to determine whether chronic schizophrenic patients manifest an apractic syndrome and whether movement peculiarities in gestural and pantomime performance correspond to: (1) distinct psychopathological features, (2) motor abnormalities that have already been described in schizophrenic patients, or (3) course variables of the disease. We applied a battery of apraxia tests to chronic schizophrenic patients who were thoroughly evaluated according to their psychopathological features and examined neurologically. Healthy subjects served as a control group.

## Subjects and methods

A group of 21 inpatients (17 men and 4 women) of the Psychiatric University Hospital, Zurich, who met DSM-III-R (American Psychiatric Association 1987) criteria for chronic schizophrenia were examined. A control group of 21 healthy volunteers who were matched with patients for age and gender were recruited from the hospital staff by an experienced psychiatrist. Healthy subjects satisfied both the *currently not mentally ill* and *never mentally ill* categories of the Research Diagnostic Criteria (RDC) (Spitzer et al. 1978). The mean age in the patient group was 40.7 years (SD ± 14.3 years), and 41.5 years (SD ± 14.3 years) in the group of healthy controls. All patients were chronically pretreated with neuroleptics. Data on the type and dosage of medication were not available for all patients. Patients differed in age at onset of disease, duration of illness (mean 16.9 years; SD ± 13.6; min. 3.0 and max 43.1) and duration of actual hospitalization period.

The mean education level, which was assessed in four degrees (primary school to university), was 2.0 (SD ± 0.6) in the patient group and 2.6 (SD ± 0.9) in the group of healthy subjects. Characteristics of the study samples are indicated in Table 1. All patients underwent a thorough neurological examination as described by DeJong (1967) and Monrad-Krohn and Refsum (1964). Additionally, Parkinsonism was assessed by the motor subscale of the Unified Parkinson's Disease Rating Scale (UPDRS; Fahn et al. 1987) and Tardive Dyskinesia (TD) by the Simpson Rating Scale for Tardive Dyskinesia (Simpson et al. 1979). Handedness was assessed by the Edinburgh Inventory (Oldfield 1971).

For the examination of praxis we applied several different inventories. Ideomotor apraxia was assessed by the Apraxia-Test developed by Kertesz and Ferro (1984), which consists of 20 items in 4 descriptive categories (facial, e.g. "whistle"; intransitive, e.g. "wave goodbye"; transitive, e.g. "pretend to use a toothbrush"; complex bimanual, e.g. "pretend to drive a car") and by an additional inventory also distinguishing object-related (transitive, e.g. "pretend to hammer a nail into a wall"), nonobject related (intransitive, e.g. "threaten"), high emotional (e.g. "blow a kiss") and low emotional (e.g. "pretend to comb your hair"), meaningless

**Table 1** Characteristics of group of chronic schizophrenic patients ($n$ = 21) and healthy subjects ($n$ = 21)

| | Chronic schizophrenic patients | | Healthy subjects | |
|---|---|---|---|---|
| Variable | Mean | SD | Mean | SD |
| Age (years) | 40.7 | ± 14.3 | 41.5 | ± 14.3 |
| Males vs females | 17/4 | | 17/4 | |
| Education[a] | 2.0 | ± 0.6 | 2.6 | ± 0.9 |
| Age at onset (years) | 23.8 | ± 6.1 | | |
| Length of illness (years)[b] | 16.9 | ± 13.6 | | |
| Length of actual hospitalization period (years) | 5.2 | ± 9.7 | | |

[a] Four levels (primary school to university)

[b] Min 3.0; max 43.1

(e.g. "touch your knees with a pencil") as well as expressive gestures with high symbolic value (e.g. "military salute"). Single items were related to more than one category.

Axial movements were examined according to the proposals of Poeck et al. (1982) (13 items; e.g. "lift your shoulders", "close your eyes") and Geschwind (1975) (7 items, not assessing head and eye movements; e.g. "kneel", "take the position of a boxer"). Additionally, buccofacial apraxia and dynamic oral movements were examined by two different tests as suggested by Poeck and Kerschensteiner (1975) and Luria (1966; 9 items and 6 items; e.g. "show your teeth", "clear your throat" and "show your teeth and then stick out your tongue"). Simple and more complex motor tasks, including the Ozeretski's test, the fist-ring test and the fist-edge-palm test for motor sequencing (Luria 1966) were assessed according to the proposals of Freeman (1969).

Movement errors were rated by the scoring system of Kertesz and Ferro (1984) and an error type called "body part as object" (BPO) was additionally assessed (Goodglass and Kaplan 1963; Rothi et al. 1988). The BPO performance means that the subject uses a part of his or her body (most typically a hand or fingers) as the imagined tool according to a given pantomime. For example, the index finger rubbed against the teeth becomes the toothbrush in response to "show me how you would pretend to brush your teeth". We did not discourage the use of body parts as objects prior to the examination.

To assess psychopathology in 19 (15 men and 4 women) of the 21 chronic schizophrenic patients, the Scale for Assessment of Negative Symptoms (SANS) (Andreasen 1982) and the Positive and Negative Syndrome Scale (PANSS; Kay et al. 1987) were used. The positive syndrome score, negative syndrome score and general psychopathology score of the PANSS were calculated separately, and the SANS summary score was defined as the sum of items 8, 13, 17, 22 and 25.

The neurological examination in particular praxis testing, and the assessment of psychopathology, were performed by two independent well-trained clinicians. Only univariate tests were used for analysis because of the small sample sizes ($n$ = 21) compared to the number of variables (15–19) subject to investigation. All calculations were carried out with the SAS statistical analysis system (SAS Institute Inc.). Because the variables being compared did not fulfil the normal distribution requirement of the two-tailed $t$-test, comparisons of independent groups were performed by the Wilcoxon Rank Sum Test (also called the Mann-Whitney-U-Test). This test provides a comparison of two independent populations with regard to their central tendency. Spearman correlation coefficients were evaluated between variables.

Type 1 error results from the probability of obtaining one or more significant results by chance amongst a set of N tests. This type of error can be estimated with the help of the binominal expansion (control of type 1 error is called "alpha-protection"). In the present investigation Wilcoxon Rank-Sum tests were carried out with an $\alpha$-level of 0.05 comparing averages of 15 variables between BPO and non-BPO groups, and on 19 variables between patient and control groups.

On the one hand, there is already a 46% probability that, by validity of the null hypothesis ("average values are equal in the two groups") at the 5% level, none of the 15 variables turn out by chance to have significant differences in their average values between groups (from the binominal term $(1-\alpha)^{15}$, with $\alpha = 0.05$). However, there are only 2 chances in 1000, for $\alpha = 0.05$, that any 5 of 19 variables turn out by chance to have significant differences in their average values between groups. Thus, our failure to find significant differences in certain features between patients and healthy subjects with vs without BPO performance (see *Results*) provides no conclusive testimony of an actual similarity between groups, whereas, our ability to find significant differences in certain features between subjects with vs without the diagnosis schizophrenia does indeed indicate an actual difference between these groups.

## Results

In both groups right-handers outweighed left-handers (19:2 in the patient group and 20:1 in the control group). The mean laterality quotient (LQ) was 69.7 (SD ± 57.3) for the patient group and 86.9 (SD ± 30.8) for controls. This difference was not statistically significant.

The neurological examination did not reveal signs of focal brain lesion in any patient. The assessment of Parkinsonian symptoms by the UPDRS (motor subscale) failed to reveal marked symptoms in any patient. Parkinsonism was moderate in one subject only. The most frequent symptom (13/21) was minimal hypomimia (not definitely above normal range) and slight rigidity could be detected in 8 subjects (absent in 13 subjects).

Tardive dyskinesia (TD) as assessed by the Simpson Rating Scale was absent in most of the patients. None of them showed moderately severe or severe dystonic/dyskinetic disturbances. In the small group of patients who revealed mild or moderate TD the oral/perioral region (3 mild and 2 moderate) and the lower extremities (3 mild and 2 moderate) were most-often affected. In the patient group the mean score was 10.3 (SD ± 5.2) for the PANSS positive syndrome, 16.3 (SD ± 8.1) for the PANSS negative syndrome and 11.7 (SD ± 4.1) for the SANS summary score.

The movements of schizophrenic patients in gestural and pantomime performance were generally more clumsy, coarse and less extensive in space and time compared to healthy controls, but in only a few cases could they be

judged as "impaired but recognizable performance" or worse (score 2–0 according to Kertesz and Ferro 1984). No differences were found between the respective performances of the right and left arm independent of object relation or emotional loading of the desired movements. Additionally, in the Kertesz and Ferro (1984) apraxia test 9 schizophrenic patients did not symbolize the object when asked to "blow out a match" (whereas only one subject made this mistake in the control group). Asked to "sniff a flower", many patients (and healthy controls) inclined forward, making it difficult to judge object symbolization in this similar task.

The test for axial movements (Geschwind 1975; Poeck et al. 1982) did not show any difference between groups. When assessed according to Geschwind no disturbances in axial praxis could be revealed in both groups. Furthermore, only two patients in the schizophrenic group performed head and trunk movements instead of eye and neck movements (en bloc) when tested according to Poeck et al. (1982). Accordingly, no statistically significant difference resulted between the groups. (Head and eye movements were not assessed by the scale of Geschwind 1975).

Oral- and facial-movement testing according to Poeck and Kerschensteiner (1975) and Luria (1966) revealed a statistically significant ($P < 0.009$) difference between groups, but this difference could be attributed to only one item ("put your tongue between your teeth and your lower lip"). Here six patients (and only one of the controls) did not perform correctly. In addition, there were only two other items where one or two patients (and none of the controls) revealed incorrect movements. Schizophrenics did worse than healthy subjects in performing dynamic oral movements as tested according to Luria (1966; $P < 0.0006$). This was true only for sequencing, not for one-step movements.

The most striking difference between schizophrenic patients and healthy subjects in apraxia tests was the frequency in using body parts as objects (BPO). This manner of performance was more frequent ($P < 0.0003$) in the patient group. There were more individuals who exhibited BPO movements and more BPO movements per individual among chronic schizophrenics (Table 2).

The BPO performance was not associated with psychopatholgoy, particularly not with negative symptoms assessed by SANS and PANSS negative-syndrome items. The subgroup without BPO movements scored even slightly higher for the negative syndrome items, particularly for the item "difficulty in abstract thinking". There was no statistically significant difference between the education level of patients and controls with and without BPO movements.

Patients and healthy subjects with BPO performance were older (without statistical significance) than those without. There was a distinct difference in the group of schizophrenics according to the duration of illness and the duration of the actual hospitalization period, both being longer in the subgroup with BPO movements (mean 20.6 vs 7.6 years and 147 vs 2608 days, respectively). Nevertheless, these differences failed to meet statistical significance at the 5% level ($P < 0.057$ and $P < 0.11$) due to the small size of the subgroups.

**Table 2** Frequency distribution of body part as object performance (BPO) for chronic schizophrenic patients ($n = 21$) and healthy subjects ($n = 21$)

| | Number of BPOs exhibited | | | | | |
|---|---|---|---|---|---|---|
| | 0 | 1 | 2 | 3 | 4 | 5 |
| Healthy subjects | 18 | 2 | 1 | 0 | 0 | 0 |
| Chronic schizophrenic patients | 6 | 10 | 2 | 1 | 1 | 1 |

The examination of motility according to Freeman (1969) revealed a statistically significant difference between healthy subjects and schizophrenics ($P < 0.0001$) due to more frequent mistakes in motor sequencing and reciprocal movements (Luria subtest; $P < 0.0001$). Mistakes in the Luria subtest (lower scores) tended to be related to the SANS summary score ($r = -0.41$; $P < 0.08$) and showed a weaker correlation ($r = -0.39$; $P < 0.10$) with the PANSS negative score.

## Discussion

The main finding of this study is that chronic schizophrenic inpatients compared with healthy subjects more often make use of their body parts as objects in pantomime performance. There were more BPO movements per subject as well as more subjects with BPO movements in the patient group at praxis testing.

Goodglass and Kaplan (1963) were the first to describe BPO in aphasic patients with apraxia. These authors presumed that BPO might express the loss of emotional distance to the performed action and allow the subject to compensate for an impaired or undeveloped function. Kaplan (1968; cited in Goodglass and Kaplan 1983) found BPO performance regularly in children up to 8 years of age. In their study on error-pattern analysis in ideomotor apraxia, Rothi et al. (1988) saw, in accordance with the findings of Goodglass and Kaplan (1963), BPO errors substantially more often in patients with brain lesions than in healthy controls, whereas Duffy and Duffy (1989) could not find a significant difference between brain-damaged patients and healthy subjects according to BPO measures. Goodglass and Kaplan (1963) stated that gestural deficiencies including BPO are part of an apractic disorder and not, as one could assume, part of a general communication disorder.

Our data do not suggest an association between BPO performance and the negative symptoms of schizophrenia, particularly not with the item "difficulty in abstract thinking". Therefore, our data do not directly support the assumption that BPO in schizophrenic patients is part of a multidimensional concrete attitude (Goldstein and Scheerer 1941) in schizophrenic patients. Nevertheless, this assumption certainly has not been falsified by our findings insofar as only one of our patients did not reveal any difficulty in abstract thinking and all patients showed

predominantly negative symptoms in clinical psychopathological examination.

One could conjecture that BPO in schizophrenic patients reflects their difficulty to deal with novelty. The absence of an object confronts the individual with a new and difficult aspect of an otherwise simple habitual motor task: This aspect can be less successfully integrated into the motor program (without BPO) by schizophrenic patients than it can by healthy subjects.

In our study we could not find clear-cut apraxia or dyspraxia in schizophrenic patients. Pantomime and gestural movements performed by schizophrenic patients were often expressionless and concentrated on the essential part of the task, thus appearing coarse and meagre. In most cases performance could not definitely be rated as "impaired" (i.e. not reaching the maximum score) in the Kertesz scale. Only a few patients showed abnormal en bloc movements when tested for axial praxia.

Schizophrenic patients also did not show an unequivocal oral or buccofacial apractic syndrome, although the patient group differed significantly from controls in both instruments used to test oral movements. This difference could be attributed to sequential movements in one test (one-step movements were done completely correctly in this test) and to mistakes in performing a single item in the other.

A more elaborate error-pattern analysis, however, and even more different items to test oral praxia, may reveal an apractic or oral apractic syndrome, which distinguishes between schizophrenic patients and healthy controls or other psychiatric control groups. We looked for apractic signs in only one group of schizophrenic patients with predominant negative symptoms. Therefore, our findings do not rule out the possibility that apraxia can be found in other schizophrenic subgroups (e.g. with predominant paranoid-hallucinatory symptomatology). The BPO performance in our schizophrenic patients tended to be related to the duration of illness and the actual hospitalization period, which means that the frequent use of body parts as object in transitive motor tasks is not an early sign in the course of schizophrenia.

Chronic schizophrenic patients performed worse than healthy subjects in tests for sequencing and reciprocal coordination. This was also true for oral-movement sequences. Furthermore, mistakes in performing these tasks were related to the negative symptoms of schizophrenia. According to Luria (1966) difficulties in motor sequencing and reciprocal coordination reflect a dysfunction of the premotor cortex, a "defect of successive kinetic organisation" or of the "kinetic melodies". Our data are in accordance with the results of Merriam et al. (1990), who found a significant correlation between prefrontal neurological impairment and negative dimensions of schizophrenia, and support again the assumption of frontal-lobe dysfunction in schizophrenic negative symptoms (e.g. Andreasen 1989; Weinberger 1988).

The assessment of Parkinsonian symptoms and TD in the patient group revealed only mild symptoms in some of the patients, and marked drug-induced extrapyramidal symptoms did not occur. Thus, it seems unlikely that movement abnormalities found in apraxia tests and examination of motility in schizophrenic patients results from chronic neuroleptic treatment. This assumption is supported by the findings of Manschreck et al. (1982), which suggest that neuroleptics lessen motor disturbances (not extrapyramidal) rather than cause them. Of course, the limitations of our data do not enable us to definitely exclude the possibility that motor findings in schizophrenic patients are not due to neuroleptic treatment.

Motor behaviour peculiarities are important contributions to the understanding of schizophrenias conceived as neurodevelopmental disorders. As a result of our findings we suggest further investigation focusing on the performance of transitive object-related pantomime movements in schizophrenic patients and in comparison with other diagnostic groups, e.g. affective disorders.

**Acknowledgements** The authors thank M. Regard and Th. Landis from the Department of Neurology, Neuropsychology Research Group, University Hospital Zurich, for their support in designing the praxis testing.

## References

American Psychiatric Association (1987) Diagnostic and statistical manual of mental disorders (DSM-III-R), 3rd edn revised. American Psychiatric Association, Washington, DC

Andreasen NC (1982) Negative symptoms in schizophrenia. Definition and reliability. Arch Gen Psychiatry 39:784–788

Andreasen NC (1989) Neural mechanisms of negative symptoms. Br J Psychiatry 155 (Suppl 7):93–98

Bleuler E (1911) Dementia praecox oder Gruppe der Schizophrenien. In: Aschaffenburg G (ed) Handbuch der Psychiatrie, spez. Teil 4/I, Deuticke, Leipzig, pp 132–178

DeJong R (1967) The neurological examination. Harper & Row, New York

Duffy RJ, Duffy JR (1989) An investigation of body part as object (BPO) responses in normal and brain-damaged adults. Brain Cogn 10:220–236

Fahn S, Elton RL (1987) Unified Parkinson's Disease Rating Scale. In: Fahn S, Marsden CD, Calne DB, Goldstein M (eds) Recent developments in Parkinson's disease (vol 2) Macmillan health care information, Florham Park, New Jersey, pp 153–163

Freeman T (1969) Psychopathology of the psychoses. Tavistock Publications, London

Frith CD, Done DJ (1988) Towards a neuropsychology of schizophrenia. Br J Psychiatry 153:437–443

Geschwind N (1975) The apraxias: neural mechanisms of disorders of learned movement. Am Sci 63:188–195

Goldstein K, Scheerer M (1941) Abstract and concrete behavior. Psychol Med Monogr Suppl 53:239

Goodglass H, Kaplan E (1963) Disturbance of gesture and pantomime in aphasia. Brain 86:703–720

Goodglass H, Kaplan E (1983) The assessment of aphasia and related disorders. Lea & Febiger, Philadelphia

Gray JA, Feldon J, Rawlins JNP, Hemsley DR, Smith AD (1991) The neuropsychology of schizophrenia. Behav Brain Sci 14: 1–84

Günther W, Günther R, Eich FX, Eben E (1986) Psychomotor disturbances in psychiatric patients as a possible basis for new attempts at differential diagnosis and therapy. Eur Arch Psychiatry Neurol Sci 235:301–308

Hécaen H (1981) Apraxias. In: Filskov SB, Boll TJ (eds) Handbook of clinical neuropsychology. Wiley, New York, pp 257–286

Heilman KM (1979) Apraxia. In: Heilmann KM, Valenstein E (eds) Clinical neuropsychology, Oxford University Press, pp 159–185

Kay SR, Fiszbein A, Opler LA (1987) The positive and negative syndrome scale (PANSS) for schizophrenia. Schizophr Bull 13: 261–276

Kertesz A, Ferro JM (1984) Lesion size and location in ideomotor apraxia. Brain 107: 921–933

King HE (1954) Psychomotor aspects of mental disease. Harvard University Press, Cambridge, Massachusetts

Kleist K (1908) Untersuchungen zur Kenntnis psychomotorischer Bewegungsstörungen bei Geisteskranken. Klinkhardt, Leipzig

Kraepelin E (1904) Psychiatrie: Ein Lehrbuch für Studierende und Ärzte. Barth, Leipzig

Liepmann H (1905) Ueber Störungen des Handelns bei Gehirnkrankheiten. Karger, Berlin

Luria AR (1966) Higher cortical functions in man. Basic Books, New York

Manschreck TC, Maher BA, Rucklos ME, Vereen DR (1982) Disturbed voluntary motor activity in schizophrenic disorder. Psychol Med 12: 73–84

Merriam AE, Kay SR, Opler LA, Kushner SF, van Praag HM (1990) Neurological signs and the positive-negative dimension in schizophrenia. Biol Psychiatry 28: 181–192

Monrad-Krohn GH, Refsum S (1964) The clinical examination of the nervous system. Lewis, London

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

Poeck K, Kerschensteiner M (1975) Analysis of the sequential motor events in oral apraxia. In: Zülch KJ et al. (eds) Cerebral localization. Springer, Berlin Heidelberg New York

Poeck K, Lehmkuhl G, Willmes K (1982) Axial movements in ideomotor apraxia. J Neurol Neurosurg Psychiatry 45: 1125–1129

Rothi LJG, Mack L, Verfaellie M, Brown P, Heilman KM (1988) Ideomotor apraxia: error pattern analysis. Aphasiology 2: 381–388

Simpson GM, Lee JH, Zoubok B, Gardos G (1979) A rating scale for tardive dyskinesia. Psychopharmacology 64: 171–179

Spitzer RL, Endicott J, Robins E (1978) Research diagnostic criteria. Arch Gen Psychiatry 35: 773–782

Walker E, Lewine RJ (1990) Prediction of adult-onset schizophrenia from childhood home movies of the patients. Am J Psychiatry 147: 1052–1056

Weinberger D (1988) Schizophrenia and the frontal lobe. TINS 11: 367–370

Yates AJ (1973) Abnormalities of psychomotor functions. In: Eysenck HJ (ed) Handbook of abnormal psychology. Pitman, London, pp 261–283