

Ball Experiments in 32 Acute Akinetic Catatonic Patients: Deficits of Internal Initiation and Generation of Movements

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Summary: We undertook ball experiments in 32 akinetic catatonic patients in order to determine specific functional deficits in the motor system in akinetic catatonia. Standardized ball experiments (catching, throwing, stopping, kicking) were conducted in 32 acute akinetic catatonic patients (23 without neuroleptics on admission), diagnosed according to Lohr, Rosebush, and the *Diagnostic and Statistical Manual of Mental Disorders (3rd ed, revised)* on days 0 and 21. Additionally, associated psychopathology was evaluated using different scales on days 0 and 21: the Global Assessment Scale, the Brief Psychiatric Rating Scale, the Hamilton-Anxiety Scale, the scale for the assessment of negative symptoms (SANS), and the Simpson scale for extrapyramidal side effects (SEPS). Significantly more patients were able to perform more externally guided tasks (catching, stopping) than internally guided tasks (throwing, kicking). Patients showed significantly more posturing and awkward movements on day 0 than on day 21. There was a significantly positive correlation between hypokinetic extrapyramidal features (SEPS) and negative symptoms with their cognitive alterations (SANS) on day 0. The findings suggest a deficit of internal initiation, as in parkinsonism, as well as a dysfunction in the generation of voluntary movements in akinetic catatonia. We assume an underactivity in the dorsolateral prefrontal cortex and the supplementary motor area with consecutive down-regulation of the cortical-striatal-thalamic circuit, the "motor loop," in catatonia. **Key Words:** Catatonia—Ball experiments—Internal initiation—Voluntary movements—Frontal cortex.

The term catatonia was first introduced by Kahlbaum when he described patients with a wide range of motor abnormalities, including immobility, mutism, posturing, grimacing, rigidity, negativism, staring, stereotypy, verbigerations, waxy flexibility, echolalia, and echopraxia (1). In contrast to Kahlbaum, who regarded catatonia as a separate disease entity, Kraepelin (2) and Bleuler (3) referred to catatonia primarily as a subtype of schizophrenia.

However, the more recent literature has empha-

sized the notion that catatonia should be considered a syndrome that occurs in different diseases, both organic and nonorganic, rather than a subtype of schizophrenia (4-6).

The pathophysiology of motor function in akinetic catatonia is likewise unknown, and its relationship with Parkinson's disease as well as schizophrenia and depression has never been clarified. It displays hypokinetic motor features (akinesia, rigidity) more or less similar to parkinsonism, in which a deficit of the internal initiation of movements and the supplementary motor area in the frontal cortex may account for akinesia (7,8). Often catatonia occurs in patients with chronic schizophrenia (9,10) or major depression (11), which both display a so-

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called psychomotor poverty syndrome (12) associated with dysfunction in the generation of voluntary movements and the dorsolateral prefrontal cortex (13,14).

Thus, the frontal cortex with the supplementary motor area (initiation of movements) and the dorsolateral prefrontal cortex (generation of voluntary movements) might be central in dysfunction of the motor system in catatonia.

Until now, the specific functional deficit in the motor system in catatonia remains unknown. In order to investigate different motor functions (generation, initiation, execution, control) we undertook ball experiments in 32 akinetic catatonic patients (see Fig. 1) and measured associated psychopathology.

METHODS

Subject Selection

The study population was composed of 32 acute akinetic catatonic patients with a mean age of 33.4 ± 10.8 years (16 women, mean age 35.3 ± 11.7 years; 16 men, mean age 31.5 ± 9.9 years). Twenty-three were neuroleptically untreated or free (at least 6 months off neuroleptics before admission) and nine had received neuroleptics either on the day of admission or in the 6 months before admission. They were selected from all consecutively admitted patients to the university psychiatric clinic in Frankfurt between January 1990 and May 1993 (incidence 2.8%).

Catatonic syndrome was diagnosed according to catatonia criteria by Lohr (catalepsy, positivism, or negativism, as well as at least two of the following signs: stereotypies, mannerisms/grimacing, bizarries, posturing, echophenomena, excessive muscular tension, mutism, staring) (15) and Rosebush (at

least four of the following signs: immobility, staring, mutism, rigidity, withdrawal, posturing, grimacing, negativism, waxy flexibility, echopraxia/lalia, stereotypies, verbigerations) (16). All patients had to be classified as retarded catatonia (predominant hypokinesia and underactivity) according to both criteria lists by two independent psychiatrists (G.N. and J.W.) with special experience in catatonia. Catatonic patients with predominant hyperkinesias and excessive activity who were diagnosed with excited catatonia were excluded (17). Moreover, all patients had to show akinesia, which, according to Rosebush, was defined as an absence of spontaneous and voluntary movements. Catatonic patients with concomitant Parkinson's disease or other extrapyramidal movement disorders were excluded. Comorbid diagnoses were made according to the *Diagnostic and Statistical Manual of Mental Disorders (3rd ed, revised) (DSM III R)* (18) by an independent psychiatrist on discharge.

Psychopathology

General psychopathology was evaluated using the Global Assessment Scale (GAS) (19) and the Brief Psychiatric Rating Scale (BPRS) (20). Extrapyramidal motor features were assessed using the Simpson Scale for extrapyramidal side effects (SEPS) (21), which is generally used for the measurement of hypokinetic and parkinsonic movements in psychiatry. Negative symptoms were assessed using the scale for the assessment of negative symptoms (SANS) (22). Anxiety was measured using the Hamilton Anxiety Scale (HAM-A) (23).

All psychopathological ratings were done by G.N. and J.W., who both had completed special rating training. Assessment of the intrarater and interrater reliability showed average intraclass corre-

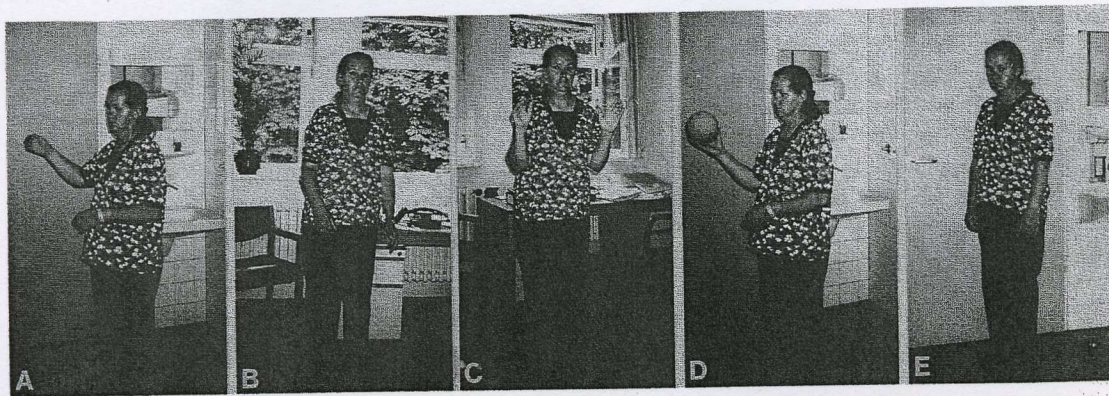


FIG. 1. Ball experiment in an acute akinetic catatonic patient: original position (A); catching (B,C); and throwing 1 (D,E).

lation coefficients of 0.90 and 0.95 for the different scales.

Motor Function: Ball Experiments

We developed a scale for the assessment of performance, based on yes/no decisions, in different motor tasks, including catching, throwing, stopping, and kicking of a ball (see Appendix).

In a first-step, execution or nonexecution of the respective task was evaluated. The way of execution (internal or external) or nonexecution (negativism or posturing) was assessed in a second step.

Furthermore, we evaluated affective reaction (parathymia, anxiety, happiness) and general movements (awkward, normal, slow) during the different motor tasks (see Appendix).

Study Design

After inclusion into our study sample, all catatonic patients participated in the above-mentioned ball experiments on the day of admission before initial medication (Fig. 1). Standardized ball experiments were conducted in a standardized setting and were directed by G.N. We used a soft ball the size of a soccer ball.

We approached the patients and ask them to play ball with us. We stood 2 m in front of the patient and threw the ball toward him so that he could easily catch it. Without saying anything further we repeated the same procedure three times. If the patient did not catch the ball, we explicitly asked him to catch it, which we repeated three times as well.

Having finished catching, we went on with the other motor tasks (throwing, stopping, kicking) in the same way. If the patient did not catch or stop the ball by himself, we put it into his hands or in front of his feet in order to throw or to kick. Videobased rating of performances in ball experiments was performed by two independent psychiatrists whose interrater reliability showed average intraclass correlation coefficients of 0.91–0.94 for the different motor tasks. If more than one item was rated with yes, only the predominant one was taken.

Over the next 3 weeks all patients received lorazepam (1 mg two to four times daily). In addition, depending on comorbid diagnosis, 24 patients received neuroleptics (haloperidol, benperidol), six patients received tricyclic antidepressants, six patients received lithium, and two patients received only lorazepam.

Ball experiments were repeated on day 21 in the same way as described above.

Psychopathology was assessed on day 0, before initial medication, as well as on day 21.

Statistical Analysis

Psychopathological results were expressed as means \pm SD. Deviations from normal distributions were calculated by use of the Kolmogoroff Smirnov or Fitt Goodness test. Differences between days 0 and 21 were calculated by *t* test for random samples. Ratings of the scale for the assessment of ball experiments (see Appendix) were calculated by means of frequencies and the McNemar test. All computations were executed using the SPSS-X statistics software system.

RESULTS

Catatonic Symptoms and Comorbid Diagnosis

According to Rosebush's criteria (10), on day 0 100% of our patients showed akinesia, 71% staring, 79% mutism, 58% autism, 63% posturing, 49% rigidity, 46% negativism, 79% waxy flexibility, 45% grimacing, 38% echolalia, 47% stereotypies, and 45% verbigerations. With regard to catatonic symptoms, on days 0 and 21 there were no significant differences between schizophrenic and nonschizophrenic, between affective and nonaffective, or between schizophrenic and affective catatonic patients. On day 21 five of our 32 catatonic patients were still classified as catatonic according to Lohr (15) and Rosebush (16).

According to *DSM III/R* criteria, the 32 catatonic patients included in the current study had the following diagnoses (diagnosis number in parentheses):

- Catatonic schizophrenia (295,2), *n* = 7
- Paranoid schizophrenia (295,3), *n* = 1
- Residual schizophrenia (295,6), *n* = 9
- Major depression (296,3), *n* = 6
- Bipolar disorder (296,4), *n* = 5
- Brief reactive psychosis (298,8), *n* = 2
- Organic catatonia (hypoxic brain disease or renal encephalopathy, respectively), *n* = 2

Psychopathology

Tables 1 and 2 shows scores for all rating scales on days 0 and 21. All differed significantly between days 0 and 21 (except for SANS alogia; Tables 1 and 2). Significantly positive correlations were found between SEPS and SANS total, affect, alogia, and apathia on day 0 (Tables 1 and 2). No significant

TABLE 1. Psychopathological scores: differences

	Day 0	Day 21	P (t test, days 0-21)
GAS	10.6 ± 4.6	42.5 ± 21.9	0.0001
BPRS	60.8 ± 13.9	33.5 ± 20.4	0.0001
HAM-A	22.8 ± 6.3	6.7 ± 3.4	0.002
SEPS	17.6 ± 7.3	6.6 ± 6.8	0.0001
SANS			
Total	103.2 ± 17.8	72.5 ± 20.3	0.0001
Affect	35.6 ± 4.8	24.2 ± 11.4	0.0001
Alogia	20.1 ± 5.7	20.2 ± 4.6	NS
Apathia	16.7 ± 5.4	10.6 ± 6.4	0.0008
Withdrawal	18.0 ± 8.1	11.0 ± 9.5	0.002
Attention	12.2 ± 4.4	6.1 ± 4.7	0.0001

correlations were found between HAM-A, SEPS, and SANS.

There were no significant differences between neuroleptically untreated and neuroleptically treated catatonic patients on days 0 and 21 with regard to GAS, BPRS, HAM-A, SEPS, and SANS.

Motor Function: Ball Experiments

Figure 2 and Table 3 show frequencies of scores of performances in our ball experiments. Approximately 50% were able to play ball on day 0 (Fig. 2). Execution differed significantly between stopping and kicking and showed higher scores in catching than in throwing (Fig. 2). Internal initiation showed significant differences between catching and throwing as well as between stopping and kicking on day 0 (Table 3). Posturing occurred significantly more in throwing and kicking than in catching and stopping (Fig. 2).

There was significantly more awkwardness of movements than slowness on day 0, whereas on day 21 the latter occurred significantly more than the former (Table 3). Predominating parathymia could be found significantly more than anxiety on day 0, whereas both almost disappeared on day 21.

There were no significant differences between the different diagnostic groups (affective, schizophrenic, nonaffective, nonschizophrenic) in the ball experiments and their different tests.

DISCUSSION

Motor Function

Approximately 50% of our akinetic catatonic patients were able to play ball (Fig. 2). Similar findings were made by Strauss, who demonstrated that some akinetic catatonic patients were able to play ball (24). Sacks observed patients with encephalitis lethargica and catatonic posturing who were able to

catch and throw a ball (25). Similar to us, both observed that the examiner had to take the initiative in order to make the patients play ball. Our results showed that more externally guided motor tasks such as catching and stopping could be executed much better by the patients than could more internally guided motor tasks such as throwing and kicking (Fig. 2). In addition, significantly more patients were able to catch and stop the ball without any external initiative than to throw and kick the ball by themselves (Table 3). More internally guided motor tasks such as throwing and kicking could either not be executed or could be performed only with initiative from the examiner (Table 3).

Such a dependence of akinetic catatonic patients on external initiative was already mentioned in the earlier literature about catatonia (26). With regard to such a functional deficit of the internal initiation of movements, there may be similarities between akinesia in Parkinson's disease and catatonia. Akinesia in Parkinson's disease is regarded as a disturbance of the internal initiation of movements (8,27,28): the patient "knows what he wants to do, but can not galvanize his muscles into action" (7). As in catatonia, the patients with Parkinson's disease are dependent to a high degree on external stimulation (29).

In contrast to parkinsonism, our akinetic catatonic patients showed different voluntary movements, either negativism or posturing in relation to the ball, when they were unable to execute the motor tasks (Table 3). They often showed at least some kind of voluntary movements that either resulted in active refusal (negativism) or haltungsverharren in relation to the ball (posturing). Moreover, unlike in parkinsonism, catatonic patients showed awkward movements rather than slowness of voluntary movements on day 0 while playing ball (Table 3). Thus, akinetic catatonic patients may not only have

TABLE 2. Psychopathological scores: correlations

Correlations	Day 0	Day 21
SEPS-SANS	r = 0.438	
Total	p = 0.011	NS
Affect	r = 0.377	
	p = 0.033	NS
Alogia	r = 0.68	
	p = 0.0001	NS
Apathia	r = 0.485	
	p = 0.0049	NS
Withdrawal	NS	NS
Attention	NS	NS
Other scales (GAS, BPRS, HAM-A)	NS	NS

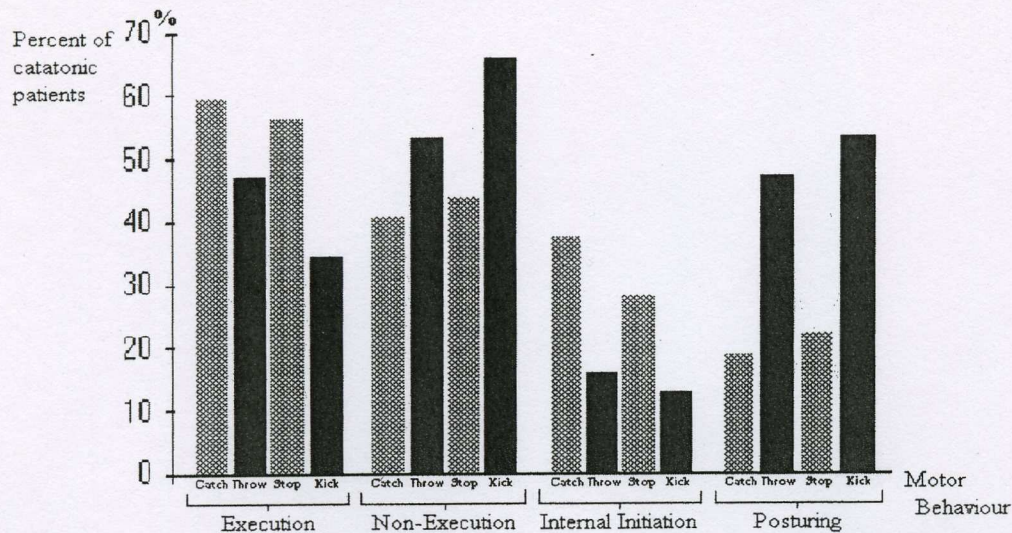


FIG. 2. Execution, nonexecution, internal initiation, and posturing in ball experiments in catatonia on day 0.

a deficit of internal initiation with slowness of voluntary movements, as in Parkinson's disease, but, in addition, they may show a dysfunction in the generation of voluntary movements resulting in negativisms, posturing, and awkwardness.

Psychopathology

We found significantly positive correlations between extrapyramidal hypokinetic features (SEPS) and negative symptoms (SANS) on day 0 (Tables 1 and 2). Our study confirms findings by other investigators who correlated hypokinetic catatonic phenomena with negative symptoms and cognitive deficits (10,15,30,31). Therefore, motor alterations in catatonia may be closely related to cognitive dysfunction (15,31). Pathophysiologically negative symptoms are related to functional deficits in the prefrontal cortex (22,32), whereas akinesia and extrapyramidal hypokinetic features, as measured with SEPS, are associated with alterations in the cortical-striatal-pallido-thalamo-cortical circuit (7,

27,28,33,34). Considering the positive correlation between motor alterations and negative symptoms, the cortical-striatal-pallido-thalamo-cortical circuit may be functionally dysregulated by the prefrontal cortex in catatonia, which would explain akinesia as well as dysfunction in the generation of voluntary movements.

CONCLUSION

Deficits of internal initiation of movements in Parkinson's disease are related to underactivity of supplementary motor area (SMA) and premotor area (7,33,35-37). Generation of voluntary movements is related to function of the dorsolateral prefrontal cortex (38), which shows underactivity in schizophrenic and depressive patients with a "psychomotor poverty syndrome" (13,14), in which generation of speech and movements is decreased (12).

Considering our findings of a functional deficit of internal initiation and a dysfunction in the genera-

TABLE 3. Initiation, noninitiation, and movements in ball experiments in catatonic patients

Ball experiment	Day	Initiation (execution)		Noninitiation (nonexecution)		Movements		
		Internal	External	Negativism	Posturing	Normal	Slow	Awkward
Catch	0	37.5	21.9	21.9	18.8	0	6.3	93.8
	21	84.4	15.6	0	0	15.6	80.6	3.1
Throw	0	15.6	31.3	6.3	46.9	0	3.1	96.9
	21	84.4	12.5	0	3.1	15.6	80.6	6.3
Stop	0	28.1	28.1	21.9	21.9	0	6.3	93.8
	21	87.5	12.5	0	0	15.6	80.6	9.4
Kick	0	12.5	21.9	12.5	53.1	0	3.1	96.9
	21	81.3	12.5	0	0	15.6	80.6	6.3

tion of voluntary movements in patients with akinetic catatonia, we suggest a hypofunction in the SMA and an altered function in the dorsolateral prefrontal cortex. Such an altered function in the dorsolateral prefrontal cortex, in combination with hypofunction of the supplementary motor area, might consecutively down-regulate the cortical-striatal-pallido-thalamic-cortical circuit, the motor-loop (34), in catatonia. Our finding of the positive correlation between extrapyramidal features (SEPS) and negative symptoms (SANS) in our akinetic catatonic patients would support such a pathophysiological hypothesis.

APPENDIX

Scale for the Assessment of Motor Performance in Ball Experiments

A. Catching

Execution: catches the ball

Internal: catches the ball without external guidance

External: catches the ball only when asked to

Nonexecution: does not catch the ball

Negativism: deliberately refuses to catch the ball

Posturing: Haltungsverharren in relation to the ball

B. Throwing

Execution: throws the ball back

Internal: throws the ball without external guidance

External: throws the ball only when asked to

Nonexecution: does not throw the ball back

Negativism: deliberately refuses to throw the ball

Posturing: Haltungsverharren in relation to the ball

C. Stopping

Execution: stops the rolling ball with feet

Internal: stops the ball without external guidance

External: stops the ball only when asked to

Nonexecution: does not stop the ball with feet

Negativism: deliberately refuses to stop the ball

Posturing: Haltungsverharren in relation to the ball

D. Kicking

Execution: kicks the ball back

Internal: kicks the ball without external guidance

External: kicks the ball only when asked to

Nonexecution: does not kick the ball back

Negativism: deliberately refuses to kick the ball

Posturing: Haltungsverharren in relation to the ball

E. Affective reaction during catching/throwing and stopping/kicking (facial expression)

Anxiety: patient looks fearful and frightened

Parathymia: grimacing, crying, or laughing without any apparent reason

Happiness: patient looks cheerful

F. General movements

Awkwardness: clumsiness, bizarreness of movements

Slowness: reduced speed in psychomotor activity

Normal: no psychomotor abnormalities

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