*British Journal of Psychiatry* (1991), **158**, 328–336

# Motor, Volitional and Behavioural Disorders in Schizophrenia
## 2: The 'Conflict of Paradigms' Hypothesis

P. J. McKENNA, C. E. LUND, A. M. MORTIMER and C. A. BIGGINS

An alternative to the conventional separation of extrapyramidal and catatonic symptoms exists in the 'conflict of paradigms' hypothesis, which proposes that there is a relative rather than absolute distinction between the two. The hypothesis predicts that a clinical association should exist between extrapyramidal and catatonic symptoms in schizophrenia. After rating 75 schizophrenic patients, a highly significant correlation between scores on the two classes of disorder was indeed found. This was composed of separate correlations between tardive dyskinesia and 'positive' catatonic phenomena, and Parkinsonism and 'negative' catatonic phenomena. The associations were not easily attributable to confounding factors and they were supported by factor analysis.

Even though it may on occasion be difficult to disentangle extrapyramidal and catatonic symptoms in schizophrenia, that they are essentially different from one another is regarded as uncontroversial. This view is based on two lines of reasoning, which might be termed the pathological and the phenomenological. *Pathologically*, extrapyramidal symptoms are the side-effects of neuroleptic drugs and reflect, for the most part, interferences with the function of the basal ganglia (Marsden & Jenner, 1980). Catatonic symptoms, on the other hand, are an expression of the disease process of schizophrenia, whose nature is as yet unknown. *Phenomenologically*, extrapyramidal and catatonic symptoms show broadly different characteristics. The former tend to be less complex and purposeful than the latter, the areas of clinical overlap between them are not great, and where there are similarities these have been considered to be limited to external appearances (Marsden *et al*, 1975). Although this reasoning seems straightforward, it is in fact open to challenge on both grounds.

From the pathological point of view, it has repeatedly been claimed that signs of basal ganglia dysfunction can sometimes be seen in untreated schizophrenia. Kraepelin (1907, 1913) and Farran-Ridge (1926) described schizophrenic patients who showed dyskinesia-like lip, mouth, tongue and jaw movements, as well as choreiform movements of the extremities. Steck (1926/27) likewise claimed that fugitive Parkinsonian signs could at times complicate catatonic stupor, and Reiter (1926) documented six cases of "Dementia Praecox Parkinsonoides" – patients with otherwise unexceptional schizophrenia who progressed through catatonia to a Parkinsonism-like terminal state.

More recently, in a retrospective examination of schizophrenic patients' case notes antedating any possible use of neuroleptics, Rogers (1985) was able to find statements like "having constant movements of hands, fingers and feet", "working his fingers like a choreic", "continually squirming about in bed", "exhibiting strange, writhing snake-like movements of the limbs" and "insane athetoid movements". Also recorded were descriptions of flexed posture, fixed, mask-like and wax-like facial expressions, and slow, sluggish, stiff or shuffling gaits. Owens *et al* (1982) discovered a group of 47 chronic schizophrenic patients who ostensibly had never received treatment with neuroleptic drugs. On examination approximately half of these patients showed evidence of involuntary movements like those of tardive dyskinesia, and which were indistinguishable from those seen in treated patients.

Phenomenologically also, the separation of extrapyramidal and catatonic symptoms has not always been considered to be absolute. Shortly after neuroleptic drugs were introduced, European authors commented that their effect seemed to be to convert acute schizophrenics into chronic schizophrenics (Rogers, 1991). The first author to describe neuroleptic-induced Parkinsonism (Steck, 1954) went further and suggested that it represented a form of catatonia modified by drug treatment. When tardive dyskinesia subsequently began to appear, its similarity to schizophrenic stereotypies and mannerisms was felt to have been close enough to have delayed its recognition (Jones & Hunter, 1969). In France, these apparent points of resemblance were elaborated into a cumbersome scheme in which different drug side-effects were systematically related to different aspects of the clinical picture (Deniker, 1960; Delay & Deniker, 1968). This failed, however, to gain wide acceptance and elsewhere the case for clinical unrelatedness gained ground and ultimately acquired the status of orthodoxy.

Recent work has re-opened this issue. After re-examining the above-mentioned long-stay schizophrenic patients, Rogers (1985) concluded that a rigid separation of their motor disorders into extrapyramidal and catatonic was not possible. Rather, such patients confronted the examiner with an array of disorders of movement, volition and behaviour, which seemed to be continuously distributed. At one end of this continuum were the simple disorders of dyskinesia and Parkinsonism; at the other were the complex disturbances of catatonia; between, however, were phenomena which showed greater or lesser degrees of affiliation with both and which in many cases had acquired dual terminologies – for example the arguably indistinguishable mannerisms and complex tics.

These suggestions that extrapyramidal symptoms in schizophrenia are not always drug induced, and that their separation from catatonic symptoms is relative rather than absolute, converge in Rogers' (1985) 'conflict of paradigms' hypothesis. According to this view, there is no fundamental distinction between the two classes of disorder: their demarcation is illusory, a reflection more than anything else of the historical divergence of neurological and psychiatric traditions at the beginning of the century, a divergence which has become increasingly inappropriate as biological psychiatry has reasserted itself. In reality, there is a single large group of abnormal motor phenomena, representatives of the entire spectrum of which can be found in schizophrenia, and only the emphasis of which is different in treated and untreated cases.

The orthodox and 'conflict of paradigms' views of schizophrenic motor disorder are irreconcilable, in particular in the ways they hypothesise extrapyramidal and catatonic phenomena to be related clinically. The former implies no particular association, while the latter suggests that they should be closely linked. In an earlier study, a correlation between extrapyramidal and catatonic symptom scores was an incidental finding (McKenna *et al*, 1988). This suggested a means of testing the different clinical predictions of the two views of schizophrenic motor disorder. The present study was undertaken to investigate just how far the two classes of symptom can be dissociated from each other.

## Method

The patients were drawn from the acute, rehabilitation and long-stay services of two hospitals, and their illnesses encompassed a wide range of severity and chronicity. All patients met DSM–III criteria (American Psychiatric Association, 1980) for schizophrenia or (in one case where the illness had lasted for less than six months) schizophreniform disorder. From these patients, two groups were selected for examination at different stages of the study. The first group consisted of 75 patients aged 18–70 years, consisting of acute, rehabilitation and long-stay cases in approximately equal numbers. The second group, examined subsequently under more rigorous conditions, comprised 40 short- and long-stay patients, aged 22–72 years, approximately half of whom were also in the first sample, the rest being new cases. The combined groups (93 patients) were used in the factor-analytic part of the study.

The 75 patients in the main sample were simultaneously rated by two interviewers, one examining and one observing. The motor disorder examination described in the previous paper (this issue, pp. 323–327) was carried out, following which ratings were made independently, without any conferring. One interviewer (PJM), who had experience in the assessment of extrapyramidal side-effects, rated tardive dyskinesia and Parkinsonism using established scales (Webster, 1968; Simpson & Angus, 1970; Simpson *et al*, 1979). The second interviewer (CEL or AMM) completed the Modified Rogers Scale (Appendix 1) and the Behavioural Observation Schedule (BOS) of Atakan & Cooper (1989), both of which include items rating catatonic phenomena. Overall severity of illness was estimated using the Global Assessment of Severity (GAS) of Endicott *et al* (1976). Chronicity of illness was assessed from case notes, as the average of maximum and minimum durations, where maximum represented the time in years from first recorded psychotic symptoms and minimum the time from first admission to hospital.

A similar procedure was carried out on the second sample of 40 patients. Here, however, an attempt was made to eliminate the possible bias introduced by simultaneous rating. Each patient was therefore examined twice on the same day, first by one rater (PJM) who performed a standard examination for extrapyramidal side-effects without accessory tests for catatonic phenomena; and then independently by the second rater (CEL or AMM) who completed the Modified Rogers Scale.

As the Modified Rogers Scale rates both extrapyramidal and catatonic phenomena, factor analysis of its constituent items permits another way of examining the degree of dissociation of the two classes of symptom. Eighteen of the scale's 36 items (to keep a ratio of 1:5 between variables and sample size) were thus selected for analysis; these included both extrapyramidal and catatonic symptoms, as well as a number potentially classifiable as either.

Scores for tardive dyskinesia and Parkinsonism were taken as the summed scores on the scales of Simpson *et al* (1979) and Simpson & Angus (1970). Two further extrapyramidal scores were also obtained. Simpson *et al*'s tardive dyskinesia scale contains a number of items that are potentially confoundable with catatonic phenomena (e.g. grimacing, caressing or rubbing of face/hair/legs, holokinetic movements) or that are arguably non-dyskinetic in nature (e.g. tremor of eyelids, akathisia). Accordingly a tardive dyskinesia (narrow) score, which excluded all such items, was also extracted. Simpson & Angus' Parkinsonism score is designed for use in psychiatric patients, but even so some of its items (e.g. reduced arm swing) could conceivably score non-Parkinsonian phenomena. The more

neurologically orientated scale of Webster (1968) was therefore also employed, excluding the last item which is directed to self-care.

Catatonic scores were abstracted from the Modified Rogers Scale as described in the previous paper (see Appendix 2 for details of rating). Because earlier work (Mortimer *et al*, 1990) had indicated that catatonic symptoms, like other classes of schizophrenic symptom, might separate into 'positive' and 'negative' groups, subscores for these were also derived. 'Positive' catatonic phenomena (those distinguished by the *presence* of an abnormality) comprised: *gegenhalten*, *mitgehen*, complex mannerism/stereotypy-like movements of face/head, complex mannerism/stereotypy-like movements of trunk/limbs, iterations, echopraxia, manneristic/bizarre gait, echolalia/palilalia, marked overactivity, excessive compliance/automatic obedience, negativism, hypermetamorphosis. 'Negative' catatonic phenomena (those characterised by the *absence* or *diminution* of a normal function) consisted of blocking/ambitendence, underactivity, poor/feeble compliance, and mutism. The four remaining items, complex abnormal posture, persistence of imposed postures, aprosodic speech and indistinct unintelligible speech, were felt not to be adequately classifiable as 'positive' or 'negative', and were included only in the total catatonic score. As discussed in the previous paper, for some of these items (manneristic/bizarre gait, marked overactivity, marked underactivity, aprosodic speech) a degree of uncertainty remained about whether they might rate extrapyramidal symptoms in some cases. Therefore 'positive' and 'negative' catatonic (narrow) scores were also calculated, which excluded these items.

Although the BOS is not designed for such a procedure, it is possible to derive a score for catatonic symptoms from its ratings. To accomplish this, scores on 14 putatively non-extrapyramidal motor and speech ratings were summed. These were defined narrowly and comprised: idiosyncratic peculiarities of gait, stupor, gross excitement, facial mannerisms or stereotypies, incomprehensibility of facial expression, body mannerisms and posturing, body stereotypies, catatonic movements, abnormally relaxed posture forms, abnormally reclined or closed posture, withdrawn posture, abnormally flat tone of voice, muteness, non-social speech.

Analysis of correlations was carried out using Spearman's non-parametric method, which does not allow partial correlations to be derived. The data were therefore also analysed using Pearson's (parametric) and Kendall's (non-parametric) procedures, from which partial correlations can be obtained. The factor analysis used to examine the 93 patients rated on the Modified Rogers Scale was that in the Statistical Package for the Social Sciences (SPSSX). The 18 items included in the analysis were selected on the basis that they encompassed extrapyramidal symptoms (5 items) and 'positive' (7 items) and 'negative' (3 items) catatonic phenomena, and those potentially classifiable as either (3 items). Most of the items had received substantial numbers of ratings of 1 and some of 2; the much less frequently rated *gegenhalten* and poor/feeble compliance were also included for reasons of interest.

## Results

In the main group of 75 patients, scores for tardive dyskinesia were significantly associated with chronicity ($r=0.27$, $P=0.01$) and age ($r=0.21$, $P=0.04$), but not with severity of illness ($r=0.13$, NS). Parkinsonism scores were not significantly associated with any of these variables. Scores for tardive dyskinesia and Parkinsonism were significantly inversely correlated with each other ($r=-0.29$, $P=0.006$). Catatonic scores from the Modified Rogers Scale were most significantly correlated with severity ($r=0.47$, $P<0.001$), less so with chronicity ($r=0.27$, $P=0.01$), and least of all with age ($r=0.24$, $P=0.02$).

The total extrapyramidal scores, calculated as the sum of scores for tardive dyskinesia and Parkinsonism for each patient, were highly significantly correlated with total catatonic scores on the Modified Rogers Scale ($r=0.58$, $P<0.001$). This also held true using the catatonic ratings derived from the BOS ($r=0.54$, $P<0.001$). When tardive dyskinesia and Parkinsonism, and 'positive' and 'negative' catatonic scores were separated, an even more striking pattern of association and dissociation was observed. Tardive dyskinesia and Modified Rogers 'positive' catatonic scores became even more significantly intercorrelated ($r=0.64$, $P<0.001$). There were similar but less marked correlations between Parkinsonism and Modified Rogers 'negative' catatonic scores ($r=0.35$, $P=0.001$). The cross-correlations between tardive dyskinesia and 'negative' and Parkinsonism and 'positive' catatonic scores were insignificant or inverse. These findings are illustrated in Fig. 1.

In order to exclude any contamination of the extrapyramidal scales with potential catatonic items, the analysis was repeated using the stricter (narrow) tardive dyskinesia scores and (Webster) Parkinsonism scores. This scarcely affected the correlation between tardive dyskinesia and Modified Rogers 'positive' catatonic scores ($r=0.60$, $P<0.001$), and the correlation between Parkinsonism and Modified Rogers 'negative' catatonic scores actually increased ($r=0.43$, $P<0.001$). When catatonic ratings were replaced by their corresponding catatonic (narrow) ratings, the correlation between tardive dyskinesia (narrow) and 'positive' catatonic (narrow) phenomena remained significant ($r=0.50$ and $r=0.46$, $P<0.001$). The correlation between Parkinsonism and 'negative' catatonic (narrow)

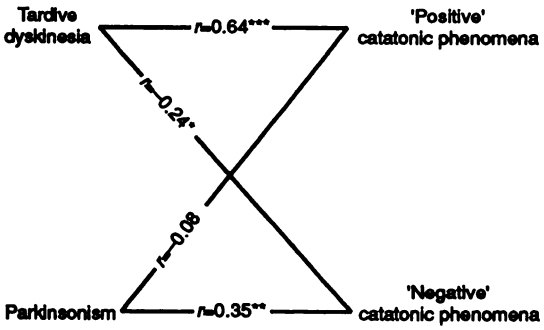


Fig. 1. Correlations between extrapyramidal and catatonic phenomena in schizophrenia (*$P<0.05$, **$P<0.01$, ***$P<0.001$).

https://doi.org/10.1192/bjp.158.3.328 Published online by Cambridge University Press

phenomena also remained significant ($r=0.20$, $P=0.04$), but that for Parkinsonism (Webster) became marginal ($r=0.17$, $P=0.07$). (It should be pointed out that 'negative' catatonic (narrow) ratings comprised only three items, all of which were not often rated as present.)

Since both tardive dyskinesia and catatonic phenomena are associated with chronicity, it is possible that their intercorrelation could merely be an artefact of their mutual correlation with this or other extraneous variables, such as severity or age. Accordingly, these were controlled for using Pearson's (parametric) and Kendall's (non-parametric) partial correlation techniques. Both methods gave an essentially similar pattern of initial correlations and significance levels as found with Spearman's method. Controlling for chronicity reduced the Pearson correlation between tardive dyskinesia and 'positive' catatonic scores on the Modified Rogers Scale from 0.58 to 0.51 ($P<0.001$) and the Kendall correlation from 0.49 to 0.48 (no significance figures derivable). The same procedures reduced the Pearson correlation between Parkinsonism and 'negative' catatonic scores from 0.29 to 0.28 ($P=0.01$) and left the Kendall correlation unchanged at 0.28. Controlling for severity or age produced no greater alterations than these and no losses of significance.

Repeating the examination on the second group of 40 patients under more rigorously blind conditions once again produced the same pattern of correlations. Total scores for extrapyramidal symptoms were significantly correlated with total catatonic scores on the Modified Rogers Scale ($r=0.49$, $P=0.001$); scores for tardive dyskinesia were significantly correlated with 'positive' catatonic scores ($r=0.57$, $P<0.001$); Parkinsonism scores were significantly correlated with 'negative' catatonic scores, in fact more highly than in the main sample ($r=0.51$, $P<0.001$). As before, the cross-correlations were insignificantly inverse. The correlations between tardive dyskinesia and 'positive' catatonic phenomena survived substituting narrow scores for the former, the latter, or both, as did that between Parkinsonism and 'negative' catatonic phenomena when the narrow Parkinsonism (Webster) score was used. As previously, when the narrow measure of negative catatonic phenomena (consisting of scores on only three items) was substituted, this correlation became insignificant.

Factor analysis of 18 items covering both extrapyramidal and catatonic disorders, carried out on the 93 ratings from the Modified Rogers Scale, produced four factors with eigenvalues above 1. One of these, however, had an eigenvalue of only 1.13; it accounted for only a small proportion of the variance (6.3%); and it failed to load on any items at a level of 0.5 or greater. This factor was therefore ignored; the remaining three are illustrated in Table 1.

Factor 1 loaded principally on 'hyperkinetic' phenomena, including both simple abnormal movements and complex mannerism/stereotypy-like movements, manneristic gait, iterations, and overactivity. There were also loadings on abruptness/rapidity of movement and exaggerated movement; a loading of 0.47 on excessive compliance/automatic obedience approached the conventionally accepted cut-off of 0.5. Factor 2, in contrast, contained predominantly 'hypokinetic' phenomena. There were heavy loadings on slow movement and slowness of gait, and also on underactivity and poor compliance. There was an unexpectedly high loading on *gegenhalten*, and that for mutism at 0.41 approached the 0.5 threshold. Factor 3 loaded highly on only two items, showing no obviously interpretable pattern. Factors 1 and 2 cumulatively accounted for nearly half the variance (32% and 17%) and both tapped an admixture of extrapyramidal and catatonic phenomena. None of the remaining factors isolated one or other class of abnormality; oblique rotation failed to achieve a convergence.

Table 1
Factor analysis of 93 ratings from the Modified Rogers Scale

| Item | Factor 1 | Factor 2 | Factor 3 |
|---|---|---|---|
| Overactivity | 0.82 | 0.00 | 0.15 |
| Exaggerated movement | 0.80 | 0.05 | -0.14 |
| Complex movements (trunk) | 0.77 | 0.31 | 0.07 |
| Abruptness/rapidity | 0.75 | -0.04 | -0.04 |
| Exaggerated gait | 0.71 | 0.00 | 0.04 |
| Complex movements (head) | 0.69 | 0.10 | 0.00 |
| Simple movements (trunk) | 0.69 | -0.02 | 0.14 |
| Simple movements (head) | 0.60 | -0.08 | 0.08 |
| Manneristic gait | 0.58 | 0.18 | 0.09 |
| Iterations | 0.52 | 0.45 | -0.43 |
| Slowness of movement | -0.35 | 0.73 | -0.07 |
| Poor compliance | -0.12 | 0.66 | 0.40 |
| Slow gait | -0.13 | 0.64 | -0.21 |
| *Gegenhalten* | 0.14 | 0.63 | 0.26 |
| Underactivity | -0.41 | 0.60 | 0.04 |
| Reduced gait | -0.49 | 0.46 | -0.28 |
| Mutism | 0.11 | 0.41 | 0.60 |
| Excessive compliance | 0.47 | 0.36 | -0.59 |
| Variance: % | 31.8 | 16.6 | 7.5 |

## Discussion

The central finding of this study was that of a clinical association between extrapyramidal side-effects and catatonic symptoms in schizophrenia, which was highly significant statistically and which survived a variety of attempts to make it disappear. On further analysis this was seen to be composed of independent associations between tardive dyskinesia and 'positive' catatonic phenomena (i.e. those distinguished by the presence of an abnormality), and between Parkinsonism and 'negative' catatonic phenomena (i.e. those featuring the absence/diminution of a normal function). The result was reinforced by a factor analysis that revealed no tendency for extrapyramidal and catatonic phenomena to separate from each other, but which instead isolated 'hyperkinetic' and 'hypokinetic' groupings of both. Finally, the association is not explicable on the basis that extrapyramidal side-effects are indiscriminately associated

https://doi.org/10.1192/bjp.158.3.328 Published online by Cambridge University Press

with all classes of schizophrenic symptoms: in an earlier study using a similar method (Mortimer *et al*, 1990), the only correlations of tardive dyskinesia or Parkinsonism other than those with catatonic symptoms, were found with negative symptoms and formal thought disorder.

Such an association between extrapyramidal and catatonic symptoms is not without precedent: lethal catatonia (Mann *et al*, 1986), neuroleptic malignant syndrome (Abbot & Loizou, 1986; Lohr & Wisniewski, 1987) and encephalitis lethargica, both untreated (von Economo, 1931) and treated (Sacks, 1983), are conditions in which they commonly coexist. At the same time, the present study's findings should not be taken to mean that all schizophrenic patients with motor symptoms will display both extrapyramidal and catatonic phenomena – the levels of correlation found make it clear that this cannot be so – but merely that such combinations are encountered more frequently than expected by chance. The clinical impression was that one substantial group of patients (mainly young and relatively acute) showed Parkinsonism or tardive dyskinesia (or both) more or less in isolation; another, smaller (acute and chronic) group showed catatonic symptoms in the absence of any marked extrapyramidal side-effects; but in the remainder (almost exclusively chronic), both kinds of abnormality appeared inextricably interwoven.

There are very few ways in which this clinical association can be accounted for. One possibility is that neuroleptic drugs, as well as causing extrapyramidal symptoms, might be able to induce catatonic phenomena as side-effects. Although not considered to be a routine complication of neuroleptic treatment, there is some evidence to suggest that this may be so in certain circumstances. On the one hand, neuroleptic administration (usually but not always to schizophrenic patients) has been documented as leading to the development of states indistinguishable from catatonic stupor (Lohr & Wisniewski, 1987). On the other, neuroleptics can cause Gilles de la Tourette's syndrome (Lees, 1985) which, if it resembles the idiopathic form of the disorder in all respects, should sometimes be accompanied by the stereotypy-like and mannerism-like phenomena of complex tics. But on present evidence these are exceptional, with only 18 cases of the former and seven of the latter having been reported (Lohr & Wisniewski, 1987). Furthermore, such a view is not easily reconciled with the presence of catatonic symptoms in schizophrenia before the introduction of neuroleptic treatment, and it predicts that they should have increased in frequency since then – a position that few would care to defend.

If catatonic symptoms are not side-effects of neuroleptic drugs, it is even more unlikely that extrapyramidal symptoms are simply manifestations of the disease process of schizophrenia. The argument that neuroleptic treatment is irrelevant to Parkinsonism in schizophrenia cannot be seriously entertained. The case for tardive dyskinesia, however, is more complicated: while there is good evidence that involuntary movements indistinguishable from tardive dyskinesia can be seen in untreated schizophrenia (Owens *et al*, 1982), it is probably only in long-stay patients that their prevalence approaches that found in treated patients. Taken overall, the evidence that neuroleptic drugs greatly promote the development of involuntary movement disorders is overwhelming (Baldessarini *et al*, 1980; Kane & Smith, 1982).

By a process of elimination, the only explanation of the present study's findings which remains feasible is one of interaction between drug and disease process. Such a suggestion has been made before (Steck, 1954; Barnes & Liddle, 1985; Rogers, 1985), and the reasoning behind it can be best illustrated with respect to tardive dyskinesia. Accepting the evidence that simple involuntary movements can be seen in untreated schizophrenia, then it is reasonable to regard them as being part of the spectrum of schizophrenic motor disorder, that is, catatonia. Neuroleptic drugs have the potential to induce involuntary movements, but this potential is only expressed in a proportion of patients who have been treated. It then becomes only a small step to suppose that those patients who are particularly prone to develop drug-induced dyskinesia will be those who already exhibit the class of symptom of which dyskinesia is a member.

The findings of the present study provide further support for Rogers (1985) 'conflict of paradigms' interpretation of schizophrenic motor disorder, which maintains that the customary distinction of neurological and psychiatric types is illusory, or relative rather than absolute. This conclusion carries a pathological corollary. If it is accepted that extrapyramidal symptoms have their basis in dysfunction of the basal ganglia, then catatonic symptoms should be the consequence of dysfunction in a closely related brain system. Just such a system in fact exists in the ventral striatal–pallidal complex (Heimer *et al*, 1982; Graybiel, 1984; Nauta & Domesick, 1984). Anatomically, the main nuclei of this show no sharp demarcation from the core nuclei of the basal ganglia; its connections also overlap with those of the striopallidal pathway at every step. In functional terms the relationship of the basal ganglia and the ventral striatal–pallidal complex appears

equally close. In animals, ventral striatal dysfunction is involved in the phenomena of hyperkinesia and stereotypy (Joyce, 1983), and in man it may be implicated in the complex and purposeful motor disorder of tics (McKenna, 1987). Finally, both the basal ganglia and ventral striatum receive innervation from the ascending dopamine pathways which can no longer be regarded as separable into striatal, limbic and cortical, but which form a single 'mesotelencephalic' dopamine system (Bjorklund & Lindvall, 1984).

**Acknowledgements**

The authors are grateful to Drs T. C. Jerram, T. E. Nelson and J. C. O'Grady for permitting them to examine their patients.

## Appendix 1

**Modified Rogers Scale**

Patient ..... Date .....
ID ..... Rater .....

0 = abnormality absent
1 = abnormality definitely present
2 = abnormality marked or pervasive

Rate all abnormalities phenomenologically, regardless of presumed basis (i.e. extrapyramidal or catatonic). Do not rate abnormalities which are questionable, subtle or only minimally present. A rating of 1 implies that the abnormality is obvious and usually of more than mild severity.

*Posture*

1. Simple abnormal posture
(generally relatively fixed) (*specify*: flexed, lordotic, twisted, tilted, other. . .)
2. Complex abnormal posture
(may be more dynamic e.g. slack, constrained, awkward; 'posturing' = 2)
3. Persistence of imposed postures
(not sustained = 1; sustained 'waxy flexibility' = 2)

*Tone and motor compliance*

4. Abnormal tone
(*specify*: increased, decreased)
5. *Gegenhalten*
('springy' resistance to passive movement which increases with increasing force)
6. *Mitgehen*
('anglepoise lamp' raising of arm in response to light pressure; do not rate if poor understanding of instruction)

*Abnormal movements: face and head*

7. Simple brief/dyskinesia-like
(*specify*: random/irregularly repetitive/rhythmical/tic-like)
8. Simple sustained/grimace-like
(e.g. spasmodic facial contortions; should not be completely fixed)
9. Complex mannerism/stereotypy-like
(usually of head, e.g. turning away, side-to-side looks, searching movements)

*Abnormal movements: trunks and limbs*

10. Simple brief/dyskinesia-like
(*specify*: random/irregularly repetitive/rhythmical/tic-like; include rocking)
11. Simple sustained/dystonia-like
(e.g. dystonic posturing of extremities, hyperpronation on arm raising, torsion movements)
12. Complex mannerism/stereotypy-like
(e.g. touching, stroking, finger play, repetitious gestures)

*Abnormal ocular movements*

13. Increased blinking
(including rapid bursts)
14. Decreased blinking
15. Eye movements
(*specify*: to-and-fro/roving/conjugate deviation)

*Purposive movement*

16. Abruptness/rapidity of spontaneous movements
(e.g. sudden gestures, acts carried out smartly, springs to attention when asked to stand)
17. Slowness/feebleness of spontaneous movements
(e.g. weak, languid, laboured)
18. Exaggerated quality to movements
(accompanied by flourishes/flurries of adventitious movements = 2)
19. Iterations of spontaneous movements
(e.g. gesture/mannerism repeated)
20. Other
(*specify*: echopraxia/blocking/ambitendence; do not rate any other abnormalities than these)

*Gait*

21. Exaggerated associated movement
(rate irrespective of 24)
22. Reduced associated movement
(rate irrespective of 24)
23. Slow/shuffling
24. Manneristic/bizarre
(may have extravagant or constrained quality or neither; do not rate merely clumsy, hunched or lordotic gaits; interpolated movements = 2)

*Speech*

25. Aprosodic
(markedly abnormal rate/volume/intonation e.g. rasping, sing-song, automaton-like; do not rate mere lack of inflection)
26. Mutism
(less than 20 words = 1, no speech = 2)
27. Indistinct/unintelligible speech
(e.g. mumbling/poor articulation/non-social speech; verbigeration = 2)

28. Other
(*specify*: echolalia/palilalia/speech mannerism; do not rate any other abnormalities than these)

*Behaviour during interview*

29. Marked overactivity
(1 = in constant motion/continual succession of mannerisms and stereotypies; 2 = approaching catatonic excitement; do not rate simple restlessness/akathisia; do not rate unless substantial)
30. Marked underactivity
(1 = sits abnormally still, inert, passive; 2 = approaching stupor. Do not rate if patient is clearly sedated/Parkinsonian; do not rate unless substantial)
31. Excessive compliance/automatic obedience
(e.g. raises both arms when asked to raise one; continues to carry out instructed actions unnecessarily; obeys instructions instantly)
32. Poor/feeble compliance
(failure to perform, carry through or maintain requested actions not due to general uncooperativeness or poor understanding; do not rate if clearly Parkinsonian)
33. Other
(*specify*: negativism/hypermetamorphosis; do not rate any other abnormalities than these)

*Reported behaviour*

34. Overactive
(e.g. restless, paces, wanders all day)
35. Underactive
(e.g. sits in same place all day, has to be brought to meal table)
36. Other
(e.g. adopts postures, performs repetitive acts, engages in rituals)

## Appendix 2

### Guidelines for rating Modified Rogers Scale catatonic phenomena

*Complex abnormal posture*. Mere ungainliness or slouching should not be rated. Rating 1, examples are: assuming obviously abnormal hunched, constrained, 'closed' or alternatively exaggeratedly slack, over-relaxed positions when sitting; hugging sides, twisting legs round each other, sitting with torso forward but legs to one side in extremely uncomfortable way. Rating 2, examples are: while sitting, repeatedly hunching forward and rocking; while standing or walking, striking a succession of poses.

*Persistence of imposed postures*. Rating 1, tendency to retain limb positions passively imposed during testing for at least several seconds; this should be observed more than once. Rating 2, typical waxy flexibility.

*Gegenhalten*. Resistance to passive movement which increases with the force exerted; typically has a 'springy' quality and appears automatic rather than wilful. May be restricted to just one muscle group, e.g. the neck.

*Mitgehen*. 'Anglepoise lamp' arm raising in response to light pressure, in the presence of an apparent grasp of the need to resist; should be demonstrable repeatedly. Severity of rating depends on the rapidity and apparent wish to anticipate the movement; other similar tests such as tipping the patient backwards by lightly pressing on the forehead or turning him/her round by light pressure on an outstretched arm can also be used.

*Complex stereotypy/mannerism-like movements*. More stereotypy-like examples are: rubbing the thumb over the forefinger, other kinds of finger play, touching, rubbing, stroking and patting various parts of the body especially the face, and repeatedly turning the head away from the examiner, looking round distractedly throughout the interview, twisting one arm up behind the back while walking, repeatedly rising from chair and approaching examiner. More mannerism-like examples are holding arms in an unnatural crooked way, holding an arm out in a meaningless gesture, keeping one arm tucked under armpit.

*Iterations*. Gestures or mannerisms repeated over short space of time, example being touching face then repeating this several times; manneristically smoothing hair, then repeating this with increasing force until striking head; touching ring finger on one hand (while alluding to ring being stolen), then doing the same on the other hand, then repeating the whole sequence.

*Echopraxia*. Incomplete copying movements should not be rated, and exercise judgement as to whether patient is just trying to be helpful. As well as being merely copied, movements may be modified or amplified, for example, smoothing of hair substituted for examiner's scratching of head, echopraxic chest patting progressively exaggerated until patient is pulling at his shirt.

*Blocking/ambitendence*. In practice not easy to distinguish from one another. Examples are: freezing in the act of sitting forward and remaining motionless, grasping the arms of the chair for nearly a minute; extending arm when examiner's is proffered, then halting in mid-action and moving arm to one side; while walking, stopping, half-turning back, then continuing.

*Manneristic/bizarre gait*. Merely clumsy or lumbering gaits should not be rated, and gait should be idiosyncratic rather than hunched, lordotic, shuffling, etc. Examples are: constrained, mincing, over-precise, or alternatively extravagant, overelaborate, featuring interpolated movements such as sidesteps and bowing, and also bizarre crab-like, crouching or anthropoid gaits, and those with multiple, not easily described abnormalities.

*Aprosodic speech*. Simply unvarying, harsh or stereotyped inflections should not be rated unless marked. Examples are: unnaturally loud, strident, high-pitched, or alternatively feeble, whispering or completely monotonous intonations. Occasionally also automaton-like, sing-song, rasping, strangled, or warbling inflections.

*Overactivity/excitement*. Typically bizarre rather than resembling simple restlessness; akathisia should be excluded where suspected. Rating 1, continual motor unrest. Examples are: crossing and uncrossing legs, looking round, half rising from the chair; executing unending series of manneristic actions, touching body, then clasping hands, then gripping the chair arm, etc. Rating 2, in more or less

constant motion, incessantly performing pointless actions which are reiterated, elaborated and transformed into one another, for example touching cardigan, then moving hands up and down the edges, then unbuttoning it and buttoning it up again, followed by breaking off interview to clamber over the tables and chairs on the ward. Also includes full-blown excitement, for example, a patient who moved round and round the ward striking an endless series of quasi-symbolic poses.

*Underactivity/stupor*. Some degree of abnormality is commonly observed and should not be rated unless very noticeable. Rating 1, sitting abnormally still throughout the interview with hardly any postural shifts; slumped in chair; very passive. Rating 2, marked hypokinesia, generally with striking absence of postural adjustments, for example, sitting perched on chair in same position throughout interview, not turning head when addressed from different direction; always sitting in same place on ward with arms held in praying position. Also includes full-blown stupor if encountered.

*Excessive compliance/automatic obedience*. May take form of exaggerated co-operation with instructed movements, for example: when asked to lift a finger, whole arm raised; when asked to lower arm, done so smartly that it overshoots backwards; when arm reached for, whole body leant forward and turned toward examiner; holding out both hands when examiner's offered for shaking. Alternatively, spontaneous continuation of actions, for example: flapping arms when asked to drop them to sides, actively continuing passive arm movements during examination for tone. Occasionally, complying with all requests to an extraordinary degree, for example: patient who screwed up eyes when asked to close them; peered intently in caricatured way when asked to look out of window; when asked to keep head up while walking, proceeded across the room with neck hyperextended.

*Poor/feeble compliance*. Inability to perform requested actions not explicable in terms of poor understanding, general uncooperativeness, blocking/ambitendence, or Parkinsonism; often has a bizarre quality. Examples are: when raising arm, movement gradually dies away; carries out most instructions promptly but fails to comply with some; cannot seem to maintain arms outstretched; when asked to hold out arms only seems able to do so in half-hearted, crooked way; when asked to raise a finger, after some delay lifts thumb.

*Negativism*. Should always reflect concrete instances rather than indefinable attitude. Examples are: pulling arm violently away whenever the examiner reaches for it, holding breath when asked to breathe deeply, shutting eyes tightly when approached with an ophthalmoscope, jumping up when asked to lie down, taking off socks when told to put shoes on; getting up from customary reclining position and walking away whenever approached by examiner. Occasionally, domination of entire behaviour by bizarre contrariness, for example a normally quiet patient who met attempts to examine him with immediate struggling and vilification; leant backwards when pulled forwards; refused to stand up, then refused to sit down again.

*Hypermetamorphosis*. Typically only seen in setting of marked overactivity, for example: attention repeatedly drawn by specks, bits of fluff, etc., on the floor, which are reached for and scrutinised; randomly approaching various objects including wastebasket, rummaging in it, extracting apple core and eating it.

## References


ABBOT, R. J. & LOIZOU, L. (1986) Neuroleptic malignant syndrome. *British Journal of Psychiatry*, **148**, 47–51.

AMERICAN PSYCHIATRIC ASSOCIATION (1980) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn) (DSM-III). Washington, DC: APA.

ANDREASEN, N. C. (1987) *Comprehensive Assessment of Symptoms and History*. Iowa City: University of Iowa College of Medicine.

ATAKAN, Z. & COOPER, J. E. (1989) Behavioural Observation Schedule (BOS): PIRS (2nd edn). *British Journal of Psychiatry*, **155** (suppl. 7), 78–88.

BALDESSARINI, R. J., COLE, J. O., DAVIS, J. M., *et al* (1980) Summary of the task force report of the American Psychiatric Association on late neurological effects of antipsychotic drugs. *American Journal of Psychiatry*, **137**, 1163–1172.

BARNES, T. R. E. & BRAUDE, W. F. (1985) Akathisia variants and tardive dyskinesia. *Archives of General Psychiatry*, **42**, 874–878.

—— & LIDDLE, P. F. (1985) Tardive dyskinesia: implications for schizophrenia? In *Schizophrenia: New Pharmacological and Clinical developments* (eds A. A. Schiff, M. Roth & H. L. Freeman). London: Royal Society of Medicine.

BJORKLUND, A. & LINDVALL, O. (1984) Dopamine-containing systems in the CNS. In *Handbook of Chemical Neuroanatomy, Vol. 2* (eds A. Bjorklund & T. Hokfelt). Amsterdam: Elsevier.

BLEULER, E. (1911) *Dementia Praecox, or the Group of Schizophrenias* (trans. J. Zinkin; 1966). New York: International Universities Press.

BROWNRIGG, A. E. (1901) Kraepelin's clinical picture of katatonia. *American Journal of Insanity*, **57**, 455–476.

DELAY, J. & DENIKER, P. (1968) Drug-induced extrapyramidal syndromes. In *Handbook of Clinical Neurology, Volume 6: Diseases of the Basal Ganglia* (eds P. J. Vinken & G. W. Bruyn). New York: Wiley; Amsterdam: North Holland.

DENIKER, P. (1960) Experimental neurological syndromes and the new drug therapies in psychiatry. *Comprehensive Psychiatry*, **1**, 92–102.

ENDICOTT, J., SPITZER, R. C., FLIESS, J. L., *et al* (1976) The global assessment scale. *Archives of General Psychiatry*, **33**, 766–771.

FARRAN-RIDGE, C. (1926) Some symptoms referrable to the basal ganglia occurring in dementia praecox and epidemic encephalitis. *Journal of Mental Science*, **72**, 513–523.

GRAYBIEL, A. M. (1984) Neurochemically specified subsystems in the basal ganglia. In *Functions of the Basal Ganglia, CIBA Foundation Symposium 107* (eds D. Evered & M. O'Connor). London: Pitman.

HALL, J. N. (1980) Ward rating scales for long stay patients. *Psychological Medicine*, **10**, 277–288.

HAMILTON, M. (1984) *Fish's Schizophrenia* (3rd edn). Bristol: John Wright.

HEIMER, L., SWITZER, R. D. & VAN HOESEN, G. W. (1982) Ventral striatum and ventral pallidum: components of the motor system? *Trends in Neurosciences*, **5**, 83–87.

JASPERS, K. (1959) *General Psychopathology* (trans. J. Hoenig & M. W. Hamilton; 1963). Manchester: Manchester University Press.

JONES, M. & HUNTER, R. (1969) Abnormal movements in patients with chronic psychiatric illness. In *Psychotropic Drugs and Dysfunctions of the Basal Ganglia: A Multidisciplinary Workshop*, publication no. 1938 (eds G. E. Crane & R. Gardner). Washington, DC: US Public Health Service.

JOYCE, J. N. (1983) Multiple dopamine receptors and behavior. *Neuroscience and Behavioural Reviews*, **7**, 227–256.

KAHLBAUM, K. (1874) *Catatonia* (trans. Y. Levij & T. Priden; 1973). Baltimore: Johns Hopkins University Press.

KANE, J. M. & SMITH, J. M. (1982) Tardive dyskinesia: prevalence and risk factors, 1959–1979. *Archives of General Psychiatry*, **39**, 473–481.

KLEIST, K. (1943) Die Katatonien. *Nervenarzt*, **16**, 1–10.

—— (1960) Schizophrenic symptoms and cerebral pathology. *Journal of Mental Science*, **106**, 246–255.

KRAEPELIN, E. (1907) *Clinical Psychiatry* (7th edn) (trans. A. R. Diefendorf; 1915). New York: MacMillan.

—— (1913) *Dementia Praecox and Paraphrenia* (trans. R. M. Barclay; 1919). Edinburgh: Livingstone.

LEES, A. J. (1985) *Tics and Related Disorders*. Edinburgh: Churchill Livingstone.

LOHR, J. B. & WISNIEWSKI, A. A. (1987) *Movement Disorders: A Neuropsychiatric Approach*. Chichester: Wiley.

MANN, S. C., CAROFF, S. N., BLEIER, H. R., *et al* (1986) Lethal catatonia. *American Journal of Psychiatry*, **143**, 1374–1381.

MANSCHRECK, T. C., MAHER, B. A., RUCKLOS, M. E., *et al* (1982) Disturbed voluntary motor activity in schizophrenic disorder. *Psychological Medicine*, **12**, 73–84.

MARSDEN, C. D. (1982) Motor disorders in schizophrenia. *Psychological Medicine*, **12**, 13–15.

——, TARSY, D. & BALDESSARINI, R. J. (1975) Spontaneous and drug-induced movement disorders in psychotic patients. In *Psychiatric Aspects of Neurologic disease* (eds D. F. Benson & D. Blumer). New York: Grune & Stratton.

—— & JENNER, P. (1980) The pathophysiology of extrapyramidal side-effects of neuroleptic drugs. *Psychological Medicine*, **10**, 55–72.

——, MINDHAM, R. H. S. & MACKAY, A. V. P. (1986) Extrapyramidal movement disorders produced by antipsychotic drugs. In *Psychopharmacology and Treatment of Schizophrenia* (eds P. B. Bradley & S. R. Hirsch). Oxford: Oxford University Press.

MCKENNA, P. J. (1987) Pathology, phenomenology and the dopamine hypothesis of schizophrenia. *British Journal of Psychiatry*, **151**, 288–301.

——, MORTIMER, A. M. & LUND, C. E. (1988) The motor disorders of severe psychiatric illness: a conflict of paradigms (letter). *British Journal of Psychiatry*, **152**, 863–864.

——, TAMLYN, D., LUND, C. E., *et al* (1990) Amnesic syndrome in schizophrenia. *Psychological Medicine*, **20**, 967–972.

MORTIMER, A. M., LUND, C. E. & MCKENNA, P. J. (1990) The positive:negative dichotomy in schizophrenia. *British Journal of Psychiatry*, **157**, 41–49.

NAUTA, W. J. H. & DOMESICK, V. B. (1984) Afferent and efferent relationships of the basal ganglia. In *Functions of the Basal Ganglia, CIBA Foundation Symposium 107* (eds D. Evered & M. O'Connor). London: Pitman.

OWENS, D. G. C. (1986) Practical problems in evaluating involuntary motor activity in schizophrenic patients. In *Working Methods in Neuropsychopharmacology* (eds M. H. Joseph & J. L. Waddington). Manchester: Manchester University Press.

—— (1990) Dystonia – a psychiatric pitfall. *British Journal of Psychiatry*, **156**, 620–634.

——, JOHNSTONE, E. C. & FRITH, C. D. (1982) Spontaneous involuntary disorders of movement: their prevalence, severity and distribution in chronic schizophrenics with and without treatment with neuroleptics. *Archives of General Psychiatry*, **39**, 452–481.

PFOHL, B. & WINOKUR, G. (1982) The evolution of symptoms in institutionalised hebephrenic/catatonic schizophrenics. *British Journal of Psychiatry*, **141**, 567–572.

REITER, P. J. (1926) Extrapyramidal motor disturbances in dementia praecox. *Acta Psychiatrica et Neurologica Scandinavica*, **1**, 287–304.

ROGERS, D. (1985) The motor disorders of severe psychiatric illness: a conflict of paradigms. *British Journal of Psychiatry*, **147**, 221–232.

SACKS, O. (1983) *Awakenings*. New York: Dutton.

SIMPSON, G. M. & ANGUS, J. W. S. (1970) Drug induced extrapyramidal disorders. *Acta Psychiatrica Scandinavica* (suppl. 212), 1–58.

——, LEE, J. H., ZOUBOK, B., *et al* (1979) A rating scale for tardive dyskinesia. *Psychopharmacology*, **64**, 171–179.

SPITZER, R. L. & ENDICOTT, J. (1978) *Schedule for Affective Disorders and Schizophrenia*. New York: Biometric Research, New York State Psychiatric Institute.

STECK, H. (1926/27) Les syndromes extrapyramidaux dans les maladies mentales. *Archives Suisses de Neurologie et Psychiatrie*, **19**, 195–233; **20**, 92–136.

—— (1954) Le syndrome extrapyramidal et diencephalique au cours des traitements au largactil et au serpasil. *Annales Medico-psychologique*, **112**, 737–743.

VON ECONOMO, C. (1931) *Encephalitis Lethargica: Its Sequelae and Treatment* (trans. K. O. Newman). London: Oxford University Press/Humphrey Milford.

WEBSTER, D. D. (1968) Clinical analysis of the disability in Parkinson's disease. *Modern Treatment*, **5**, 257–282.

WING, J. K., COOPER, J. E. & SARTORIUS, N. (1974) *Measurement and Classification of Psychiatric Symptoms*. Cambridge: Cambridge University Press.

WYKES, T. & STURT, E. (1986) The measurement of social behaviour in psychiatric patients: an assessment of the reliability and validity of the SBS. *British Journal of Psychiatry*, **148**, 1–11.

C. E. Lund, MB, ChB, MRCPsych, *Senior Registrar in Psychiatry, High Royds Hospital, Menston, Ilkley LS21 2LY*; A. M. Mortimer, MB, ChB, MRCPsych, *Consultant Psychiatrist, St Luke's Hospital, Crosland Moor, Huddersfield HD4 5RQ*; D. Rogers, MA, MRCP, MRCPsych, *Consultant Neuropsychiatrist, Burden Neurological Hospital, Stoke Lane, Stapleton, Bristol BS16 1QT*; C. A. Biggins, MB, ChB, *Research Registrar, Academic Department of Psychiatry, University of Leeds, 15 Hyde Terrace, Leeds LS2 9LT*; *P. J. McKenna, MA, MB, ChB, MRCPsych, *Consultant Psychiatrist, Fulbourn Hospital, Cambridge CB1 5EF*

*Correspondence