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Commentary on the Modified Rogers Scale and the 'Conflict of Paradigms' Hypothesis

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The variety and complexity of schizophrenic movement disorders renders them difficult to assess. In particular, the complex disorders of motility that appear to reflect disturbance of the will demand an evaluation of the purpose of the movement. The difficulties of assessment are compounded by the fact that the antipsychotic drugs used to treat the illness can produce motor disorders that resemble the disorders intrinsic to the illness. In advancing his 'conflict of paradigms' hypothesis, Rogers (1985) implied that emphasis on the distinction between intrinsic and drug-induced movement disorders might be misplaced because of overlap not only in the observable characteristics of these disorders, but also in the underlying neuropathology.

If there is a substantial overlap between intrinsic and drug-induced movement disorders, attempts to devise clinical rating scales based on distinguishing these types of disorders are doomed to failure. Clearly, a non-prejudicial assessment that makes no attempt to make such a distinction would be more sensible. McKenna *et al* (this issue, pp. 328–336) use ratings made according to a modified version of the non-prejudicial rating scale developed by Rogers (1985) to test his hypothesis that there is an association between intrinsic and drug-induced movement disorders. They have identified a group of movement disorders that are generally regarded as not being drug induced and hence might be considered intrinsic (that is, catatonic) phenomena. They divide the catatonic phenomena into negative phenomena, which reflect a deficit of activity, and positive phenomena, which entail abnormal activity. They then demonstrate a correlation between the occurrence of negative catatonic phenomena and Parkinsonism, and a separate correlation between positive catatonic phenomena and dyskinetic movements characteristic of tardive dyskinesia.

The relationship between positive catatonic symptoms and tardive dyskinesia is the more convincing finding. Ironically, this is probably because there is a relatively clear-cut difference in character between the complex disorders of motility that are regarded as characteristic of catatonia and the simpler motor disorders regarded as characteristic of tardive dyskinesia. Hence McKenna *et al* were able to devise a satisfactory 'narrow', positive catatonia subscale, which comprised items that most clinicians would regard as intrinsic to schizophrenia. The finding of a strong correlation between the 'narrow' positive catatonia score and the tardive dyskinesia score is convincing evidence for Rogers' hypothesis.

Conversely, in the case of the negative movement disorders, the situation is less clear cut. The most prevalent negative catatonic item, marked underactivity, is similar in character to the hypokinesia of Parkinsonism, and hence McKenna *et al* found it difficult to define an adequate 'narrow' negative catatonia scale. They did define a narrow negative catatonia score obtained by adding scores for ambitendence, poor/feeble compliance and mutism, but these items had a relatively low prevalence and the correlation between this score and a 'narrow' score for Parkinsonism (which excluded potentially ambiguous items such as reduced arm swing) was not significant. There was a statistically significant correlation of moderate strength ($r=0.35$, $P=0.001$) between a score for negative catatonia broadened to include marked underactivity, and a broad Parkinsonism score. Thus it appears that the correlation between negative catatonia and Parkinsonism reported by McKenna *et al* is mediated almost entirely by the shared phenomenon of underactivity.

The assessment of underactivity in schizophrenia

The guidelines of the Rogers scale for rating underactivity (item 30) include descriptions of behaviour such as "sits abnormally still; inert; passive", which is relatively common in Parkinson's disease. Unfortunately, there is a contradiction between the general instruction that all abnormalities should be rated without regard to whether the presumed basis is extrapyramidal or catatonic, and the specific instruction for item 30 that underactivity associated with Parkinsonism or sedation should be excluded from the rating.

Perhaps the authors' justification for this departure from Rogers' intention that the scale should be non-prejudicial, is that they regard item 30 as a measure of underactivity arising from impaired volition.

Volition is essentially a subjective experience and is difficult to assess directly. In a study of the subjective experience of deficits in schizophrenia, Liddle & Barnes (1988) found that there was virtually no correlation between the patients' subjective experience of impaired drive/energy and observed psychomotor retardation. In the absence of a satisfactory means of assessing volition directly, it is preferable to base the rating of underactivity purely on observation of the amount of activity. Furthermore, the fact that the results obtained by McKenna *et al* imply a significant correlation between their ratings of marked underactivity and Parkinsonism, despite the instruction to exclude underactivity associated with clear Parkinsonism, indicates that it is in practice difficult to distinguish Parkinsonian underactivity from underactivity from other causes.

While the direct clinical assessment of decreased volition in schizophrenia is difficult, recent developments in functional brain imaging using positron emission tomography offer the possibility of direct measurement of the brain activity associated with self-generated actions. According to contemporary theories concerning the generation of action, the pre-frontal cortex plays an important role in the initiation of activity (Shallice, 1988). In a comparison of regional cerebral perfusion in normal subjects during the performance of matched tasks which differed only in whether the plan for action was generated internally by the subject or externally by the experimenter, Frith *et al* (paper in preparation) have found that the internal generation of the plan for action was associated with an increase in perfusion in parts of the pre-frontal cortex.

Brain malfunction underlying impaired initiation of activity

The difficulties in the clinical assessment of underactivity and decreased volition indicate the need for caution in accepting that the results of McKenna *et al* support Rogers' hypothesis with regard to negative disorders of movement and volition. Nonetheless, there is evidence from other sources suggesting that underactivity intrinsic to schizophrenia reflects a brain malfunction similar to that associated with Parkinsonian underactivity.

McKenna and colleagues have reported elsewhere an analysis of the relation between catatonia scores and other aspects of schizophrenic psychopathology (Mortimer *et al*, 1990). They found a correlation between negative catatonia and negative schizophrenic symptoms that was maintained even after correcting for the effects of chronicity and severity of illness. This is consistent with my own finding that decreased spontaneous movement together with poverty of speech and flatness of affect constitute a distinguishable schizophrenic syndrome, which I have designated 'the psychomotor poverty syndrome' (Liddle, 1987). This syndrome is associated with decreased cerebral perfusion in the left pre-frontal cortex (Liddle *et al*, 1990) and with impairment in neuropsychological tests of frontal lobe function (this issue, pp. 340–345). In addition, Lindstrom (1985) has demonstrated that a very similar group of schizophrenic symptoms is associated with low levels of the dopamine metabolite homovanillic acid (HVA) in the cerebrospinal fluid.

There is substantial evidence that a psychomotor poverty syndrome characterised by decreased initiation of activity, decreased HVA levels, and impaired performance on frontal lobe tests, can occur not only in schizophrenia, but in other neuropsychiatric conditions, including Parkinson's disease, Alzheimer's disease and depression (Wolfe *et al*, 1990; Van Praag *et al*, 1990). This evidence suggests that a common neuropathological process resulting in underactivity and impaired volition can occur in these various conditions.

Conclusion

The findings of McKenna *et al*, together with additional evidence suggesting that impaired ability to initiate activity can reflect a neuropathological process that occurs in schizophrenia, Parkinson's disease and other neuropsychiatric conditions, provide good grounds for accepting that there are common elements in the neuropathology of movement disorders intrinsic to schizophrenia and movement disorders induced by antipsychotic medication. Therefore, the non-prejudicial approach to the assessment of these disorders advocated by Rogers is well justified.

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