level, 17 subjects per group are required to obtain a significant effect with 80% power in the detection of an effect size 1 (group means separated by one standard deviation) (Meredith, 1967).

(v) Designs both of change and of final evaluation have advantages and disadvantages, so that both can be considered valid (Cronbach & Furby, 1970).

(vi) Articles like that of White (1979) are useful both in statistics seminars and in the planning of a clinical trial, but in the latter it is necessary to take into account other considerations, like those of Kraemer (1981), more removed from "research work that essentially exists only in textbooks" but which help us in the "real world of psychiatric clinical research".

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References

BONNEAU, C. A. (1960) The effects of violation of assumptions underlying the *t*-test. *Psychological Bulletin*, **57**, 49–64.

CRONBACH, L. E. & FURBY, L. (1970) How we should measure change or should we? *Psychological Bulletin*, **74**, 68–80.

KRAEMER, H. C. (1981) Coping strategies in psychiatric clinical research. *Journal of Consulting and Clinical Psychology*, **49**, 309–319.

MEREDITH, W. M. (1967) *Basic Mathematical and Statistical Tables for Psychology and Education*. New York: McGraw Hill.

WHITE, S. (1979) Statistical errors in papers in the British Journal of Psychiatry. *British Journal of Psychiatry*, **135**, 336–342.

The Motor Disorders of Severe Psychiatric Illness: A Conflict of Paradigms

SIR: After examination of 100 chronically ill inpatients, Rogers (*Journal*, September 1985, **147**, 221–232) concluded that the traditional separation of their motor abnormalities into 'neurological' and 'psychiatric' was fruitless; in particular, there was no sharp division between the extrapyramidal signs of dyskinesia and parkinsonism on the one hand and the catatonic motor disorders of psychosis on the other. Choreic, athetotic, and dyskinetic signs could be demonstrated in untreated schizophrenic patients, and phenomenologically related abnormalities like tics and mannerisms had acquired dual terminologies. One of us (McKenna, 1987) argued further that this apparent continuum between involuntary movement disorders and motor catatonic phenomena might be consistent with a ventral striatal contribution to a basal ganglia dysfunction.

In a recent study of the relationship of negative symptoms to various types of motor disorder (McKenna *et al*, 1988), we unexpectedly obtained what we believe to be preliminary clinical evidence for such a position. Eighty patients meeting DSM–III/RDC criteria for schizophrenia and encompassing all grades of chronicity and severity were rated for negative symptoms, and also for tardive dyskinesia and parkinsonism using established scales (Simpson & Angus, 1970; Simpson *et al*, 1979). General motor disorder was measured using the scale developed by Rogers, modified slightly to incorporate a measure of severity. This scale allows a detailed assessment of abnormal motor behaviour, from simple abnormal movements to complex disturbances in overall behaviour, in a way which does not pre-empt their designation as neurological or psychiatric. It also permits a separation of schizophrenic motor phenomena into 'productive' (distinguished by their presence) and 'deficit' (distinguished by the absence/diminution of normal function) analogous to the positive/negative dichotomy, this being a particular focus of interest of the study.

During analysis of correlations using Spearman's non-parametric correlation coefficient we observed a striking pattern of inter-correlations among the various motor disorder ratings. In particular, tardive dyskinesia total scores correlated highly significantly with Rogers' 'productive' motor disorder scores ($r = 0.68$, $P < 0.001$), but there was no correlation with Rogers' 'deficit' scores ($r = 0.11$, NS). A mirror image pattern of correlations was seen between parkinsonism scores and Rogers' 'productive' ($r = -0.11$, NS) and 'deficit' ($r = 0.47$, $P < 0.001$) scores. The significance of these correlations persisted essentially unchanged even when items on the Rogers' scale which represented or might have been confused with tardive dyskinesia or parkinsonism were removed from consideration, leaving more purely catatonic 'productive' and 'deficit' scores.

This patterning of clinical associations is difficult to explain on the traditional basis that extrapyramidal signs and catatonic phenomena are entirely separate domains of pathology. On the other hand, it is just what would be predicted on Rogers' 'conflict of paradigms' view. If extrapyramidal and catatonic phenomena are merely points along a continuum of motor abnormality, then their frequent co-occurrence would be anticipated. Our findings also point to an extension of the concept of hyperkinetic and hypokinetic motor abnormalities beyond dyskinesia and parkinsonism, a finding which fits well with their postulated basis in a combined ventral striatal:basal ganglia dysfunction. These results can only be considered preliminary, as the relevant ratings were not made independently of one another;

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we are currently attempting to replicate the findings in more detail and under blind conditions.

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References

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

——, Lund, C. E. & Mortimer, A. M. (1988) Negative symptoms: relationship to other schizophrenic symptom classes. *British Journal of Psychiatry*, (in press).

Simpson, G. M, Lee, J. H., Zoubok, B. & Gardos, G. (1979) A rating scale for tardive dyskinesia. *Psychopharmacology*, **64**, 171–179.

—— & Angus, J. W. S. (1970) A rating scale for extrapyramidal side effects. *Acta Psychiatrica Scandinavica*, **Suppl. 212**, 11–19.

Quinine Pychosis

Sir: We report a case in which a psychotic illness may have been precipitated by exposure to quinine.

Case report: A 36-year-old female was admitted under Section 2 of the Mental Health Act with a three-day history of persecutory delusions and increasingly disturbed behaviour. She had no previous psychiatric history. She had not taken any medication but admitted to the possibility that she may have taken homeopathic doses of quinine.

On admission she was carrying a crucifix and praying aloud. She appeared agitated and suspicious, keeping her distance, and behaving aggressively if approached. She refused to answer questions, and her talk was rapid and rambling with outbursts of shouting. She expressed delusions of reference, and claimed that she had uncovered a drugs ring involving her employer. She did not appear to have any disorder of perception and there was no clouding of consciousness. A provisional diagnosis was made of acute psychosis – cause unknown: she was treated with parenteral chlorpromazine (100 mg) and diazepam (17 mg). She received no other medication, and within 36 hours of admission mental state examination had become essentially normal. Her relatives confirmed that she was by then her usual self.

Physical examination was normal. Investigations that were reported as normal included full blood count, plasma urea and electrolytes, and tests of liver, renal, and thyroid function. Blood glucose was 3.40 $mmol^{-1}$. However, a routine drug screen of urine revealed the presence of quinine and its metabolites: quantitative assay was not possible.

At follow-up 3 months later she remained well and the final diagnosis was therefore acute psychosis probably secondary to quinine ingestion.

While it is difficult to establish direct cause and effect in these conditions, the clinical picture, time course, and rapid resolution with very little treatment in this case were typical of the 'symptomatic' psychoses (Lishman, 1987; Granville-Grossman, 1971) and no other psychological or physical precipitants could be identified.

Quinine is the optical isomer of quinidine, which has itself recently been reported as causing a transient psychotic state in two cases (Deleu & Schmedding, 1987). Quinine is used mainly as an anti-malarial drug and quinidine in the treatment of cardiac arrythmias, but both are apparently also used in homeopathic medicine. The main side-effects of both drugs are described as 'cinchonism' and include nausea, tinnitus, and blurred vision; these are usually dose-related, but hypersensitivity reactions to smaller doses do occur (Dukes, 1984).

The mechanism for any possible effect is unclear. Deleu & Schmedding (1987) suggested that it may be an idiosyncratic reaction, but this does not really explain any relationship. In view of their two cases and our findings we suggest that quinine and quinidine should be added to the list of possible precipitants of symptomatic psychoses and that they should be considered in the differential diagnosis of acute psychotic reactions. The urine assay is relatively simple and may be carried out as part of a routine drug screen.

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References

Deleu, D. & Schmedding, E. (1987) Acute psychosis as idiosyncratic reaction to quinidine: report of two cases. *British Medical Journal*, **294**, 1001.

Dukes, M. N. G. (ed.) (1984) *Meyler's Side-Effects of Drugs* (10th edn). Amsterdam: Elsevier.

Granville-Grossman, K. (1971) Symptomatic Mental Disorders. In *Recent Advances in Clinical Psychiatry*. London: Churchill.

Lishman, W. A. (1987) *Organic Psychiatry – the Psychological Consequences of Cerebral Disorder* (2nd edn), p 548. London: Blackwell Scientific.

Bulimia Nervosa in an Atypical Setting: Case Report from Nigeria

Sir: Eating disorders (bulimia nervosa and anorexia nervosa) have been proposed as culture-bound syndromes occurring in Western and rapidly westernising cultures (Swartz, 1985; Prince & Tchengh-Laroche, 1987; Selvini Palazzoli, 1985). Until recently, the typical profile of sufferers was of young, middle class, female Caucasians. Selvini Palazzoli has

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