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

also observed that "in proportion as food becomes abundant, so each person is obliged to be thin" and fad becomes an instrument for expressing distress within family systems. Cassidy (1982) even suggests protein-energy malnutrition or kwashiorkor as the culture-bound eating disorder typical for West Africa and the Third World. Cultural factors may even selectively encourage the presentation of the more life-threatening anorexia nervosa (e.g. Gregory & Buchan, 1984) rather than bulimia nervosa which has not been reported so far among Africans.

Case report: A 24-year-old single professional woman from Southern Nigeria first presented socially to the second author (SMN), to whom she complained of prolonged impulsive overeating which had recently worsened. She claimed to have often eaten bread loaves enough for 6 persons. At times she also binged on biscuits. Binges had recently been followed by severe abdominal pain and spontaneous vomiting. Relief so provided was often overtaken by feelings of disgust, sadness, and guilt. Other weight-related habits included jogging and fasting. She had at times gained up to 3 kg, but at time of presentation had lost about 1 kg.

Background information revealed that patient was a twin of middle-class background. The mother was described as emotionally cold. Family experience was characterised by the stresses of upward social mobility as well as changes of location. For example, the family had moved five times before her eighth birthday, after which they all travelled to a Western country. Having lived and schooled in that country for 5 years she returned with her family to go to secondary school and on to university and professional studies. From early life, therefore, there seemed little opportunity to form lasting attachment to people, places, or values. One characteristic pattern was a preference for older, non-Nigerian or mixed race Nigerian females, the other was a definite difficulty in heterosexual relationships.

The circumstances surrounding the consultation were very instructive. The initiation of contact with SMN fitted her characteristic pattern. In fact, that contact seemed to have been used because SMN had social contacts with another lady to whom the patient had an unusually strong emotional attachment but who had left the country. The patient was actually going through complicated separation/grief reactions, since she was about to emigrate to another continent where she would be near this other woman. The consultations (four in all) enabled the patient to meaningfully appraise her feelings in the light of her impending emigration, unusually strong attachment to another female, and the relationship of her emotional turmoil to the worsening of overeating.

The authors suggest that where, as seems to be the case in Gregory & Buchan (1984), bulima nervosa or anorexia nervosa is found in the African setting, the patients would have become deculturated in addition to the usually reported difficulties in attachment, personal and sexual identity clearly elicited in this case.

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References

CASSIDY, C. M. (1982) Protein-energy malnutrition as a culture bound syndrome. *Culture, Medicine and Psychiatry*, **6**, 325–345.

GREGORY, L. D. & BUCHAN, T. (1984) Anorexia nervosa in a Black Zimbabwean. *British Journal of Psychiatry*, **145**, 326–330.

PRINCE, R. & TCHENGH-LAROCHE, F. (1987) Culture-bound syndromes and international disease classifications. *Culture, Medicine and Psychiatry*, **11**, 3–19.

SELVINI PALAZZOLI, M. (1985) Anorexia nervosa; a syndrome of the affluent society. *Transcultural Psychiatry Research Review*, **22**, 199–204.

SWARTZ, L. (1985) Anorexia nervosa as a culture-bound syndrome. *Transcultural Psychiatry Research Review*, **22**, 205–207.

Catatonia in a 90-Year-Old Patient After Depot Pipothiazine Injection

SIR: Extrapyramidal side-effects are well recognised in patients receiving phenothiazine medication (Gedenberg & Mandel, 1977). These side-effects are less common with drugs containing piperidine rings in their chains (Riley *et al*, 1976). We report here the case of a patient who developed a catatonic reaction while on neuroleptic therapy.

Case report: A 90-year-old patient with senile dementia was admitted to hospital for holiday relief. No other psychiatric or physical abnormalities were noted. There was no previous psychiatric illness known. She was receiving thioridazine (12.5 mg b.d.) and nitrazepam (12.5 mg) upon admission, and this was continued. After admission she became agitated and was given 25 mg of pipothiazine (piportil depot) intramuscularly. Forty-eight hours later she began refusing food and fluids, and five days later was admitted to an acute medical unit in view of the risk of dehydration.

On examination, she was only mildly dehydrated and there were no neurological abnormalities other than her dementia. Biochemically, her renal and hepatic function were normal. She was continued on her oral medication and oral intake encouraged.

The next day (the eighth day after her injection) she was found to be unrousable unless painfully stimulated. There were no localising neurological signs, but she did exhibit waxy flexibility of her limbs and catatonic posturing. There was no evidence of tardive dyskinesia or other extrapyramidal change. Body temperature remained normal. The