the columns

correspondence

Catatonia and NMS

Sir: The grand rounds report of catatonia by Carey *et al* (*Psychiatric Bulletin*, February 2002, **26**, 68–70) is a useful reminder that our knowledge of catatonia has progressed since its 19th-century delineations. However, the report only partially reflects this progress.

Catatonia is not rare when patients are systematically assessed for motor abnormalities. Kraepelin reported 20% of his patients with dementia praecox to be catatonic (see Fink & Taylor, 2003) and surveys since 1990 find that about 10% of acutely hospitalised psychiatric patients meet DSM criteria for catatonia (Bush *et al*, 1996*a*).

The official linking of catatonia and schizophrenia in classification systems is a misreading of the literature. Kahlbaum described the characteristic motor signs of catatonia among patients diagnosed as suffering from both mood and general medical illnesses (see Fink & Taylor, 2003). Kraepelin and Bleuler incorporated the syndrome to serve their view of dementia praecox (see Fink & Taylor, 2003). Since 1920, however, studies of catatonic subjects find 40–60% with an underlying mood disorder, whereas only 15% have schizophrenia. The clinical information in the report suggests that the patient's 1998 condition was a psychotic depression, for which his clinicians later prescribed lithium.

The report cites the vigorous use of antipsychotic drugs leading to neuroleptic malignant syndrome (NMS). The authors are unsure as to the relation between NMS and catatonia, first suggesting NMS to be distinguishable from catatonia and later stating that 'features common to both catatonia and NMS are increasingly recognised, with NMS felt to closely represent advanced catatonia'. We agree with the latter interpretation as attempts to demarcate the two syndromes have failed and the syndrome is induced by drugs other than neuroleptics. The more parsimonious view, based on the similarity of signs, symptoms and response to treatments, argues that NMS and malignant catatonia are best considered as one disorder (Fink, 1996).

Successful interventions for catatonia began with the demonstration in 1930 that intravenous barbiturates resolve catatonic stupor. In 1935, patients with catatonic schizophrenia were reported to recover when treated with chemically induced seizures (now electroconvulsive therapy (ECT)). The barbiturates were replaced by the benzodiazepines that are now reported as effective in 80% of catatonic patients (Bush *et al*, 1996*b*). When these drugs fail, especially in patients with malignant catatonia or delirious mania, ECT is remarkably effective (Fink, 1999).

The use of antipsychotic drugs in patients with catatonia is problematic. A malignant syndrome is associated with antipsychotic drugs, especially among patients with a medical illness, fever and dehydration. Rising serum creatine-phosphokinase and falling serum iron levels are findings that antecede the emergence of the malignant catatonia/NMS syndrome. The present report illustrates this hazard, as high potency antipsychotic drugs prescribed during the acute psychotic episode were associated with an NMS syndrome, relieved as 'benzodiazepines and anticholinergic medication were required on a number of occasions'. With ECT, the patient 'improved swiftly and substantially'. The continuation treatment with antipsychotic drugs was not helpful, requiring a second course of ECT. The avoidance of potent antipsychotic drugs and the prescription of diazepam probably contributed to his ongoing well-being.

BUSH, G., FINK, M., PETRIDES, G., *et al* (1996*a*) Catatonia: I: Rating scale and standardized examination. *Acta Psychiatrica Scandinavica*, **93**, 129–136.

—, —, —, *et al* (1996*b*) Catatonia: II. Treatment with lorazepam and electroconvulsive therapy. *Acta Psychiatrica Scandinavica*, **93**, 137–143.

FINK, M. (1996) Neuroleptic malignant syndrome. One entity or two? *Biological Psychiatry*, **39**, 1–4.

— (1999) *Electroshock: Restoring the Mind*. New York: Oxford University Press.

— & TAYLOR, M. A. (2003) *Catatonia: A Clinician's Guide to Diagnosis and Treatment*. Cambridge: Cambridge University Press, in press.

Max Fink Professor of Psychiatry, Long Island Jewish Medical Center, P.O. Box 457, St James, New York 11780, USA, **Michael A. Taylor** Professor of Psychiatry, University of Health Sciences/The Chicago Medical School, Illinois, USA

Sir: We read with interest Carey *et al*'s description (*Psychiatric Bulletin*, February 2002, **26**, 68–70) of a patient with catatonia and neuroleptic malignant syndrome (NMS). We have also recently treated a patient for both conditions.

Our patient was a 48-year-old woman who had suffered an intracranial bleed at birth resulting in left-sided hemiplegia and mild learning disability. Diagnosis of schizoaffective disorder, manic type, was made at the age of 16 years. She had only four previous admissions, most recent in 1983, and had been effectively treated with thioridazine.

She developed a florid psychosis following the change from thioridazine to quetiapine (as per Committee on Safety of Medicines guidelines) and the treatment of menopausal hot flushes with clonidine. Treatment was again changed, this time to chlorpromazine. Four days later, she developed NMS. When the NMS symptoms resolved, she remained mute, akinetic, doubly incontinent and had poor fluid intake for 4 weeks, with no evidence of psychosis. Resolution occurred spontaneously after listening to her favourite tape, *Beatles*. Since the resolution of the catatonia, she has suffered repeated epileptic seizures of all types. However, there is a history of falls in the months prior to admission.

Our case raised several important diagnostic and management issues. The general psychiatric staff (nursing and doctors) attributed the early signs of NMS to a combination of this patient's learning disability, mental illness and hemiplegia. The medical team believed her symptoms were 'behavioural' and attributed the raised creatine-phosphokinase to a fall 1 month earlier. Once they accepted the diagnosis of NMS, diagnosed by the learning disability team, she was transferred back to the psychiatric unit and care was by the in-patient staff and community learning disability team.

Management was purely supportive while there was no evidence of a disturbed mental state or deterioration in physical health, since there appeared to be no clear consensus in the literature about the treatment of catatonia and the family had concerns about the use of medication and electroconvulsive therapy. The thioridazine was restarted slowly

https://doi.org/10.1192/pb.26.10.393 Published online by Cambridge University Press