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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.

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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

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after a normal electrocardiogram at the request of the family, when the psychosis re-emerged. A literature search also suggested this was a suitable drug following NMS. Sodium valproate was added for treatment of the epilepsy but has also been a very effective mood stabiliser.

Interestingly, when the patient emerged from her catatonic state, she used the hemiplegic arm without any difficulties for a few days before it returned to a spastic premorbid position. Finally, the family are extremely pleased with the patient's progress, believing her mental state to be the best they have ever seen it and this is echoed by all staff who have known this woman.

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Clinical capacity assessment

Sir: Dr Raymont (*Psychiatric Bulletin*, February 2002, **26**, 201–204) is right to draw to our attention the complexities involved in the legal basis of our ministrations to the patient who may lack the capacity to give informed consent. We especially welcome discussion of the issue of belief and insight in this philosophical, legal and ethical morass, although we would have liked to see elaboration of terms like 'full insight' and 'greater level of capacity'. However, a particular suggestion made us wince.

Dr Raymont answers her own question, 'So how can we proceed currently with any physical treatment of those who lack capacity?' with 'Certainly a full psychiatric assessment should be made initially' – apparently before life-saving treatment. This presumably applies to many with major stroke or an acute cardiac event, a large number of the 30–60% admitted to medical wards with dementia or delirium (Ramsay *et al*, 1991; Treloar & Macdonald, 1997), a high proportion of all those in nursing homes (Macdonald *et al*, 2002) and every single unconscious patient. If by 'psychiatric assessment' she includes presenting complaint, history of presenting complaint, collateral history, and so on by mental health professionals, we wonder where all these professionals will come from?

Apart from this practical problem, we object on principle to the growing tendency for physicians and surgeons to involve psychiatrists in judgements about capacity to consent. Under current UK law (as opposed to some of the US jurisdictions in which the MacArthur Competence Assessment Tool for Treatment (MacCAT-T) (Appelbaum & Grisso, 1998) was developed) it is not necessary to diagnose the cause of any impaired capacity in order to make the judgement that it is impaired. All professionals must surely be able to make such judgements in relation to each decision, great or small, confronting their patient if they are not to be constantly exposed to accusations of battery on the one hand or neglect of duty of care on the other.

Each trust must ensure that its doctors are competent to assess capacity and have policies in place for treatment when capacity is lacking. It is the responsibility of every treating physician to gain the informed consent for the treatment they are delivering and to take appropriate measures if they do not believe the person to have that capacity. These include timely interventions to save life and, when more leisurely interventions are allowed, involving the relatives, consulting colleagues (almost never a psychiatrist) and other measures, in accord with the Bolam standard (Bolam v. Friern, 1957).

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Bolam v. Friern. Hospital Management Committee [1957] 2 *AllER* 118, 1 *W:R* 582.

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Off-label prescribing

Sir: We enjoyed reading the article by Lawrence *et al* (*Psychiatric Bulletin*, June 2002, **26**, 230–232). The findings complement some work that we have done in this area (Lowe-Ponsford, *Psychiatric Bulletin*, 2000, **24**, 415–417). Our postal questionnaire found that 65% of psychiatrists who replied had prescribed 'off-label' within the preceding month.

Medico-legal advice that we obtained (from the Medical Protection Society) was that, not only does Bolam (1957) need to be taken into account, but also the case of Bolitho (1997). This judgement means that the treatment has to withstand logical analysis as well as be accepted by a body of opinion. These considerations need to be taken into account in prescribing, alongside the capacity of the patient.

Many psychiatrists are worried about off-label prescribing and our study showed that 4% of respondents had received complaints about this matter. In our paper, we suggested some guidelines that may avoid many future medico-legal problems for clinicians if they should prescribe off-label.

The College's Psychopharmacology Special Interest Group has discussed this matter and is setting up a small working group (chaired by D. B.) to review the practice of off-label prescribing. We would be delighted to receive the thoughts of colleagues on this subject.

Bolam v. Friern. Hospital Management Committee [1957] 2 *AllER* 118, 1 *W:R* 582.

Bolitho *v.* City and Hackney Health Authority [1997] 3 *WLR* 1151.

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Community psychiatry in Nigeria

Sir: Accredited accommodation: an alternative to in-patient care in rural north Powys (Readhead *et al*, *Psychiatric Bulletin*, July 2002, **26**, 264–265).

Although separated by historical period, civilisation and culture, it is interesting to compare this scheme to a similar one in a developing country. The 'Aro village' in Nigeria is set in a semi-rural culture, and operated by the Department of Psychiatry, University of Ibadan. It was pioneered by Professor Adeoye Lambo, an eminent psychiatrist who later became the Deputy-Director of The World Health Organization. This initiative was the very first attempt in community psychiatry in Nigeria (Boroffka & Olatawura, 1976).

As in the case of the north Powys project, Aro village was adapted from the already existing infrastructure of a village community. It offered a social model of care and a rich rehabilitation resource.

Community confidence in the scheme was achieved through liaison between the psychiatrists and the community leaders, a delicate balance between traditionalists and Western psychiatry, a relationship based on trust and the prospect of mutual benefits from the project. In return, the Aro village witnessed infrastructural developments and on-site health clinics. Among other research interests, the project was the subject of an international research collaboration by Leighton *et al*, 1963.

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