References

BURKE, R. E., FAHN, S., MAYEUX, R., WEINBERG, H., LOUIS, K. & WILLNER, J. (1981) Neuroleptic malignant syndrome caused by dopamine-depleting drugs in a patient with Huntington's disease. *Neurology*, **31**, 1022–1026.

CHAYASIRISOBHAN, S., CULLIS, P. & VEERAMASUNENI, R. R. (1983). Occurrence of neuroleptic malignant syndrome in a narcoleptic patient. *Hospital and Community Psychiatry*, **34**, 548–550.

DESTEE, A., PETIT, H. & WAROT, M. (1981) Le syndrome malin des neuroleptiques. *Nouvelle Presse Medicale*, **19**, 178.

GRANT, R. (1984) Neuroleptic malignant syndrome. *British Medical Journal*, **288**, 1690.

HENDERSON, V. W. & WOOTEN, G. F. (1981) Neuroleptic malignant syndrome: a pathogenetic role for dopamine receptor blockage? *Neurology*, **31**, 132–137.

RITCHIE, P. (1983) Neuroleptic malignant syndrome. *British Medical Journal*, **287**, 561.

SIR: Kellam (*Journal*, June 1987, **150**, 752–759) has included Stauder's lethal catatonia on inadequate grounds, quoting Mann *et al* (1986), who consider NMS to be a neuroleptic-induced iatrogenic form of lethal catatonia. A similar opinion has also been expressed by Lindesay (1986), who perceived NMS as a hybrid of iatrogenic disorder and mis-diagnosed lethal catatonia. Lindesay suggested that lethal catatonia represents an idiopathic form of the disorder, whereas NMS may represent an iatrogenic form.

While not rejecting these opinions outright, it has to be kept in mind that the so-called lethal catatonia has only face validity as a nosological entity. There have been no well-planned prospective studies on its descriptive validity, construct validity, or predictive validity. Whatever information is available at the moment on lethal catatonia is a compilation of anecdotal reports such as the reviews by Mann *et al* (1986) and Kellam. Moreover, lethal catatonia is not mentioned in the current diagnostic and classification systems (DSM-III, ICD–9), and most psychiatrists no longer diagnose Stauder's lethal catatonia. Indeed, the term 'catatonia' is obsolete and confusing and should be eliminted from psychiatric terminology (*Lancet*, 1986), despite recent attempts to rehabilitate it (Barnes *et al*, 1986; Mann *et al*, 1986).

A superficial clinical resemblance between an iatrogenic syndrome and an entity of historical importance should not be grounds to contest the nosological status of NMS.

ADITYAN JEE

Department of Psychological Medicine
University of Malaya,
59100 Kuala Lumpur
Malaysia

References

BARNES, M. P., SAUNDERS, M., WALLS, T. J., SAUNDERS, I. & KIRK, C. A. (1986) The syndrome of Karl Ludwig Kahlbaum. *Journal of Neurology, Neurosurgery and Psychiatry*, **49**, 991–996.

LANCET (1986) Catatonia. *The Lancet*, *ii*, 954–956.

LINDESAY, J. (1986) Neuroleptic malignant syndrome and lethal catatonia. *British Journal of Psychiatry*, **148**, 342–343.

MANN, S. C., CAROFF, S. N., BLEIER, H. R., WELZ, W. K. R., KLING, M. A. & HAYASHIDA, M. (1980) Lethal catatonia. *American Journal of Psychiatry*, **143**, 1374–1387.

SIR: I agree with Singh & Maguire. Possibly if a new name for the syndrome is required it should be simply descriptive, as the aetiology remains obscure. I would suggest 'pyrexial catatonia'.

I am grateful to Adityan Jee for directing me to the invaluable article by Barnes *et al*. I would agree that 'catatonia', and especially its 'acute lethal' sub-type, only have face validity as clinical entities. I am not sure that any of the ICD–9 or DSM–III categories have much more. If we were to restrict ourselves to those categories whose validity had been demonstrated by well-planned prospective studies on their descriptive validity, construct validity, and predictive validity we should have few diagnostic entities left to use, either clinically or for further study.

My impression remains that a syndrome marked by rigidity and abnormalities of movement (catatonia) has been observed by several generations of psychiatrists, probably since they started to record their observations systematically. It was most often associated with other symptoms of what we now call schizophrenia, but has become rare since the advent of neuroleptic drugs. Occasionally it was associated with a fulminating course and death in hyperpyrexia. If this syndrome were to occur now it would usually do so in a patient already on neuroleptics, which would account for the Parkinsonian features now commonly seen and thus be indistinguishable from the neuroleptic malignant syndrome. I am therefore prepared to question whether neuroleptics or the other well-documented changes in dopaminergic drive, such as stopping L-dopa, are always to blame for the syndrome. I am currently enquiring of Barnes *et al* whether their idiopathic cases were pyrexial, in the hope that they may have recorded neuroleptic malignant syndrome in a drug-free patient.

The syndrome is probably stuck with its current name. My thesis is that we need to remind ourselves that we are not certain of either the implied aetiology or outcome. Vigorous treatment is to be encouraged in view of the reduction in mortality which can apparently be expected.

Recent work by Addonizio *et al* (1986) suggested the presence of a partial NMS in eight patients, as well as the full syndrome in two patients, out of a series of 82. All showed pyrexia and extra-pyramidal rigidity or tremor, and in the partial cases the symptoms remitted without the neuroleptics being

stopped. I hope to repeat this work on a larger series to see if a mild pyrexia is common in patients receiving neuroleptics and showing extra-pyramidal side-effects.

Finally, I should like to retract my statement that neuroleptic malignant syndrome is not mentioned in any British textbook (see O'Shea & Falvey, 1985).

ALEX M. P. KELLAM

University Hospital of Wales
Heath Park
Cardiff CF4 4XW

References

ADDONIZIO, G., SUSMAN, V. L. & ROTH, S. D. (1986) Symptoms of neuroleptic malignant syndrome in 82 consecutive inpatients. *American Journal of Psychiatry*, **143**, 1587–1589.

O'SHEA, B. & FALVEY, J. (1985) *A Textbook of Psychological Medicine for the Irish Student*. Dublin: Eastern Health Board.

Edinburgh Post-natal Depression Scale

SIR: Cox *et al* (*Journal*, June 1987, **150**, 782–786) have produced a concise instrument for detecting post-natal depression. I would question the inclusion of question seven in its present format. Probably the majority of new mothers experience sleepless nights for many weeks, because neonates generally take some time learning to sleep through the night. For such people, it might prove difficult to answer this question, which implies that unhappiness is the only cause for their insomnia. Rephrasing the question as follows might help:

"I have been so unhappy that I have had difficulty sleeping, even when my baby has been quiet and the opportunity for sleep was there. . .".

G. E. VINCENTI

The Duchess of Kent's Military Hospital
Catterick Garrison
North Yorkshire DL9 4DF

SIR: Vincenti has drawn attention to the central dilemma of the validity of established self-report mood questionnaires when administered to pregnant or puerperal women and to our attempt to overcome these difficulties.

The main problem which was discussed in our paper (*Journal*, June 1987, **150**, 782–786) is that certain somatic symptoms, such as weight loss or palpitations, may be caused by the physiological changes of childbearing as well as by a mood disturbance. The wording of the questionnaire items may also need to take into account the particular social circumstances of the mother and the incessant demands that may be made on her. The Edinburgh Post-natal Depression Scale (EPDS) was developed to reduce these ambiguities as far as possible.

Because many mothers experience sleepless nights for many weeks after childbirth, the sleep item (item 7), "I have been so unhappy that I have had difficulty sleeping", was worded to detect those mothers whose sleep difficulty was secondary to a mood disturbance and not caused *directly* by a noisy baby or by a restless partner.

The correlation matrix (available on request) between the 10 items on the EPDS confirmed that we had been successful in this endeavour. The sleep item had its highest correlations ($r = 0.52$) with item 3 ("I have blamed myself unnecessarily when things went wrong") and item 8 ("I have been feeling sad or miserable"), which would indicate that this item is detecting women whose sleep difficulties relate to their depression.

The modification suggested by Vincenti is, in our opinion, unlikely to be an improvement and might cause yet further problems; for example, what *are* the requirements for "the opportunity to sleep" – a comfortable bed, or perhaps a quiet partner?

JOHN COX

Department of Postgraduate Medicine
University of Keele
Stoke-on-Trent ST4 7QB

Predictions of Outcome in Depressive Illness

SIR: Eagles (*Journal*, May 1987, **150**, 715) appears to claim that we were unjustified in assuming that patients presenting for admission to hospital on clinical grounds were more likely to be depressed than those who remained in the community (*Journal*, January 1987, **150**, 43–48). We would like to hear the evidence supporting his rather idiosyncratic view.

We will answer Eagles' points seriatim. He observes that the sample was not "normally distributed" on the Newcastle Scale. However, elsewhere in the article, we clearly showed that there was evidence of discontinuity in the distribution.

He questioned the representative nature of the sample. However, we made no claim that the sample was representative of anything other than severely depressed patients. We made this clear on p. 46, second column: "we were probably not looking at a random sample of the general population, sources of potential bias being the mode of referral to hospital and the criteria of selection for the trials". Moreover, this was confirmed by the use of the Newcastle Scale worked out for use with similar in-patients.

We accept that there is a distinction between treatment response and outcome, and this may become