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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 Indeed, the term 'catatonia' is obsolete and confusing and should be eliminted from psychiatric terminology (*Lancet*, 1986), despite recent attempts to rehabilitate it (Barnes *et al*, 1986; Mann *et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.

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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