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Contents: Catatonia and NMS/Dementia with parietal signs/Eating disorders in Asians/Selection of controls/Computerised tomography in schizophrenia/Tardive dyskinesia and HLA/Menopausal depression/EPDS by post/Educational status and neurological abnormalities in schizophrenia/Brain lesions and cognitive function in late-life psychosis.

Catatonia and NMS

SIR: In their paper "Catatonia: harbinger of the neuroleptic malignant syndrome" (*Journal*, March 1991, **158**, 419–421), White & Robins described five consecutive cases in which there was a prodromal period of withdrawal, followed by psychomotor agitation and excitement alternating with mutism. This was followed by stupor, fluctuating temperature, rigidity, autonomic instability, increased creatine phosphokinase (CPK) and white blood count (WBC), after administration of one to three doses of neuroleptics. The authors concluded that patients with catatonic excitement would be at risk for NMS.

The cases were interesting as their presentations were similar to lethal catatonia which was often described in pre-neuroleptic literature. In Mann's (1986) review of this condition, he noted similarities in these early descriptions. There was a prodrome followed by psychomotor hyperactivity, alternating with mannerisms, stereotypes, hallucinations, delusions and confusion, before lapsing into stupor with rigidity, hyperthermia, autonomic instability, and finally death. In the same paper, Mann also noted that in 31% of the cases he reviewed, the patients received neuroleptics before they lapsed into stupor and fever. The similarities between the two conditions extend to laboratory investigations such as increased CPK, WBC, erythrocyte sedimentation rate (ESR) and liver function tests (Fleischhacker *et al*, 1990).

Fleischhacker *et al* (1990) maintained that patients with psychomotor excitement are likely to receive neuroleptics and any subsequent fever and rigidity would be diagnosed as NMS; the reasons being the lack of awareness among clinicians of lethal catatonia and the emphasis on NMS in recent literature.

White & Robins reported that their patients received supportive treatment but failed to say whether they continued to be treated with neuroleptics or electroconvulsive therapy (ECT) for their psychosis. This is important because the treatment of lethal catatonia would be the continuation of neuroleptics and ECT (Mann *et al*, 1986).

The cases described by White & Robins could represent either NMS or lethal catatonia, and therefore we cannot agree with them that their five cases truly represented NMS nor that catatonic excitement would be a risk factor for NMS.

We do agree, however, that NMS is a life-threatening condition, and that neuroleptics should be used with caution. Patients with catatonic excitement could be treated with benzodiazepine so that any subsequent fever, stupor and rigidity could be more reliably diagnosed as lethal catatonia and so treated with neuroleptics and ECT.

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MANN, S. C., CAROSS, S. N., BLEIER, H. R., *et al* (1986) Lethal catatonia. *American Journal of Psychiatry*, **143**, 1374–1381.

AUTHORS' REPLY: Drs Tang & Ong cite Mann *et al* (1986) who, in their comprehensive literature analysis of lethal catatonia, described a similar course in many cases to that which we observed in our series: progression of the catatonic state into a rigid, febrile stupor after initiation of neuroleptic treatment. Furthermore, they noted a mortality rate of 78.4% for the catatonic patients who received only a neuroleptic.