Drs Tan & Ong remark that the clinical and laboratory features of lethal catatonia are indistinguishable from the NMS and yet they suggest that they need to be differentiated for management purposes. We have come to view these two conditions as having a common pathophysiology (probably on the basis of hypodopaminergic function) and not as separate entities. We see catatonia as a spectrum disorder with gradations from benign to malignant (and potentially lethal) variants. When neuroleptics are administered to catatonics there is a shift in the clinical state towards the malignant pole.

The term 'malignant catatonia' might more appropriately embrace all conditions presently labelled as 'lethal catatonia' and the 'NMS'. Aetiological probabilities would then need to be explored, e.g. psychogenic, drug-induced, viral, idiopathic. This concept may help to resolve the diagnostic dilemma raised by Drs Tan & Ong which is currently confounding work in this area.

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SIR: Craddock *et al* (*Journal*, January 1991, **158,** 130) are correct to point out that creatine phosphokinase (CPK) may be raised in many conditions other than NMS. It was for this reason that other possible causes of raised CPK were considered in the case we reported (*Journal*, September 1990, **157,** 437–438). We concluded that NMS, or a variant of it, was the only condition which could account for both the clinical picture and the elevated CPK, and suggested that carbamazepine may have prevented the development of a fever. Interestingly, Coulter & Corrigan (*Journal*, March 1991, **158,** 434–435) postulate a mechanism whereby this could have occurred.

Craddock *et al* describe a case of catatonia to illustrate that CPK is raised in motor disorders other than NMS. However, White & Robins (*Journal*, March 1991, **158,** 419–421) report five patients in whom catatonia immediately preceded NMS, and support the view that these conditions may have a common neurochemical basis. NMS was first described as a rare and frequently fatal condition, but further case reports suggest that this may be just one end of a spectrum of NMS. Stauders Acute Lethal Catatonia occupies an equivalent position in relation to catatonia, and the similarity is compelling. Kellam (1987) suggests that "the resemblance of these catatonic conditions to NMS is especially striking if the possibility is accepted that the extrapyramidal signs of NMS could be due to the neuroleptic drugs now almost universally involved".

The boundaries of catatonia and NMS are indistinct, and their independent status uncertain. Published reports to date have not identified features unique to either condition.

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Reference

KELLAM, A. M. P. (1987) The neuroleptic malignant syndrome, so called: a survey of the world literature. *British Journal of Psychiatry*, **150,** 752–759.

Dementia with parietal signs

SIR: O'Carroll *et al* followed up demented patients with parietal signs (*Journal*, March 1991, **158,** 358–361). Their total dementia sample comprised 29 patients, of whom 18 were putative Alzheimer cases. It is clinically naive to assume that parietal disturbance caused by an infarct carries the same prognosis as parietal disturbance caused by other forms of cortical pathology. Therefore, their analysis, on their total sample, is irrelevant to the issue of heterogeneity within an Alzheimer's group. They do mention analyses on the 18 subjects whose Hachinski Index was less than or equal to four, but this group contained no patients under the age of 65 and no patients over the age of 86. In addition to this age restriction, the death rate of only six out of 18 over a four-year follow-up argues further that the group was unrepresentative of the population with global cortical dysfunction, which is the population in question. To omit the higher age-range patients is to omit just those patients with the lowest error score on a parietal test and the greatest longevity, and by so doing to minimise the chance of obtaining differential prognoses.

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AUTHOR'S REPLY: McDonald (1969) initially reported that patients with senile dementia who made 'parietal test' errors were (a) younger and (b) had a poorer prognosis in terms of life expectancy. In 1987, we failed to replicate (a) (*Journal*, January 1987, **150,** 114–117), and more recently we failed to