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Lethal catatonia and NMS

SIR: We would like to make some remarks in connection with the two letters by Tan & Ong (*Journal*, June 1991, 158, 858 and November 1991, 159, 729–730).

They suggested that the mother and the younger daughter in our paper on the familial occurrence of neuroleptic malignant syndrome (NMS) (Journal, June 1991, 158, 850–853) could be diagnosed as lethal catatonia (probably they meant that of psychogenic origin) rather than NMS, since they had past histories of catatonia. However, we believe that the episodes presented were neuroleptic-induced, e.g. the first episode of the mother, which was not preceded by catatonia, developed within eight days of the initiation of neuroleptic treatment.

More importantly, Tan & Ong appear to consider NMS and lethal catatonia (LC) as separate entities, despite the following discussions suggesting that NMS is a subtype of LC; Gelenberg (1976), and Barnes et al (1986) emphasised that catatonia was a syndrome with various causes, and neuroleptics could cause catatonic state. Mann et al (1986) emphasised that LC was also a syndrome rather than a specific disease based on a comprehensive review, and suggested that NMS was a neuroleptic-induced iatrogenic form of LC. More recently, White & Robins (Journal, March 1991, 158, 419-421) described five cases of NMS in which catatonia preceded the syndrome. This paper and our paper indicate that a patient with a past history of catatonia is at high risk of developing NMS, suggesting that NMS and LC have a common pathogenesis, probably hypodopaminergic function.

Tan & Ong suggested that NMS and LC should be differentiated, since the treatment of LC would be the continuation of neuroleptics and ECT. However, as reviewed by Mann et al (1986), neuroleptics are generally inadequate in treating LC and, in fact, may aggravate or complicate the disorder. The two case reports by Kish et al (1990) illustrate this view; these

two cases, in which clinical pictures were indistinguishable from NMS, were diagnosed as LC, and neuroleptics were continued, which ended in death despite electroconvulsive therapy (ECT). This finding suggests that neuroleptics should be discontinued in the conditions now labelled as NMS or LC, regardless of which diagnosis is given. Incidentally, Tan & Ong misquoted Mann et al (1986) who suggested the discontinuation of neuroleptics whenever LC was suspected, for the reasons mentioned above. The report by Mahmood (Journal, March 1991, 158, 437-438) on the effectiveness of bromocriptine for catatonic stupor, and the review by Davis et al (1991) on the effectiveness of ECT for NMS also suggest that the two disorders respond to common measures.

Our paper on the familial occurrence of NMS suggests that the predisposition to this syndrome may be genetically transmitted. This further suggests the close linkage between NMS and LC, since the familial tendency to catatonia (including LC) has also been reported (Barnes et al. 1986).

Thus, it is far more practical to consider NMS as a subtype of LC than to consider them as separate entities for the understanding and management of these disorders.

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Pisa syndrome-a confusing term

SIR: I read with interest the article by Turk & Lask ("Pisa syndrome in an adolescent on neuroleptic medication", *Journal*, March 1991, 158, 422–423). The case report, concerning a 15-year-old girl, and the discussion are important because they describe an impressive acute dystonic reaction which could have been mistaken for malingering or naughtiness. I want to make two remarks.