been divergent opinions regarding the interpretation of the cause of the clinical picture. It has been suggested that these patients have persistent neurological after-effects of acute lithium neurotoxicity (Donaldson & Cuningham, 1983; Schou, 1984). A detailed analysis of the patients described by Cohen & Cohen reveals that all were on high doses of lithium carbonate (1165 mg/day to 1800 mg/day). The maximum serum lithium ranged from 1.48 mmol/litre to 2.45 mmol/litre during the acute phase of lithium toxicity. All four patients were female. A preponderance of females has been reported in patients with persistent neurological sequelae of lithium (Donaldson & Cuningham, 1983; Schou, 1984), unlike NMS (Shalev & Munitz, 1986).

Moreover, each of the four patients was left with permanent brain damage (two became grossly demented, two had persistent dyskinesias). NMS, despite a mortality rate of 20%, is an acute condition and generally regresses without sequelae. Shalev & Munitz (1986) reviewed 120 patients with NMS and could identify only four with permanent sequelae. In one of these, permanent brain damage was probably due to brain anoxia during ECT given in the course of NMS and not due to the condition itself. In contrast to this, I have identified 48 patients with long-lasting sequelae of lithium intoxication (either alone or in combination with other drugs) in the literature.

Although there is controversy about lithium/haloperidol interaction (Cohen & Cohen, 1974; Frankel & Spring, 1982) it is well established that lithium intoxication at times resolves leaving persistent neurological sequelae (Donaldson & Cuningham, 1983; Schou, 1984). Since NMS is only a descriptive term, superficial resemblance may be seen with any other descriptive syndrome, e.g. lethal catatonia and some cases of lithium neurotoxicity.

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Gilles de la Tourette's Syndrome in Down's Syndrome

SIR: Karlinsky *et al* (*Journal*, May 1986, **148**, 601–604) speculate on a possible causal link between these two conditions. They appear to have neglected a more straightforward aetiology for the Tourette's syndrome.

It is reported that the patient developed seizures at the age of 24 years. Age-related epilepsy, with an onset in adult life, is a well recognised complication of Down's syndrome (Veall, 1974; Tange, 1979). The patient was being treated with carbamazepine two years later, at the onset of the Tourette's syndrome. Carbamazepine has recently been recognised as one of a number of pharmacological compounds (including dextroamphetamine, methylphenidate, and other adrenergic agents) which may precipitate tics, (Gualtieri & Evans, 1984). This tends to fit in with the dopaminergic theory of Tourette's syndrome, mentioned by the authors of the paper. Although, as they also point out, tics may be missed in people with mental retardation, there is as yet no clearly established link with any particular syndrome.

Precipitation of the tics by carbamazepine may account for the late onset of Tourette's syndrome in this patient, and seems a more likely explanation than those proposed by the authors. It is an iatrogenic complication of which psychiatrists should be aware.

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Mianserin and Blood Dyscrasias

SIR: It is with some concern that I note that manufacturers of mianserin (Norval, Bencard and Bolvidon, Organon) have written to the profession in the UK advising of changes in the Data Sheet concerning bone marrow depression and blood dyscrasias, the changes having been instituted at the insistence of the Committee on Safety of Medicines. The essential change is that a full blood count is recommended every four weeks during the first three months of treatment. This action follows concerns highlighted in a recent CSM update (1985).

There appears to be confusion between a drug-related depression in the white cell count and drug-