impairment, psychosis, alcoholism, or a history of head injury (Harvey, 1986). They were given tests of frontal lobe functions and sub-tests of the Weschler Adult Intelligence Scale (WAIS). Using matched normative data for Nelson's Modified Wisconsin Card Sorting Test (MWCST), the obsessionals were shown to perseverate significantly more than normals ($t=2.80$, $P=0.01$). Their mean percentage perseveration (50%, s.d. = 31) was greater than for patients with gross frontal lobe damage (42%, s.d. = 25). Obsessionals with or without significant perseveration were comparable on age-scaled sub-tests of the WAIS. Perseveration correlated with degree of obsessionality on the Leyton Obsessional Inventory (Spearman's $r=0.50$, $P=0.01$), which also correlated, negatively, with alternating category verbal fluency (ACVF) ($r=-0.62$, $P=0.002$). This latter test and the MWCST assess cognitive set-shifting ability, a specific frontal lobe function.

Although OCD could affect cognitive tasks, such as the ACVF test, via impaired performance efficiency, it seems unlikely that a qualitatively distinctive error, such as perseveration, would occur on this basis. An overall impairment of performance would seem more likely, although this was not evident from the WAIS sub-tests. Whether or not idiopathic OCD is associated with frontal impairment therefore needs to be looked at as a specific issue. It should not be assumed that such impairment necessarily implies the presence of a known brain injury.

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Gamma Glutamyl Transpeptidase and Mean Cell Volume in Alcoholics

SIR: Latcham (*Journal*, September 1986, **149**, 353–356) suggests that measurement of serum gamma glutamyl transpeptidase (GGT) and red blood cell (RBC) mean corpuscular volume (MCV) are of "little value in the assessment of patients admitted to psychiatric beds for problem drinking".

Macrocytosis in alcoholics has at least four different causes, some of which are independent of the duration of drinking habit. Lindenbaum (1980) comments that alcoholics' enlarged RBC can be secondary to liver dysfunction, reticulocytosis in response to blood loss, or folate vitamin deficiency, as well as "the macrocytosis of alcoholism".

Nearly a quarter of Latcham's male subjects and a sixth of the females were not clinically diagnosed as alcohol-dependent. What differences were there, in the correlations reported, between those diagnosed as alcohol-dependent and those diagnosed as suffering from alcohol abuse?

If only 143 male subjects had GGT assays performed then the maximum number of subjects in his Table III should be the same.

His data suggest that a considerable proportion of the male subjects had been abstinent for two or more weeks. This is long enough for mildly elevated GGT levels to settle to 'normal'.

The upper limit of normality for GGT of 50 i.u./litre is probably excessively high. We have recently shown (Hambridge & Jones, in preparation), that an upper limit of 40 i.u./litre is probably more clinically valid. Also, many clinicians will have experience of assessing younger, fitter alcoholics with high consumption and minimal damage – however defined.

I suggest that Latcham has not proven his case and that the measurement of GGT, RBC, and MCV remain of considerable value in assessing and managing alcoholics, when considered with the full clinical picture.

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Neuroleptic Malignant Syndrome or Lithium Neurotoxicity

SIR: I refer to the comments made by Lowe & Batchelor (*Journal*, September 1986, **149**, 385–386) on the review of the neuroleptic malignant syndrome (NMS) by Abbott & Loizou (*Journal*, January 1986, **148**, 47–51).

Their criticism regarding omission of the original report by Cohen & Cohen (1974) seems to be founded on inconclusive evidence. The descriptive picture in the four patients has been found to be similar to NMS (Frankel & Spring, 1982), but there have

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-