to demonstrate such a division. Secondly, it might be a genuine interform between schizophrenia and affective disorder (Kendell, 1983). This implies that both illnesses are polygenic, with the presence of some elements from each genotype predisposing to schizoaffective disorder. A third possibility, which does not imply polygenic inheritance of both Kraepelinian psychoses, is that it represents the presence of both the schizophrenic and the affective genotypes in the same patient: it occurs in individuals who, in genetic terms, have both illnesses. It has been argued by Kendell (1983) that this is unlikely because schizoaffective disorder is more common than would be expected given that "the chance coincidence of two illnesses each affecting around one person in a hundred is one in ten thousand".

We disagree with such a conclusion on several grounds. Firstly, there is no reason to suppose, as Kendell does, that for schizoaffective disorder to occur a genetic diathesis to schizophrenia would have to exist with one for bipolar rather than unipolar affective disorder. The morbid risk for all types of affective disorder combined is much greater than 1%, and therefore Kendell's expected figure would be larger. Secondly, assortative mating might take place to increase the likelihood of both genotypes being present in combination. Thirdly, Kendell assumes that possession of one genotype will not affect expression of the other. On the contrary, it seems likely that schizophrenic symptoms will trigger the onset of affective illness in a predisposed individual and vice versa. Moreover, it is quite possible that *subclinical* expression of one genotype increases the likelihood of the other being expressed in a predisposed individual. For example, possession of an affective diathesis might render an individual with a schizophrenic genotype more likely to develop a psychotic reaction to stressful life events. Such a psychosis might be expected to show both schizophrenic and affective features.

In summary, we suggest that the symptomatic continuum reflects phenotypic interaction rather than the relationship proposed by Crow.

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Procyclidine Abuse

SIR: In reporting five more cases of procyclidine abuse, Fenech & Khoosal (*Journal*, October 1986, **149**, 524) pointed out that the latest edition of the *British National Formulary* (BNF) did not mention the potential for abuse of that drug. Pullen *et al* (1984*a*) drew attention to the fact that the BNF omitted reference to the abuse of any anticholinergic.

At last the BNF (Number 11, 1986) does include one sentence on abuse in its section on the use of antipsychotic drugs (p. 139), although there is still no mention of this danger in the main section on anticholinergics (pp. 182–184). These preparations continue to be freely prescribed, and there still seems to be continuing ignorance about these significant drugs of abuse (Pullen *et al*, 1984*b*).

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Neurological Factors in Obsessive-Compulsive Disorder

SIR: In their article on obsessive-compulsive disorder (OCD) (*Journal*, September 1986, **149**, 315–319), Kettl & Marks emphasise the case for organic precipitants, probably operating 'downstream' from the primary mechanisms initiating the condition. Whether cognitive abnormalities in OCD attributed to these organic precipitants could be intrinsic or secondary features of the condition is not assessed. This remains unclear in the recent studies quoted (Behar *et al*, 1984; Flament & Rapoport, 1984), which showed computerised tomogram and cognitive abnormalities in adolescents with OCD. Brain injury was not used as an exclusion criterion in the group concerned. Some patients had histories of head trauma and birth injury, and one is mentioned as having tardive dyskinesia.

I have studied 19 DSM-III-diagnosed obsessive-compulsive patients, none of whom had neurological

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