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