The authors mention the use of caffeine as a possible measure for lowering seizure threshold. Subsequent to the case report which they cite (Shapira *et al*, 1985), we have evaluated the effect of pre-ECT administration of caffeine sodium benzoate on seizure parameters in a controlled study (Shapira *et al*, 1987). The results supported our original observation and showed a significant increase in seizure duration during treatments preceded by caffeine administration. Caffeine did not induce untoward haemodynamic effects, nor a greater degree of cognitive impairment than is normally associated with ECT, and was subjectively well-tolerated. These findings have been borne out by other studies (e.g. Hinkle *et al*, 1987). The patient reported by Drs Sharpe & Andrew eventually remitted following lithium supplementation of ongoing treatment with amitryptiline and chlorpromazine. We have recently reported (Shapira *et al*, 1988) seven patients who were unresponsive to ECT and eventually remitted following lithium supplementation of tricyclic antidepressant (TCA) treatment. Two had not responded to this combination prior to ECT. Four of these patients were administered ECT in our hospital; they all had seizures exceeding 25 s in length during their courses of 11 or more bilateral treatments. Three reports by other authors (e.g. Nelson & Mazure, 1986) mention cases in which lithium supplementation of TCA induced remission in patients unresponsive to ECT. We therefore suggest that lithium supplementation be considered in TCA non-responders prior to referral for ECT.

This work was supported in part by NIMH grant No. 40734.

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Cerebral Ventricular Enlargement in Chronic Schizophrenia

SIR: Farmer *et al* (*Journal*, March 1987, **150**, 324–330) reported that mean ventricular brain ratio in Type I schizophrenia with positive symptoms was significantly larger at the $P=0.05$ level than that in Type 2 schizophrenia with negative symptoms, in a sample of 35 patients with chronic schizophrenia. This finding is in contrast to the finding of Johnstone *et al* (1978) and does not support Crow's (1980) proposal that Type 2 schizophrenia is associated with cerebral ventricular enlargement.

In a study conducted at the Psychiatric Unit of the All India Institute of Medical Sciences, New Delhi, 30 patients suffering from schizophrenia with continuous illness of at least two years duration were studied regarding cerebral ventricular enlargement. The diagnosis of schizophrenia was made using Feighner *et al*'s research criteria for schizophrenia, and computerised tomography of the brain was performed using CT-1010. Negative and positive symptoms were rated in each patient using the Comprehensive Psychopathological Rating Scale for Schizophrenia (Jacobsson *et al*, 1978). Of 30 patients, 18 were assessed to have negative symptoms, whereas 12 had positive symptoms. The lateral ventricular size was measured by using free-arm planimetry, and was expressed as ventricular brain ratio. The width of third ventricle and sylvian fissures were measured by using callipers, and were expressed to the nearest millimeter. We found that there was no significant difference between the group of patients with negative symptoms as compared with the group with positive symptoms with regard to ventricular brain ratio ($t=0.14$), third ventricle ($t=1.05$), and sylvian fissure ($t=1.27$).

We are not able to offer any explanation as to why there is such a difference in the findings of different centres regarding association of cerebral ventricular enlargement with Type 2 schizophrenia. We think that Dr Crow's contention regarding the association of ventricular enlargement with Type 2 schizophrenia is not supported by these research findings.

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The Concept of Disease in Psychiatry

SIR: A recent editorial in *Psychological Medicine* on the concept of disease in psychiatry (Häfner, 1987) helped to clarify many problems connected with this issue. I would like to emphasise an approach which might add to our understanding.

For any doctor in therapeutic practice – and to the general public – a disease is what can be treated with a certain success by a physician. This definition is operational in the extreme; nevertheless, it should influence our theoretical thinking.

The estimate of the prevalence of endogenous depression at the beginning of this century was 0.5–1%. At this time patients would be in hospital for months, and all that could be done was custodial care and prevention of suicide. In some institutions opium was administered, with ambiguous results. In the period 1940–1950, the prevalence of endogenous depression was estimated to be 2–3%. Again, the patient had to be admitted to hospital, and could be treated with ECT. The duration of hospital stay would be 4–5 weeks, and the patient would usually be discharged after a satisfactory remission. Nevertheless, understandably, only severe cases were chosen to be treated in this way.

With the introduction of antidepressants, which enabled doctors to treat depressed patients as outpatients, an ever-increasing number of depressive patients has been found in the population, and current estimates of the prevalence reach 10% or more. Again, the influence of the second generation of antidepressants (e.g. mianserin, maprotiline) and of the antidepressive benzodiazepines (e.g. alprazolam, bromazepam) can be observed: their side-effects are fewer and less unpleasant than those of, for example, imipramine and amitriptyline.

It might be speculated that the increasing possibility of drug treatment of distress, considered some 10–20 years ago as a common human condition, has been an important factor contributing to the change in the image of mental illness and to the changes in the diagnostic criteria for depression, as reflected *inter alia* in DSM–III.

For a doctor in routine practice, the differentiation of patients who will respond to neuroleptics from those who will respond to antidepressants is more important than theoretical consideration about the nosological entity of schizoaffective, mixed, or other psychoses. The same is true when deciding whether to begin lithium prophylaxis of a periodic psychosis.

Medicine has been always action-oriented. Physicians have been interested in cases which can be treated, in conditions which can be changed. An ever-increasing interest in genetic and other biological factors in the pathogenesis of mental disorders is due to the perspective of the possibility that errors in the human genome will be accessible to therapeutic intervention. In this light, the changes in opinions about possible biological causes leading to new concepts of nosological classification of alcoholism, panic anxiety, drug dependence, and criminality can be better understood.

The opposite also seems to be true: apart from the ethical and political issues, the failure to treat homosexuality contributed – according to this line of reasoning – to the disappearance of this diagnosis in nosological classification. If – 20–30 years ago – some simple drug treatment had been discovered for homosexuality, this variation of sexual behaviour would have remained in psychiatric nosology – irrespective of the possible theoretical interpretation that this fact alone would mean that homosexuality was an illness.

Therapeutic pragmatism thus may play a decisive role when constructing the concept of disease and when dealing with the problems of nosological classification.

Are mild monosymptomatic headaches, insomnia, tiredness, or feelings of emotional tension illnesses? Patients begin to believe so if they get a pill which helps – and so do doctors. The pharmaceutical industry does not object to such an evolution. This does not decrease the merit of the industry in promoting research in neurophysiology, molecular biology, and other scientific disciplines as long as their progress remains related to the relief of human suffering. A rational concept of disease should serve to further such an aim.

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Combined Minaserin and Tranylcypromine

SIR: The early controversy about the risk of side-effects occurring with combined tricyclic