cerebral ventricles in schizophrenia. As Dr Miller states, one or two studies have reported a weak correlation. By the same token, at least one study has reported an inverse correlation: that lateral ventricles somehow shrink as the illness progresses.

Overall, this pattern of findings suggests an occasional Type 1 statistical error, hardly surprising given the large number of studies now in existence and the many possible confounding factors. Even if Dr Miller were correct, it is still a big jump to suggest that progressive brain changes might be arrested by antipsychotic drug treatment. It would be equally reasonable to say that prolonged drug treatment were the cause. In any case, conscientious clinicians who already pay close attention to drug treatment will continue to do so, whichever view turns out to be right.

SHÔN LEWIS

*Academic Department of Psychiatry*
*Charing Cross and Westminster Medical School*
*London W6 8RP*

**'The disease concept of alcoholism'**

SIR: Professor Griffith Edwards has kindly commented on my review of Jellinek's monograph, *The Disease Concept of Alcoholism* (*Journal*, March 1991, **158**, 431). Like him I have been unable to find any information that is not generally known about Dr E. M. Jellinek. As far as I am aware, after making enquiries, no biography of Jellinek has ever been written, although my understanding is that one is currently being undertaken in the USA. Mr Archer Tongue, a former Director of the International Council on Alcohol and Addictions, who has more knowledge of workers in the alcoholism field than most, was also unable to provide any further information.

It is interesting that Professor Edwards states that Jellinek's ideas really have to be seen as part of a continuing process, with which I would agree, although I myself am not familiar with the references he cites; but there is no doubt, as outlined in the excellent review by Levine (1978) that such concepts as "The disease concept of alcoholism", extended, particularly in the United States, throughout the 19th and 20th centuries, as has the continued debate as to whether alcoholism is best regarded as an illness or as deviant behaviour.

The comment that the alcohol dependence syndrome is a more socially acceptable form of the disease concept is not my original statement but the view of Heather & Robertson (1981) who state ... "therefore the alcohol dependence syndrome is a conception of abnormal drinking based primarily on psychobiological dependence with impaired control as its leading symptom. With the substitution of loss of control for impaired control, how different is this from Jellinek's (1960) formulation?"

Despite this criticism, the alcohol dependence syndrome offers those of us working in this field a practical definition, linking alcohol dependence to drug dependence, and also giving us a model in which the dependence phenomenon is not all or none, but graded, a concept which has considerable importance, both in treatment goals and research into different populations varying in their level of dependence.

HEATHER, N. & ROBERTSON, I. (1981) *Controlled Drinking*. London: Methuen.

LEVINE, H. G. (1978) The discovery of addiction, changing conceptions of habitual drinkers in America. *Journal of Studies of Alcohol*, **39**, 143–174.

BRIAN HORE

*Withington Hospital*
*West Didsbury*
*Manchester M20 8LR*

**Double blind acceptance**

SIR: I am certainly happy to accept that my position and that of Double (*Journal*, April 1991, **158**, 573–574) do not now differ materially. He is right to point out that blindness is prone to be used as a buzz word emptied of its legitimate meaning, so that one should be happier about a study in which the investigators accept they cannot achieve blinding, than one in which blindness is claimed, but is illusory. (In the same way, the key concept of random allocation becomes vacuous when 'random' is taken to mean 'arbitrary' and no longer implies specific measures to minimise the possibility of bias being introduced by the admitting clinician.) The application of a method that will be as scientifically valid as possible in a real clinical situation, with the limitations which that inevitably introduces, remains the goal of study design. Scientific rigour in anywhere near an absolute sense is rarely attainable, and the editorial peer review process should regard a study report in which the limitations of the results are specified clearly as more acceptable than one in which the problems have been swept under the carpet, although inferable by a reasonable measure of experience-based lateral thinking.

The application to clinical and preventive practice, in the absence of clearcut results, is also a very important issue. Often one cannot simply ignore an issue: to do so is effectively to act as if the status quo were established. This applies whether the problem

lies in inadequacy of method or simply inconclusive results: should one treat mild hypertension, although the (very large) MRC mild hypertension trial did not detect any clear effect on mortality?

ROBERT NEWCOMBE
*University of Wales College of Medicine*
*Department of Medical Computing and Statistics*
*Heath Park*
*Cardiff CF4 4XN*

*This correspondence is now closed. I.P.*

**Ordering thoughts in thought disorder**

SIR: McGrath proposes a hypothesis suggesting the involvement of frontal lobe dysfunction in schizophrenic thought disorder (*Journal*, March 1991, **158**, 307–316). He argues mainly from the similarity between the pattern of language deficit in thought disorder and the pattern of cognitive–behavioural deficit in frontal lobe syndrome.

One further link between thought disorder and frontal lobe dysfunction is their association with eye tracking disorder. Among other symptoms and signs, thought disorder has one of the strongest associations with eye tracking disorder (Keefe *et al*, 1989). Eye tracking is controlled by a number of brain areas. One such area, thought to be affected in schizophrenia, is the frontal eye field (Fukushima *et al*, 1990), located in the prefrontal cortex.

Thought disorder encompasses a variety of phenomena. The proposed "prefrontal-basal ganglia loop" may account for only part of the diversity. Apart from the basal ganglia and the thalamus, the prefrontal cortex has reciprocal connections with the posterior association cortex, and limbic structures such as the hippocampus and the amygdala (Fuster, 1987). For example, one such area, the temporal cortex, is important in relation to category knowledge (e.g. Damasio, 1990). Categorisation is affected in some patients with thought disorder.

It seems plausible to propose that many prefrontal related loops (a distributed network radiating from the prefrontal area) including for instance, a prefrontal-temporal pathway, are affected in schizophrenic thought disorder. The different aspects of thought disorder (loosening of association, poverty of content of speech, etc.), may reflect differential dysfunction of these loops. It is important that studies relating thought disorder to brain mechanism take into account the various dimensions of thought disorder (rather than a global thought disorder measure), and study simultaneously a number of brain areas in order to capture some of the more subtle interactions.

DAMASIO, A. R. (1990) Category-related recognition defects as a clue to the neural substrates of knowledge. *Trends in Neuroscience*, **13**, 95–98.

FUKUSHIMA, J., MORITA, N., FUKUSHIMA, K., *et al* (1990) Voluntary control of saccadic eye movements in patients with schizophrenic and affective disorders. *Journal of Psychiatric Research*, **24**, 9–24.

FUSTER, J. M. (1987) Prefrontal cortex. In *Encyclopaedia of Neuroscience* (ed. G. Adelman) pp. 972–975. Boston: Birkhauser.

KEEFE, R. S., SIEVER, L. J., MOHS, R. C., *et al* (1989) Eye tracking, schizophrenic symptoms, and schizotypal personality disorder. *European Archives of Psychiatry and Neurological Sciences*, **239**, 39–42.

ERIC Y. H. CHEN
*Fulbourn Hospital*
*Cambridge*

SIR: McGrath's review (*Journal*, March 1991, **158**, 307–316) omitted mention of cognitive behaviour therapy strategies which are attracting increasing attention in the management of schizophrenia (e.g. Perris, 1989). The stages of therapy would appear to be rapport building, examination of the antecedents of psychotic breakdown, weighing of evidence, and reality testing, with generation of alternative hypotheses using a normalising rationale (Kingdon & Turkington, 1991). Supplementary techniques include reduction of the emotional investment of psychotic symptoms and inference chaining to identify underlying schemata. These techniques seem to be most effective in conjunction with standard management regimes in the setting of a comprehensive psychiatric service. We describe the first reported use of these techniques in a thought-disordered hebephrenic patient.

*Case report*. A 22-year-old single man presented himself to our psychiatric reception area in a floridly thought-disordered state, with prominent paranoid delusions and incongruity of affect. These symptoms had worsened over a two-week period, during which time his sleep became progressively disturbed. He had experienced two schizophrenic episodes during the previous four years, which were treated with neuroleptics and cognitive therapy, and had returned to work on both occasions. No underlying disorder or history of drug abuse was detected. He was admitted, and treated with neuroleptics. Cognitive therapy sessions were included in his management programme. Rapport was established early but progress was impeded by intermingling of themes and derailment, which at times lapsed into incomprehensibility. He gave informed consent to the videotaping of an interview for teaching purposes. Review of this demonstrated that four clear themes were present. These were: references to a road traffic accident that he had witnessed, his mother's ill-health, concern about somatic symptoms of anxiety, and finally intermittent references to the videotaping process itself as being a form of experimentation. In relation to the latter, he commenced the interview by referring to his half-Jewish parentage and later