Professor Kendell seems quite concerned about the results of recent comparisons of the effect of real and simulated ECT in the treatment of depressive illness, particularly the results of the Northwick Park trial (Johnstone *et al*, 1980). This study seriously questions the generally assumed efficacy of ECT as anti-depressant method. However, when reading the paper by Johnstone *et al*, one finds on page 1318 the following: "Of the 62 patients who finished the course, 18 (8 on real ECT and 10 on simulated) were given benzodiazepines, mainly either as diazepam 5 mg regularly twice daily or as diazepam 10 mg in occasional doses to relieve distress. Improvement scores were similar in patients with and without diazepam. The only other psychotropic medication was a benzodiazepine hypnotic prescribed for all patients".

Clinical ECT experience and some research data indicate that benzodiazepines can be an effective means of decreasing the efficacy of ECT. In a retrospective study (Sand Strömgren *et al*, 1980), it was found that ECT-treated depressive patients, who received benzodiazepine, showed shorter seizure duration and a need for a significantly greater number of treatments.

Johnstone *et al* write that improvement scores were similar in patients with and without diazepines. It is however unclear what they mean. Benzodiazepine hypnotic was prescribed for all patients.

The administration of benzodiazepines to ECT-treated patients in the Northwick Park trial does not logically allow any conclusion about the efficacy of real ECT compared to simulated ECT.

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PROPRANOLOL IN SCHIZOPHRENIA

DEAR SIR,

Dr Carr (*Journal*, November 1981, **139**, 47) makes a relevant point in his criticism of our trial of propranolol in schizophrenia (*Journal*, August 1981, **139**, 105–11). It would be surprising if chronic schizophrenic in-patients showed a clear response to a new drug within three months. However, this was precisely the claim which was being made for propranolol by earlier workers, and our trial was specifically designed to test this claim, with a negative result. The lack of response to chlorpromazine was not surprising, as these patients were still in hospital with schizophrenic symptoms in spite of previous treatment with neuroleptics. Chlorpromazine is normally an effective treatment for schizophrenia, and, therefore, the term 'chlorpromazine-resistant' is appropriate for a group of schizophrenic patients who fail to respond to chlorpromazine. It is, of course, possible that our patients were also 'propranolol-resistant' and that propranolol might be effective in a different patient group, but our reasons for believing that we chose an appropriate group of patients for this trial are detailed in the discussion of our paper, to which Dr Carr does scant justice by quoting half-sentences out of context. Dr Carr appears to suggest that propranolol may be effective in the long-term maintenance treatment of chronic schizophrenic patients. This is not the principal claim which has previously been made for propranolol, and an entirely different trial design would be required to investigate it.

A number of psychiatrists have been encouraged by enthusiastic claims for propranolol to 'give it a try' in their chronic drug-resistant patients. They should appreciate that nobody has yet succeeded in demonstarting that propranolol as a sole agent is more effective than a placebo in the treatment of schizophrenia. On the other hand, there is good evidence that propranolol enhances the efficacy of neuroleptic drugs. The pharmacokinetic interaction which we demonstrated between propranolol and chlorpromazine, which leads to increased plasma levels of chlorpromazine, is sufficient to explain this effect which could probably be parallelled by using very large doses of neuroleptics rather than by co-prescribing propranolol. In my view the use of megadose propranolol in the treatment of schizophrenia is unjustified outside a research setting, in view of the lack of evidence of efficacy and the known risks of cardiovascular complications with such high doses.

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SEASONALITY OF SCHIZOPHRENIC BIRTHS: HARMFUL EFFECTS OR GENETIC MORPHISM?

DEAR SIR,

That schizophrenics tend to be born in the colder months of the year has been clearly established (Torrey, 1980). There are negative correlations

between temperature of month and number of schizophrenics born, and positive correlations between temperature and conceptions (Templer *et al*, 1978; Templer and Austin, 1980). There are two dominant general sorts of explanations for the relationship between temperature and schizophrenic births. The 'harmful influence' hypothesis (McNeil *et al*, 1971) is that there are harmful effects surrounding birth or during gestation such as infection, trauma, or nutritional variables. The other explanation (Hare and Price, 1969) is based on the genetic morphism hypothesis of Huxley *et al* (1969) and contends that schizophrenics have physiological advantages that better enable them to survive infancy. There is a dearth of research data giving greater support to one hypothesis or the other. The seasonality of schizophrenic birth has been found to be significantly greater in Europe than in the United States (Templer *et al*, 1978). This difference was accounted for in terms of the generally greater prosperity and protection from the elements in the United States over the past century. On the basis of this improved technology explanation, Templer and Austin (1980) predicted and found a significant decrease in seasonality of schizophrenic births from 1900 to 1960 in Missouri. However, it is apparent that decrease in seasonality and greater seasonality in Europe could be explained both by fewer harmful influences and in terms of fewer non-schizophrenic infants dying.

The present study was designed to differentiate between the two alternative hypotheses. Paranoid schizophrenics tend to have a lesser percentage of schizophrenic relatives than other schizophrenics and have been presumed to have a lesser genetic predisposition, whereas catatonic and hebephrenic schizophrenics, grouped together as 'nuclear' or 'kernel' schizophrenics, tend to have a greater percentage of schizophrenic relatives than other schizophrenics and have been presumed to have a greater genetic predisposition. If the harmful influence hypothesis is valid, then environmental effects of a seasonal sort should play a relatively larger role in persons who have a lesser genetic predisposition. We would then expect seasonality of births to be more pronounced in paranoid than in catatonic and hebephrenic schizophrenics. If the genetic morphism hypothesis is valid, then the infants more likely to survive adverse circumstances would be those with a greater genetic predisposition to become schizophrenic. We would then expect the hebephrenic and catatonic schizophrenics to display greater seasonality of births.

Analysis was carried out on months of birth from 1900 to 1959 for the 10,495 paranoid and 1532 combined catatonic and hebephrenic unduplicated admission schizophrenics supplied from the Missouri Department of Mental Health. The years used were those that constitute the six decades for which complete information is available. For each decade, the number of schizophrenics born were totalled for each of the 12 months. Rank order correlation coefficients between Missouri average temperature of month and number of schizophrenics born that month, corrected for number of days per month, were calculated for each decade.

For all six decades the rank order correlations are larger (in the negative, expected direction) for the paranoid group than for the catatonic-hebephrenic group ($\chi^2 = 6.00$, df = 1, $P < .025$). A ranking of all 12 correlations shows little overlap ($U = 3$, $P < .01$). Thus, the harmful influence hypothesis is supported. It would appear that the development of schizophrenia in those that have less genetic predisposition is determined relatively more by harmful influences related to seasonal factors. Yet, the nature of the harmful influence(s) is not clear. However, Torrey and Peterson's (1976) postulation of a slow virus could be more tenable than explanations involving malnutrition or brain trauma because of the paranoid schizophrenics' superiority over other schizophrenics in cognitive functioning. As Torrey (1980) pointed out, viruses can alter the function of nerve cells without altering their histological structure.

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