

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 Strömngren *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 diazepam. 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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#### References

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- SAND STRÖMNGREN, L., DAHL, J., FJELDBORG, N. & THOMSEN, A. (1980) Factors influencing seizure duration and number of seizures applied in unilateral electroconvulsive therapy. *Acta Psychiatrica Scandinavica*, *62*, 158-65.

#### 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 schizo-

phrenic 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 demonstrating 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 paralleled 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