metabolism (Rivera-Calimlim *et al*, 1973). These two pieces of evidence suggest that reduction of the dosage of neuroleptic might be a more appropriate way by which to combat drug-induced Parkinsonism.

In his final sentence, Dr Bennie suggests that anti-parkinson drugs be administered to neuroleptic treated schizophrenic patients who appear to be depressed. Certainly it is easy to confuse the mask-like facies of Parkinsonism with retardation attributable to depression and in such cases the administration of anti-parkinson drugs, might be useful. Some anti-parkinson drugs have been claimed to possess anti-depressant properties but the evidence for this is weak (Onuaguluchi, 1964; Mindham *et al*, 1972).

For the reasons given here and elsewhere, I do not believe that patients receiving long-term medication with neuroleptics should also be given anti-parkinson drugs on a routine measure. Firm recommendations, such as that made by Dr Bennie, require better evidence than is at present available.

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DAYTIME ENURESIS

DEAR SIR,

Dr Barton and Dr Felker have responded to my *Comments* article Child Psychiatry and Enuresis (*Journal*, September 1981, **139**, 247–8) with a letter (*Journal*, March 1982, **140**, 325) in which they suggest that imipramine is effective in treating diurnal enuresis. However, they admit that they have no evidence from a controlled trial to support their view. I have, to support mine.

I recently participated in a randomized, double blind controlled trial of imipramine in diurnal enuresis, carried out by Professor Roy Meadow, Paediatrician in Leeds. This has not yet been published. Twenty-seven children were included in the study. Although there was some improvement in day wetting during treatment there was no difference in response between the placebo and the active drug groups of cases. In my view, considering the dangers for children of overdose with this hazardous drug, until there is some real evidence for its efficacy in treating diurnal enuresis it should not be used for this condition.

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PROPRANOLOL IN SCHIZOPHRENIA

DEAR SIR,

In a recent article in this *Journal* (August, 1981, **139**, 105–11), Peet *et al* concluded that propranolol did not improve schizophrenic symptomatology relative to placebo, while the effects of chlorpromazine were small and inconsistent. It is not the purpose of this letter to question the effects of these drugs, but rather to examine the quality of the evidence marshalled to draw these conclusions.

Finding that treatments do not differ from each other may arise from two different circumstances: they do not in fact differ, and the authors have arrived at the correct conclusion; or they do differ, but the investigators have failed to detect this difference. The reason for the latter result (known in statistical jargon as a Type II error) is that the power of the

statistical test was inadequate; most often, this is due to an insufficient sample size.

In their placebo group, the mean BPRS baseline score was 21.3 with a standard deviation of 8.91 ($2.1 \times \sqrt{18}$). Assuming that a 5 point difference was clinically significant, that they wanted the power of their test to be 80 per cent (i.e., they would be able to detect a difference this large 80 per cent of the time), and they used the traditional significance level of 5 per cent, then there would have had to have been at least 53 subjects per group, or three times the sample size used. Looked at in another way, if 50 per cent of the patients on chlorpromazine developed a specific side effect, and the authors were looking for a reduction to 25 per cent with propranolol, then they would have had to test 77 subjects in each group, as opposed to 16 to 19 actually assessed. Consequently, their conclusions regarding the lack of effect of propranolol, and the poor showing of chlorpromazine, must remain unproven: the study did not have sufficient power to have detected an effect if it were there.

While these calculations pertain to this specific study, the criticism is more general: negative findings should be viewed with suspicion unless the sample size is sufficient to avoid a Type II error.

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ECT AND THE GROWTH HORMONE RESPONSE TO APOMORPHINE

DEAR SIR,

We read with interest the paper by Dr Janice Christie and her colleagues (*Journal*, March 1982, **140,** 268–73). Over the last three years we have conducted a similar investigation into the effects of electroconvulsive therapy (ECT) on the growth hormone (GH) response to apomorphine. The results of this study will be published shortly. In contrast to the findings of Christie *et al*, we showed in a group of fifteen patients that ECT significantly increased the apomorphine GH response. Some of our patients were taking antidepressant drugs but a significant increase in response was still seen when the tests of these subjects were excluded from the analysis. A concurrent study in other depressed subjects indicated that neither antidepressant drugs nor clinical recovery *per se* caused increased apomorphine responses.

The dose of apomorphine used in our study was lower than that of Christie *et al*. Our initial studies showed that a dose of 0.005 mg kg^{-1} of apomorphine, given subcutaneously, was well tolerated and produced a reliable and reproducible increase in plasma GH. Higher doses often resulted in unpleasant side effects such as nausea. During this preliminary investigation we noted that subjects with high baseline GH levels often showed an attenuated GH response to apomorphine challenge. In our patient study three baseline samples were taken over a period of 30 min. Patients whose tests showed an elevated GH level (>6.5 ng ml^{-1}) in any of the baseline samples were excluded from the study. We excluded three patients by this criterion, but if the results of these patients are taken into account the increase in apomorphine GH response following ECT is no longer significant. Although Christie *et al* stated that exclusion of patients with high baselines did not alter their findings, the exact nature of the exclusion criteria may have been different from our own. In addition, unlike Christie *et al*, we found no difference in basal GH levels following ECT, again suggesting the potential importance of baseline effects.

Clearly there are many other possible differences between our studies which will need to be discussed. We believe, however, that the best way to resolve the matter would be to repeat the investigation in the setting of a double-blind ECT trial, where the effects of repeated anaesthetic (Steiner and Grahame-Smith, 1980) might also be assessed. If ECT does produce enhancement of monoamine responses (Grahame-Smith *et al*, 1978) the implication for its mode of action seems too important an issue to be left in doubt.

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PLACEBO-CONTROLLED STUDIES OF ECT

DEAR SIR,

In an effort to complete the record on placebo-controlled studies of ECT, both Dr Kendell (*Journal*, October 1981, **139,** 265–83) and Dr Mendelson (*Journal*, March 1982, **140,** 322), omit a 1958 random assignment study in which we compared the clinical, electrophysiologic, and neuropsychologic effects of grand-mal and subconvulsive (sham) treatments. All treatments were given under barbiturate anesthesia, and only the treating physician knew which patients