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