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DEAR SIR,

Since we did not claim to have carried out 'a faithful replication study' of Lader and Wing's (1966) work, and indeed described our modifications in detail, your correspondents' dismay appears to be an inappropriate emotional reaction. But perhaps their dismay is understandable after all, as they are about to publish on the same topic and may feel that the foundations on which they have built have been somewhat shaken by our results. Turning to their specific points:

(i) We used Sternbach's (1960) criterion of counting the number of spontaneous fluctuations greater than 1 Kilohm because this happens to be a much simpler method than that used by Lader and Wing. Sternbach's (1960) own results, as well as the highly significant intercorrelations we found between spontaneous fluctuations at all times during the test, no matter what changes in skin conductance had taken place (see Table I, Greer *et al.*, 1973), support the validity of the 1 Kilohm criterion;

(ii) We were concerned to measure, not habituation to one-second sounds, but the responses of patients to arousal provoked by auditory stimuli of varying duration at irregular intervals—which might be supposed to be slightly more akin to arousal-provoking situations in real life. Incidentally, there was only one interval of 20 seconds, the remainder all being more than 40 seconds;

(iii) Like Lader and Wing (1966) we did not take into account the phase of the menstrual cycle, but our patients were tested on three separate occasions during a period of four weeks (Ramsay *et al.*, 1973). We are not aware of any published work demonstrating the relevance of McKinnon's (1954) findings to skin conductance measures in patients with anxiety states or thyrotoxicosis;

(iv) All our patients were tested between 10.30 a.m. and 11.30 a.m.

Finally, may we repeat our main finding, viz. that clinical ratings of anxiety, verified by an independent psychological test, were not correlated with any measures of skin conductance. These results cast doubt, in our view, on the *validity* of such skin conductance measures as indices of anxiety.

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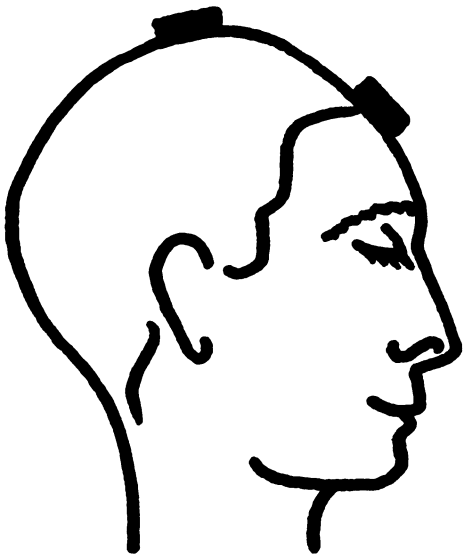
ANTERIOR BIFRONTAL ECT

DEAR SIR,

After Professor James Inglis (*Journal*, August 1970, **117**, 149–56) had suggested an improved ECT technique by electrode placement as remote as possible from the temporal lobes as is consistent with the production of a convulsion, I began to use a midline fronto-central electrode application (see Figure); and having never failed to induce a convulsion with 65 volts a.c. applied for 5–8 seconds during relaxed anaesthesia, I now use this technique routinely. However, Abrams and Taylor in their recent paper on this subject (*Journal*, May 1973, **122**, 587–90), using a low bifrontal electrode placement, appear to have failures, not all of which are overcome by adding an intravenous convulsant drug,



even with an electric current heavy enough to produce skin erythema. The technique would be unattractive with such problems, and in addition I fear that such low electrode placement over the eyes adds a risk of electric cataracts. According to Inglis (personal communication, 12 February 1973) a higher bi-frontal placement had been envisaged, reducing such risk. However, midline placement is more advantageous.

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WITHDRAWAL SYNDROME AFTER NEUROLEPTICS

DEAR SIR,

In my Psychiatric Emergency Service we often receive severely disturbed patients necessitating large dosages of neuroleptics, chiefly chlorpromazine and haloperidol; and in several cases I have noticed the emergence of more or less important extrapyramidal and dystonic symptoms upon abrupt discontinuation of this medication. The following will exemplify. About a month ago we admitted a man aged 27 who presented with a mystic-religious delirium associated with vivid auditory hallucinations in the form of celestial music played on other-worldly instruments, whereby the patient felt that he was in contact with unearthly or extrasensory forces. In this frame, he had felt compelled to burn a pornographic book while holding it in his own hands—and had suffered severe burns in carrying out this 'command'. His IQ was 93; there was no evidence of mental deterioration. On 7 May we started treatment with haloperidol (8 mg. daily i.m.), later increasing the dosage to 24 mg. i.m. plus 8 mg. orally. In addition, the patient was given 100 mg. of chlorpromazine at night. No anti-parkinson drugs were used. This treatment alleviated the delirium and hallucination, and eventually these abated. On 25 May, while haloperidol was being withdrawn, the patient became somnolent; and two days later he developed severe sialorrhea without any evidence of parkinson-like or dystonic disorders. By that time neuroleptic therapy had been discontinued altogether. Two days later, in the absence of medication and with continuing sialorrhea, the patient developed a syndrome of linguo-bucco-facial dystonia, torticollis and trismus. These symptoms were partially controlled by repeated intravenous injections of diazepam, and abated in about three days, while the sialorrhea continued for another week and was still extant, though not severe, at discharge. Now, drowsiness is known to herald an extrapyramidal and dystonic syndrome in many cases (Delay and Deniker), and