Which patients will respond to ECT?

SIR: Scott (*Journal*, January 1989, **154**, 8–17) suggests that a rise in prolactin levels following ECT may be a response to the stress of the ECT procedure, and supports his argument by citing Deakin *et al* (1983), who showed that simulated ECT increases plasma prolactin concentration twofold. However, what Dr Scott fails to mention in his article is that Deakin *et al* also showed a sixfold rise in plasma prolactin concentration following real ECT. The difference in the rise of prolactin between the two procedures was statistically significant, which allowed Deakin *et al* to conclude that the pattern of hormonal changes following ECT are specific to ECT and not simply an effect of stress. The most consistently reported endocrine effect of ECT is a rapid increase in plasma prolactin, and in fact it is so consistent and characteristic that Trimble (1978) suggested that a plasma prolactin increase may be used to differentiate an epileptic fit from hysteria.

With regard to the TRH stimulation test, and its usefulness in identifying depressed patients who will recover with ECT, we would like to draw Dr Scott's attention to Krog-Meyer *et al*'s (1984) study, which reported the results of 39 patients with unipolar depression who recovered after ECT. TRH tests were carried out before and after a course of ECT treatment. All patients received maintenance amitriptyline for 3 weeks, and then were assigned to one of two groups: one group showing an increase in Δmax TSH response to TRH greater than 2.0 mIU/ml after ECT, and the other showing a lesser increase in Δmax TSH. The first group and half of the second group received placebo for the next 6 months; the other half of the poor TSH responders received amitriptyline. At the end of 6 months the TSH response tests were repeated, and all patients received no further medication for 6 months. After 6 months, 3 of 15 patients in Group I, and 9 of 13 patients in Group II who received placebo, relapsed, while only 2 of 11 patients in Group II who received amitriptyline relapsed. The authors concluded that failure of TSH response to TRH to normalise is a poor prognostic sign in depressed patients treated with ECT and, furthermore, that maintenance with amitriptyline may prevent relapse.

The amount of cortisol released during the course of ECT may have a relationship to outcome, and in this context we would like to cite Swartz & Chen (1985), who showed that 10 of the 11 patients who responded to ECT had a progressive fall in the amount of cortisol released during the ECT course, while one non-responder showed an increase in the amount of cortisol released after the last treatment compared with the first.

In relation to EEG studied in ECT, Scott fails to mention the important work of Stromgren & Juul-Jensen (1975), who concluded that following a course of ECT, the therapeutic effect of ECT is positively correlated to the incidence and degree of EEG changes. In this study, the importance of obtaining strong universal seizures was emphasised. Failure to elicit such seizures can lead to inefficient and ineffective treatments which prolong the duration of a course.

Finally, regarding seizure threshold, preliminary results of a study we are conducting suggests that although there is a wide variation in baseline seizure threshold, patients who respond to ECT and who maintain this response at six months are those whose seizure threshold rises significantly over the course of their treatment.

Obviously, the whole area needs further exploration and it is difficult to draw any firm conclusions at this stage.

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SIR: I was interested to learn of the work of Dr Yatham *et al* concerning seizure threshold and prolonged recovery after ECT. The correspondents also highlight a few of the many physiological changes that occur during a course of ECT.

My review focused on the everyday clinical problem of predicting which treatment will be best for a particular depressed patient. If the aim is to predict, or foretell, then the observation on which the