on smaller doses of antidepressants, at the time of suicide. Although Myers & Neal (1976) found that only 5 out of 44 patients had been given antidepressants in optimal dosage at the time of the suicide, their optimal dose was based on the "manufacturers recommended full dose". Often this is less than the therapeutic full dose employed by clinicians, and therefore it is not possible to draw any definite conclusions from their results.

From these findings it is difficult to justify the authors' above recommendation. There is an urgent need for further long-term studies in unipolar depression to establish the adequate prophylactic dosage of antidepressants. Until such time as these are carried out, it seems reasonable to use 'subtherapeutic doses' for prophylaxis, just as we do with lithium in bipolar disorder and neuroleptics (depot medication) in schizophrenia.

Secondly, as the authors mention, it has been shown that the full prophylactic effect of lithium may not be achieved until after 6–12 months of treatment. If this is the case, how could one justify its use and continuation/prophylactic treatment of first choice in unipolar patients, since most patients are at risk of relapse in the first 6–12 months after recovery? For this reason I am inclined to agree with the recommendation made by Coppen *et al* (1978) that lithium prophylaxis should be used only in those unipolar patients who have had three or more attacks of depression.

As pointed out by Myers & Neal (1978), "the major difficulty in preventing suicide in psychiatric patients is to know when, in what may be a long illness, the danger periods occur". For example, in the above study the majority of suicides (28 patients) occurred during unfinished treatment with ECT or antidepressants. This suggests that the chances of suicide are greater when patients begin to recover from a severe depressive illness, and extra vigilance and care is necessary during this period. This is obviously of great importance when ECT is given to out-patients.

Again, most of the relapses are known to occur within the first 6 months of recovery after an illness. Hence careful, regular follow-up in a psychiatric clinic by an experienced psychiatrist, who knows the patient well, is as important as prophylactic drug treatment.

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Chronic Depression

SIR: The article by Scott (*Journal*, September 1988, **153**, 287–297) provides an excellent review of chronic depression. However, I do feel that she fails to emphasise the major role that alcohol abuse is likely to play in both chronic and resistant depression. Alcohol abuse is extremely common, and a recent study (Glass & Jackson, 1988) found that in two psychiatric hospitals over a 12-year period a steady 9–11% of the total number of diagnoses was accounted for by alcoholism, with 30–40% of alcoholics receiving an additional diagnosis. Most doctors, including psychiatrists, are also extremely poor at detecting alcohol problems among their patients.

Heavy alcohol use and affective illness often coexist (Dorus *et al*, 1987), and it is common clinical experience that depression rarely resolves with treatment while the patient continues to drink.

Recent work has demonstrated that one possible mechanism for this effect is the ability of alcohol to prevent the β-adrenergic receptor down-regulation that occurs with chronic antidepressant treatment (Linnoila *et al*, 1988). As the regular amount of alcohol intake in a clinical population to produce this effect is not known, great care should be taken in establishing the drinking habits of any patient with a seemingly chronic or resistant depression.

Scott mentions that some studies have demonstrated particularly low serotonergic function in chronic depression compared with acute depression, and recent work (Badawy *et al*, 1988) indicates that alcohol may be potent in decreasing serotonergic activity. Interestingly, current research also suggests that the new, highly selective serotonin reuptake blockers may have a special role to play in chronic and resistant depression.

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Failure to Convulse with ECT

SIR: The patient who fails to convulse when an electric stimulus is applied for electroconvulsive therapy (ECT) always causes concern. Sharpe & Andrew (*Journal*, January 1988, **152,** 134–136) recently described such a case and explored the possible explanations and remedies in detail. Although propafol was not used in the case described, I would like to draw attention to the effect this newly introduced induction agent has on seizure activity in ECT.

ECT was recently administered to a depressed 55-year-old male patient. The seizure duration was routinely timed, and it was observed that on two occasions this was reduced to 5 s instead of the usual 12–25 s (mean = 20 s). The intensity of the seizure was also noted to be correspondingly reduced. On both these occasions propafol had been used, as opposed to methohexitone, as the induction agent, while the doses of muscle relaxant, suxamethonium, were equivalent. Exploring this further, it appears that our anaesthetist colleagues are already aware of this phenomenon.

Propafol is a recently introduced induction agent which appears to have many advantages from an anaesthetic viewpoint, including fewer side-effects, painless administration, rapid induction and recovery, and a half-life of 1.8–8.3 min. However, Dwyer *et al* (1988) have shown in a study using patients as their own controls that propafol, compared with 1% methohexitone, reduces both observed seizure duration and duration of cerebral electrical seizure activity by an average of 25% ($P < 0.001$). They also found that it obviates the hypertensive response to ECT.

As propafol is becoming increasingly popular among anaesthetists, it is important that psychiatrists are aware of the curtailing effects it has on seizure activity. It is suggested that, until this phenomenon is explored further and propafol is shown not to reduce the therapeutic benefits, alternative anaesthetic agents are used for ECT. Propafol is easily identified as an opaque white emulsion, compared with the clear colourless solutions of methohexitone and thiopentone.

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SIR: It is with great interest that I read the letter of Pippard & Russell (*Journal*, May 1988, **152,** 712–713), regarding the paper by Sharpe & Andrew (*Journal*, January 1988, **152,** 134–136) which describes failure to convulse with ECT. Of particular interest are their assertions that "the patient resistance is unknown, but probably between 200 and 500 ohms", that the "last 4 ms of each semi-sine wave is largely ineffective because of falling potential", and finally that "owing to the rapid exponential fall of potential with this capacitor discharge type of apparatus [Transpsycon] the effective stimulus is limited to the first 0.5–0.75 s". They go on to suggest that the "use of a constant current stimulus largely compensates for variation in the patient resistance".

The new ElCoT MF-1000 ECT unit was designed with precisely these kinds of difficulty in mind. It is a microprocessor-based unit, which measures the patient's impedance continuously, once every cycle. Thus, for instance, with a brief-pulse stimulus of frequency 80 Hz, the unit measures the patient impedance 80 times per second, and readjusts the stimulus accordingly to deliver precisely the prescribed dosage. In this manner, the physician can be certain that, despite the well-documented impedance variability across patients, as well as for a given patient from day to day and even in the course of a single treatment session (Gangadhar *et al*, 1985), the stimulus delivered by the ECT unit will remain fixed.

Drs Pippard & Russell are correct in stating that "the optimum parameter levels for ECT are still uncertain". Indeed, it has been argued that the exact effects, if any, of each parameter are either unknown or, at best, ill understood. This need not be so, however. No other medical treatment is administered "blindly", to use the term of Drs Pippard & Russell, and it would appear that remaining uncertainties in ECT are due more to the inability to fully control dosage because of the difficulties mentioned above than to the nature of the treatment itself. Now that treatment can proceed in a controlled and repeatable fashion, thanks to computer technology, one can hopefully expect the publication of research results providing more and more information on the effects of duration, frequency, pulse width, potential, current, and energy on the effectiveness of ECT.

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