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Lithium augmentation

SIR: Katona *et al* (*BJP*, January 1995, **166**, 80–86) described a well-designed study of lithium augmentation. I would, nevertheless, like to highlight some weaknesses that the authors did not discuss.

It was admitted that the 'self-generated' group of anti-depressant non-responders were less refractory than patients included in previous studies. I think it is questionable whether many of them were refractory to monotherapy at all. Given that nearly half the patients in Phase II were non-compliant with lithium it seems probable that many apparent non-responders to lofepramine or fluoxetine in Phase I were actually non-compliant. Preskorn (1989) has appealed for the use of antidepressant plasma concentrations as a criterion of 'true' refractory depression. Such blood tests, although complicating a multi-centre trial, are essential if claims of treatment resistance are to be sustained. Assessing compliance by means of a blood test is far superior to tablet counts.

The assertion that the study sample had 'controlled and documented prior antidepressant treatment' is only true concerning the six week Phase I period. No summary of the lifetime treatment histories of these patients was offered. Thus patients with extensive histories of failed treatment, those most clinicians would perceive as 'refractory', were mixed up with those suffering first episodes of depression.

Finally, it was baldly stated that lithium augmentation is the most important pharmacological strategy in the management of refractory depression. I would argue, following Bridges (1983) and Quitkin (1985), that reviewing compliance with monotherapy by means of an antidepressant plasma concentration, then increasing the dose of that compound where possible, is of equal importance. A multi-centre trial comparing these two strategies is due.

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Hypomania induced by gabapentin

SIR: We report a case of a 49-year-old man with epilepsy and mild learning disabilities who developed symptoms of hypomania on starting gabapentin. The patient had a long-standing history of tonic clonic and partial complex seizures, the latter of which were difficult to control. We added gabapentin 300 mg into his existing regime of carbamazepine and lamotrigine, increasing daily by 300 mg to a maintenance dose of 300 mg t.d.s. Within the next 48 hours his behaviour began to change and he became markedly disinhibited and over familiar towards female staff on the ward, making inappropriate sexual remarks and becoming physically demonstrative. Although he had no flight of ideas, there was evidence of pressure of speech and his sleep pattern was mildly disrupted.

We reduced his gabapentin and his mental state improved without the need for psychotropic medication while maintaining good control of his partial complex seizures. Unfortunately his behaviour became aggressive and unpredictable, culminating in a violent attack on a fellow resident two weeks later. We stopped the gabapentin, and he reverted to his usual self.

The patient did have a past psychiatric history although he had not received a formal diagnosis. In 1988 he had an episode of disinhibited behaviour which in retrospect may have been hypomania although he did not receive any psychotropic treatment at the time.

Gabapentin is a relatively new anti-epileptic drug which is recommended as an adjunctive treatment for patients with refractory partial epilepsy. It is rapidly absorbed, does not bind to protein, is not metabolised, and does not affect serum concentrations of other anticonvulsants. It has been marketed as an ideal anticonvulsant pharmacokinetically. In clinical trials it showed a low relative toxicity. There have been a total of 32 reports received by the Committee of Safety of Medicine of