Reply

Sir,

The points raised by Dr. Goswami et al are only partially correct. We would like to clarify that the patient in question was receiving carbamazepine only for control of seizures, and not in combination with phenobarbitone. The other two patients too were not on phenobarbitone. Lithium does not alter after discharge threshold and seizure severity of hippocampal kindled seizures in rats after acute administration, and is unlikely to do so after prolonged used (Clifford et al 1985). Kobayashi's statement has already been referred to in our *letter*, along with a contrary observation. It only serves to highlight the divergence of opinion in the field.

They go on to suggest regarding the state of "relative CBZ withdrawal". Induced seizures have not been studied at various blood levels, which in any case fluctuate, it would be worthwhile to remember that peak CBZ levels occur more than 6 hours after (range 2-12 hrs), whereas the half life is around 20 hrs in those receiving it chronically (Kutt 1978). It is not very clear yet whether experimental seizures depend upon peak or steady state level, and which one of these the 'relative withdrawal' refers to. In any case, it is an interesting observation as it illustrates the disparity between induced and spontaneous seizures. It is not clear how PB, even if it had been given, could have accentuated the withdrawal.

ECT induces a variety of effects associated with anti kindling and anti-convulsant properties. It increases benzodiazepine binding, and potentiates GABA-ergic and Catecholaminergic mechanism in addition to various effects on peptides and hormones (Post et al 1984). These authors propose these observations as an explanation for the paradox of an anti-convulsant drug and a convulsive treatment being simultaneously efficient in the treatment of affective disorder. The mechanism of the anti-epileptic action of CBZ is not well understood. GABA-ergic, adenosine receptor and benzodiazepine receptor effects have all been implicated, as also in the affective disorders (Morselli & Lloyd 1985, Post et al 1984). We have specifically stated that CBZ may interfere with the therapeutic induction of seizures and not the treatment response per se. We agree that the site of action of CBZ is predominantly on the limbic structures.

References

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