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Letters to the Editor

Does Carbamazepine Alter the Electro-Convulsive Threshold?

Sir,

The use of Carbamazepine has increased greatly over the past few years, especially in the management of episodic psychoses resistant to other therapies. We would like to bring to the notice of readers, the possible influence of carbamazepine on electro-convulsive threshold.

A thirty year old female suffering from schizoaffective illness of 10 years duration and major epilepsy of 6 years duration was admitted to our wards. Her epilepsy was well controlled with 60 mgm Phenobarbitone OD but she was suffering frequent relapses of psychosis. Carbamazepine (200 mg BD and 400 mg HS) and Lithium (600 mg t.i.d. with a serum level of 1.0 m Eq/L) was instituted for therapeutic and maintenance purposes after Lithium alone or with antipsychotics had failed over the past few years. Two weeks trial of this combination having produced no results, we decided to add electro-convulsive therapy. In her previous episodes she had shown moderate response to ECT at less than 120 V and 0.8s electrical stimuli. However, during the present course, she failed to obtain convulsions even at 150 V and 1.0s duration stimuli given repeatedly (Direct ECT). Empirically it was decided to alter the dosage regimen of Carbamazepine to 400 mg morning and afternoon, avoiding a dose on the night prior to the ECT. With this she obtained convulsions with ECT. To confirm this observation an A-B-A-B design was executed in this patient as well as in two others (who were on similar treatment, i.e., ECT with Carbamazepine) and we consistently found

that a reduction or withdrawal of Carbamazepine on the night before ECT produced satisfactory convulsions whereas continuation of the drug, as usual, hampered induction of seizures or raised the required voltage and duration of stimuli. No changes were made in any other treatment modalities which the patients were receiving, during this trial.

These observations indicate that carbamazepine may be interfering with seizure threshold and hence with the therapeutic induction of seizures. In our experience a similar phenomenon has neither been noticed with other anticonvulsants when used with ECT for epilepsy associated psychoses, nor is a similar observation reported in literature. Majkowski (1983) has shown an increase in current intensity required for after discharge and for producing seizures in ferrets on Carbamazepine. However, Okuma (1983) reports no significant change in seizure threshold by this drug in animals. It is likely, though speculative, that the combined GABAergic effect of Carbamazepine (Okuma, 1983) and ECT (Sackeim et al., 1983) may be responsible for our observation. Interestingly, contrary to reports of Choudhary et al., (1983) and Shukla et al., (1984), none of our patients had any neurotoxic complications due to the above combination therapy.

Systematic research in this area may throw more light on the explanatory mechanism of this phenomenon and on the mode of action of Carbamazepine.

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