LETTER TO EDITOR

EFFECT OF REPEATED ELECTROCONVULSIVE SHOCKS ON AMPHETAMINE AGGREGATION-TOXICITY SYNDROME

Sir,

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Electroconvulsive shocks (ECS) are used since long in depressive states in preference to chemical convulsions, though the precise mechanism of action of ECS has been particularly studied only in recent years (4, 5). The available evidence in animals and in man strongly suggests that the benefit is derived from enhancement of one or more of the central monoaminergic functions. Chronic ECS treatment in mice significantly decreases the time of oneset of convulsions and increases the percentage mortality following subthreshold doses of some CNS stimulant drugs (7). The present work was done to study the effect of chronic ECS on functional activity of brain dopaminergic and nor-adrenergic system taking amphetamine aggregation toxicity as a model.

Male albino mice of Haffkine strain weighing 20–30 *g* were housed in groups with diet and water provided *ad libitum* except during the actual experimental procedure. A daily single ECS of 48 mA strength and 0.2 second duration was given through ear-clip electrodes for 7 days with an electro convulsiometer. No anaesthetic agent was used prior to ECS. 24 hr after the last ECS administration, mice were used for amphetamine aggregation toxicity studies during which they were housed in metal cages (23 cm x 15 cm x 15 cm). The ambient temperature was maintained at 26 $\pm$ 1°C throughout the experiment. In preliminary studies amphetamine sulphate, 6 *mg/kg* (ip) produced 100% mortality at 4 hr but not at 1 hr while a dose of 4 *mg/kg* (ip) failed to produce any mortality upto the end of 4 hr. Both doses were tested in the present work and were given to control (non-shocked) mice and ECS treated mice. An additional non-shcocked control group received only 0.9% NaCl solution (10 *ml/kg*). Mortality was assessed at the end of 1 hr, 4 hr and 24 hr. The results are shown in Table I.

Evans *et al.* (3) showed enhanced 5-HT mediated behavioural responses in rats treated with a single daily administration of ECS for 10 days. The change appears to be occurring post-synaptically. Similarly, Green *et al.* (6) showed that enhanced post-synaptic dopamine responsiveness follows single daily administration of ECS for 10 days.

Bhavsar *et al.* (2) showed that clonidine-induced NA-mediated responses were also enhanced after chronic ECS administration although Akagi *et al.* (1) found inhibition of clonidine responses. Enhanced locomotor activity and stereotyped behavioural responses to amphetamine are due to release of monoamines in central nervous system. The morta-

TABLE I : Effect of chronic administration of ECS on amphetamine aggregation-toxicity syndrome.

Group and Treatment	*Percentage mortality*		
	1 hr	*4 hr*	*24 hr*
Control : Non-shocked saline, 10 *ml/kg*	0	0	
Non-Shocked + Amphetamine 6 *mg/kg*	6	100	100
E.C.S. treated + Amphetamine, 6 *mg/kg*	100*	100	100
Non-Shocked + Amphetamine, 4 *mg/kg*	0	0	0
E.C.S. treated + Amphetamine, 4 *mg/kg*	40*	60*	60*

Each group had 50 mice, all drugs were given ip.

*Value differs significantly ($P<0.05$) from the respective control group (Chi^2 test).

lity in ECS-treated group with subthreshold dose of amphetamine appears to be due to either increased neuronal sensitivity and/or increased post-synpatic sensitivity to dopamine and noradrenaline.

Our study further substantiates that chronic administration of ECS enhances the sensitivity to various neurotransmitters in the central nervous system.

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