LETTER TO EDITOR

EFFECT OF REPEATED ELECTROCONVULSIVE SHOCKS ON AMPHETAMINE
AGGREGATION-TOXICITY SYNDROME

Sir,

(Received on June 21, 1982)

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 onset 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 ± 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 up to 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-shocked
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 1.

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.

<table>
<thead>
<tr>
<th>Group and Treatment</th>
<th>Percentage mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 hr</td>
</tr>
<tr>
<td>Control:</td>
<td></td>
</tr>
<tr>
<td>Non-shocked</td>
<td>0</td>
</tr>
<tr>
<td>Non-shocked + Amphetamine</td>
<td></td>
</tr>
<tr>
<td>6 mg/kg</td>
<td>100*</td>
</tr>
<tr>
<td>E.C.S. treated</td>
<td></td>
</tr>
<tr>
<td>+ Amphetamine</td>
<td></td>
</tr>
<tr>
<td>6 mg/kg</td>
<td>100*</td>
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<tr>
<td>Non-shocked + Amphetamine</td>
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<tr>
<td>4 mg/kg</td>
<td>60*</td>
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<td>+ Amphetamine</td>
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<tr>
<td>4 mg/kg</td>
<td>60*</td>
</tr>
</tbody>
</table>

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

*Value differs significantly (P<0.05) from the respective control group (Chi² test).

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

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

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REFERENCES


