EFFECT OF DI-HYDRO-B-ERYTHROIDINE ON THE SOMATOSENSORY EVOKED POTENTIALS IN THE RAT

Sir,

Application of strychnine or d-tubocurarine to the exposed cerebral cortex, increases the amplitude of the early negative wave of the somatosensory evoked potentials (1). Peripheral neuromuscular blocking drugs, which are structurally similar to curare, e.g. toxiferine and alloferin, when applied to the cerebral cortex also produce strychnine like action on the cortical evoked potentials. Similarly the neurotoxic fraction of the cobra venom also produces a long-lasting abnormal negative wave in the cortical evoked potentials (2) while at neuromuscular junction it produces a non-depolarizing blockade of the cholinergic transmission. Involvement of a cholinergic synapse for the action of these substances is suggested by the observation that treatment of the rat cortex with eserine blocks the effect of strychnine, curare and alloferin on somatosensory evoked potentials (3).

Di-hydro-B-erythroidine has a curare like neuromuscular blocking action, but is structurally unrelated to curare. It would be interesting to find if it has a similar action on the acetylcholine mediated receptors on the cortex.

In rats (CFE strain from Carworth, Europe) lightly anaesthetised with pentobarbitone, the averaged somatosensory evoked potentials were recorded according to the techniques described earlier (2). Computer derived averages of 32 consecutive somatosensory evoked potentials in response to the stimulation of contralateral and ipsilateral forepaws, were obtained from both cortices. Di-hydro-B-erythroidine, an alkaloid possessing curariform properties at neuromuscular junction, dissolved in CSF was applied to the pial surface in different concentrations (5×10^{-4}M, 10^{-3}M, 2×10^{-3}M) for a period of 15—30 minutes. In all experiments the drug was applied to only one cortex while the other cortex was bathed with normal CSF and served as control. Spontaneous electro-cortical (EEG) activity, cortical and rectal temperatures were continuously monitored on a polygraph.

Di-hydro-B-erythroidine in most of the experiments failed to produce any effect on the primary cortical evoked potentials. It also had no effect on the repetitive after discharges. In the control cortex the evoked potentials following contralateral or ipsilateral stimulation were unchanged.

Only strychnine, curare, alloferin and toxiferine which are structurally related to curare produced the strychnine like action on the cortical evoked potentials. Other substances like Di-hydro-B-erythroidine which though possess a curare like action at the neuromuscular junction do not have a similar action on the cortical evoked potentials. It is possible there

fore that the nature of cortical receptors on which curare acts is different from the recepto at neuromuscular junction which are blocked by curare.

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