INFLUENCE OF SOME ANTIARRHYTHMIC AGENTS ON ELECTRICALLY INDUCED CONVULSIVE SEIZURES IN RATS*

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The anticonvulsant drug, phenytoin was found to be effective in experimentally induced arrhythmias (5). It has been found useful even clinically (1). The antiarrhythmic effect of the drug seems to be related to its stabilizing effect on hyperexcitable tissues (4). This prompted us to study the anticonvulsant activity of known antiarrhythmic agents.

MATERIALS AND METHODS

160 adult albino rats o either sex and weighing 140—175 *g* were used for this study. The test procedure employed was the supramaximal electroshock technique described by Toman *et al.* (9). An alternating current of 150 mA and 0.2 sec duration was delivered by ear clip electrodes from the electroshock apparatus (TECHNO).

The rats exhibiting the typical tonic extensor phase of the hind limbs were selected for further work. They were divided into eight equal groups and drugs were administered as indicated in Table 1. After 30 min the modification of the supramaximal shock pattern was studied in each animal.

Statistical analysis was done by 'Chi square' test.

TABLE I

Effect of some antiarrhythmic drugs on tetanic convulsions in rats.

Drug (dose and route)	*Number of animals*	*% of animals showing abolition of tonic extensor phase*	*Significance by 'Chi square' test*
Distilled water (orally)	20	0	—
Phenytoin sodium (30 *mg/kg* orally)	20	100	$p<0.001$
Xylocaine hydrochloride (35 *mg/kg* ip)	20	100	$p<0.001$
Procaine hydrochloride (30 *mg/kg* ip)	20	50	$p<0.05$
Procainamide hydrochloride (30 *mg/kg* ip)	20	5	p—insignificant
Quinine hydrochloride (100 *mg/kg* ip)	20	25	p—insignificant
Quinidine sulphate (20 *mg/kg* ip)	20	0	p—insignificant
Chloroquine sulphate (25 *mg/kg* ip)	20	15	p—insignificant

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RESULTS

The results are presented in Table I. The interval between drug administration and electroshock was usually 30 min except in the case of phenytoin group where it was 60 min and procaine group where it was 15 min. In phenytoin and xylocaine groups complete abolition of hind limb tonic extensor phase was noted. However, there was an exaggeration of clonic phase. In procaine group also, there was a significant protection against supramaximal shock. Procainamide, quinine, quinidine and choloroquine did not influence convulsant effects in a significant way.

DISCUSSION

It is apparent from the results that xylocaine and phenytoin offered 100% protection. The anticonvulsant activity of xylocaine has been described by Essman (2) and is confirmed by our findings. Procaine, afforded only 50% yet significant ($P<0.05$) protection. Our results are consistent with the observations of Tanaka *et al.* (7).

Choroquine and quinine could offer protection in a few animals but this was not significant. The absence of anticonvulsant property of chloroquine has already been reported (6). Quinidine and procainamide failed to protect.

The effects of procaine and xylocaine on CNS are complex, since in toxic doses they produced convulsions and death (8).

The convulsant effects of these drugs are probably due to inhibition of central inhibitory synapses (8). In lower doses we find the anticonvulsant effect. Similar inhibitory mechanism might be operative in low doses which prevent maximal interneuronal facilitation occuring during tonic extensor phase.

SUMMARY

The effects of antiarrhythmic drugs on supramaximal electroshock pattern was studied in rats. Phenytoin, xylocaine and procaine offered significant protection. The others failed to offer any significant protection.

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