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THE EFFECTS OF 5-HYDROXYTRYPTAMINE ON ISOLATED RIGHT AND LEFT RAT ATRIA

Parimongkol Wongchuengam[*], *Prasan Dhummaupakorn*[**] *and Prakorn Chudapongse*[**]

* *Buddhachinaraj Nursing College, Phitsanulok and* ** *Department of Pharmacology, Faculty of Pharmaceutical Science, Chulalongkorn University.*

Male albino rats weighing 250-300 gm were killed and the left and right atria were separated. The rate and contractile force were recorded with isometric force transducer connected to a recorder. The frequency of stimulation was kept constant at 250 beats per minute. The 5-hydroxytryptamine (5-HT) produced the dose dependent positive chronotropic and inotropic effects on the right and left atria respectively. Cyproheptadine (0.02 µg/ml) and methysergide (0.47 µg/ml) reduced the positive chronotropic effect of 5-HT (2 µg/ml) to about 50% of controls, but completely abolished the positive inotropic effect of 5-HT on the left atria. Propranolol (0.15 µg/ml) attenuated the positive chronotropic effect of 5-HT similar to those of methysergide and cyproheptadine. However, propranolol did not reduce the positive inotropic effect on the left atria. Combination of two antagonists, cyproheptadine and propranolol, or methysergide and propranolol significantly depressed the positive chronotropic effect of 5-HT on the right atria. The percentage of the reduction in left atrial isometric contraction was similar to those produced by either cyproheptadine or methysergide alone. The positive chronotropic and inotropic effects of 5-HT in reserpinized rats (5 mg/kg/day i.p. for two consecutive days) were slightly less than those observed in nonreserpinized rats. However, cyproheptadine or methysergide dramatically reduced the positive chronotropic effects which significantly differed from nonreserpinized rats.

It is concluded that 5-HT mediatesthe positive chronotropic and inotropic effects on isolated rat right and left atria respectively by different mechanisms. The effect on right atria may be due to the combined direct effect on 5-HT receptor and indirect effect which is most likely the release of catecholamine from the adrenergic nerve in right atria. The positive inotropic effect is mainly due to a direct action of 5-HT and does not involve endogenous catecholamine release.