SHORT COMMUNICATION

ADRENOCEPTORS IN THE HEART OF RANA TIGRINA

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Summary: The order of potency for the inotropic effect in Rana tigrina heart was found to be isoprenaline > adrenaline > phenylephrine. The effect of adrenaline is reduced and that of phenylephrine and isoprenaline was unchanged after DHE perfusion. Subsequent perfusion with dichloroisoproterenol blocked the cardiac stimulant effect of all the three agents completely. It thus appears that the heart of Rana tigrina contains both the types of adrenoceptors and the cardiac effects of isoprenaline and phenylephrine are due to activation of beta adrenoceptors while those of adrenaline are due to activation of both the receptors.

Key words: adrenoceptors heart Rana tigrina

The effects of sympathomimetic amines on the hearts of Rana pipens and Rana temporaria are due to activation of beta-adrenoceptors (3, 4). On the other hand Pradhan (5) has reported that the effects of adrenaline on the heart of Rana tigrina are reduced after dihydroergotamine treatment and has suggested that this is due to the presence of a significant number of alpha-receptors in the frog heart. In view of the fact that Rana tigrina is most commonly employed in India for experiments, the reported differences could be due to a variation in the species used. In the present study, therefore, the nature of adrencoceptors in the heart of Rana tigrina have been investigated, in some more details.

MATERIALS AND METHODS

Isolated frog (Rana tigrina) heart was set up as per the technique of Bulbring (2) and was perfused with frog Ringer solution at a constant pressure of 40-60 mm.Hg. The adrenoceptor agonists used were dl-isoprenaline hydrochloride, dl-adrenaline hydrochloride, phenylephrine hydrochloride, while the adrenoceptor blocking agents employed were, dihydroergotamine tartrate (DHE 45) and dl-dichloroisoprenaline hydrochloride (DCI). The solutions of all the agents were freshly prepared and the experiments were conducted from June to August at room temperature (28-30°C).

Equi-effective concentrations of isoprenaline, adrenaline and phenylephrine, producing 60% of the maximum response, were first determined and the effect of these concentrations was then observed during perfusion with DHE 45 ($1x1^{-6}$ *g/ml*) and DCI ($6.5x10^{-6}$ *g/ml*) over a period of 60 min. Concentration response curves with phenylephrine and adrenaline were also recorded before and after perfusion of DHE 45 ($1x10^{-7}$ *g/ml*) for 30 min. (n=5 each).

RESULTS

The range of equieffective concentrations for the positive inotropic effect for isopenaline, adrenaline and phenylephrine were 8.5×10^{-12} to 1.7×10^{-8} *g*., 8.3×10^{-9} to 1×10^{-7} *g*. and 2.05×10^{-6} to 3.3×10^{-7} *g* respectively (n=15). Thus the order of potency was isoprenaline < adrenaline phenylephrine. The effect of isoprenaline and phenylephrine remained unchanged after DHE 45 perfusion, but the effect of adrenaline was reduced by about 10 to 30%. However, subsequent perfusion with DCI completely blocked the effect of all the three drugs (Fig. 1). After perfusion

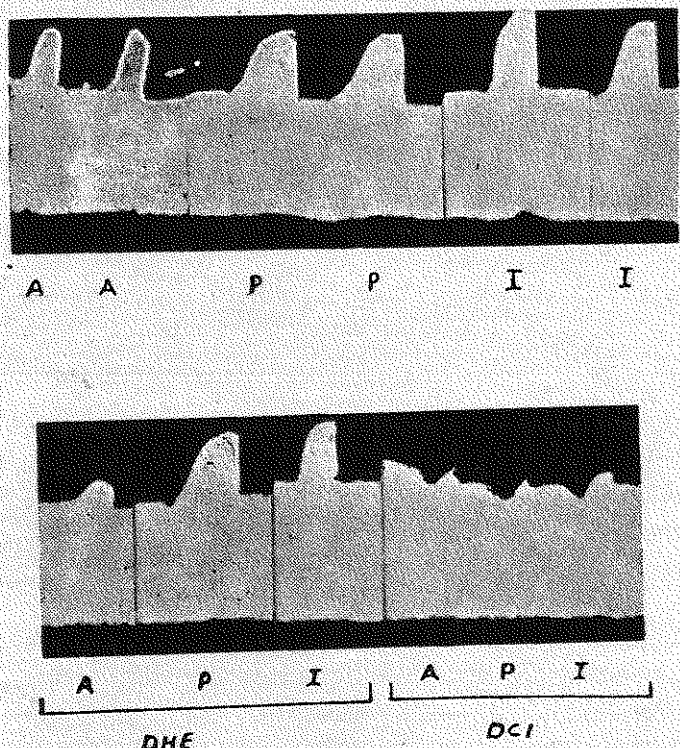

Fig 1: Record of isolated heart of frog (R. Tigrina) showing the effect of adrenaline (at A), Phenylephrine (at P) and isoprenaline (at I) before (Upper Pannel) and during DHE 45 perfusion (⟝ DHE 1×10^{-6} *g/ml* and) DCI perfusion (⟝ DCI 0.5×10^{-6} *g/ml*). (Lower Pannel).

with DHE 45 the concentration response curve of phenylephrine was not affected but that of adrenaline was shifted to the right, significantly (Fig. 2).

DISCUSSION

The observations indicate that in the heart of R. tigrina both the types of adrenoceptors are present and are active at room temperature (28-30°C). This is in contrast to the reported results for R. pipiens where only beta-receptors have been reported to be active at this temperature (1).

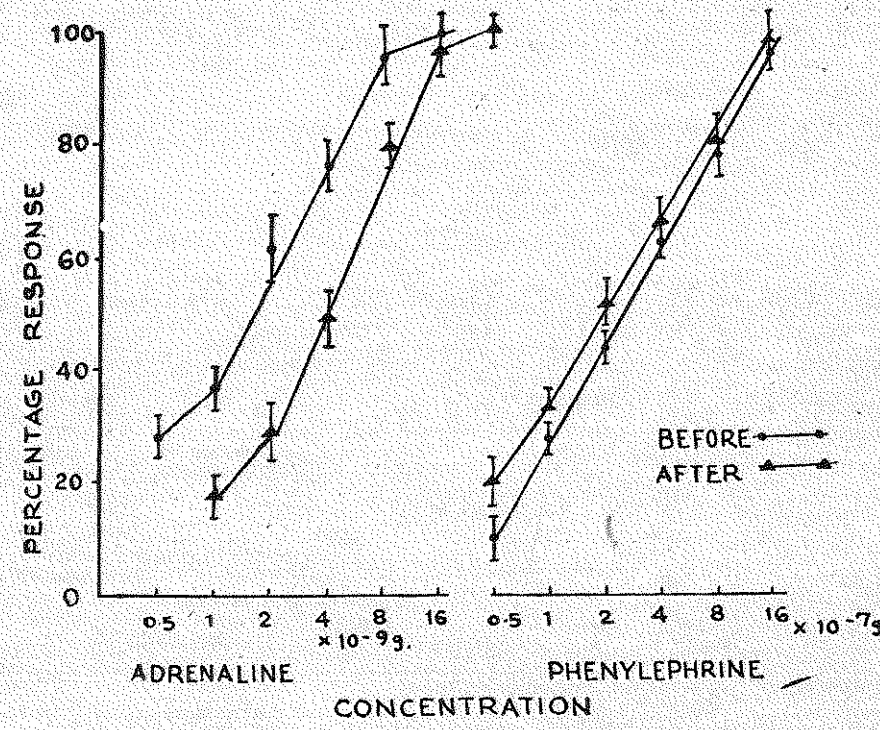

Fig. 2: Dose response curves of adrenaline and phenylephrine before and after DHE 45 ($1x10^{-7}$ g/ml for 30 min.) perfusion.

The proportion of the beta-receptors, however, appears to be far more than those of the alpha-receptors as indicated by the order of potency of the different agonists. Reduction in the response to adrenaline after perfusion with DHE 45 is in agreement with that reported by Pradhan (5) and is in favour of the presence of both the types of adrenoceptors. It is interesting that the cardiac stimulant effect of phenylephrine, a predominent alpha-activator, was not blocked by DHE 45 but was blocked by DCI, indicating that the effect is mediated through the activation of beta-receptors. Such a possibility has also been suggested by Erlij *et al.* (3).

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