THE EFFECTS OF CATECHOLAMINES AND THEIR ANALOGUES ON MOTONEURONES AND SPINAL REFLEX RESPONSES IN THE ISOLATED SPINAL CORD OF NEWBORN RATS

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The present experiment was carried out to investigate the effects of catecholamines and their analogues on motoneurones and spinal reflex responses using the isolated spinal cord of newborn rats.

1) Noradrenaline ($10^{-7}-5\times10^{-5}$M), adrenaline ($5\times10^{-6}-10^{-5}$M), dopamine ($10^{-6}-2\times10^{-4}$ M), octopamine ($2\times10^{-5}-10^{-3}$ M), and isoproterenol (10^{-3} M) caused depolarization of motoneurones in a concentration dependent manner. ED_{50} was 3×10^{-6} M for noradrenaline, 10^{-6} M for adrenaline, 5×10^{-5} M for dopaimne, and 10^{-4} M for octopamine. The depolarizing effect of these drugs persisted in Ca^{2+} deficient Krebs solution containing Mg^{2+} 3.5 mM.

2) The effect of noradrenaline and adrenaline on the monosynaptic reflex response (MSR) varied in different preparations such as inhibition and potentiation followed by inhibition and potentiation. On the other hand, the polysynaptic reflex response (PSR) was always inhibited by these drugs. Isoproterenol caused only a limited inhibition of either MSR or PSR.

3) Dopamine and octopamine inhibited both MSR and PSR in a concentration dependent manner. The rate of inhibition of MSR was almost the same as that of PSR. The concentration producing 50% inhibition of MSR was 4×10^{-5} M for dopamine and 2×10^{-4} M for octopamine.

4) Both depolarization and inhibition of reflex responses induced by the catecholamines and their analogues were blocked by phentolamine ($2.7\times10^{-6}-2.7\times10^{-5}$ M), phenoxybenzamine (5×10^{-6} M), and chlorpromazine (10^{-5} M) but not by propranolol (5×10^{-6} M). Haloperidol (10^{-5} M) failed to inhibit the effect of dopamine.

5) Adamantanamine ($10^{-3}-10^{-2}$ M) depolarized motoneurones and inhibited both MSR and PSR. All of these effects were blocked by phentolamine but not by either propranolol or haloperidol.

6) These results suggested that catecholamines and their analogues cause depolarization of motoneurones and inhibition of spinal reflex responses due to the activation of α-receptors which presumably are located in the cell body, the dendrite of motoneurones, and the primary afferent terminals.