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Motor transmission in the vas deferens of guanethidine-treated guinea-pigs

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There are reasons to believe that motor transmission to the longitudinal muscle of guinea-pig vas deferens is non-adrenergic (Ambache & Zar, 1971; Euler & Hedqvist, 1975). Recently, Furness (1974) has advanced fresh evidence against this view. He has found a great reduction in the nerve-mediated contractions of the vas deferens from guanethidine-treated guinea-pigs; the attenuated contractions were restored to normal levels by exposing the isolated preparation first to (+)-amphetamine (DA) and then to noradrenaline (NA). He has interpreted these results to indicate that NA is the motor transmitter in this tissue.

The present experiments were carried out to determine whether or not repletion by exogenous NA of depleted NA stores was an essential pre-requisite for the restoration of nerve mediated responses in the vas deferens.

Vasa from guanethidine-treated guinea-pigs (100 mg/kg, i.p., 24 h earlier) were set up in 10 ml. organ baths in Krebs; contractions on electrical field stimulation (5 pulse-trains, 1 ms, 10 Hz, 12 V, once every min.) were recorded isometrically. The preparations were exposed first to DA, 1 μg/ml for 15 min, then to NA, 0.5 μg/ml for 6 minutes. Twenty minutes after NA-wash-out, DA, 1 μg/ml was re-introduced into the bath. In control contralateral vas, exposure to NA was omitted.

In all experiments ($n=7$) nerve-mediated contractions were virtually completely lost; restoration of the responses, after exposure for 15 min to DA and 6 min to NA was partial ($8.4 \pm 1.7\%$) and substantially lower than in control (NA-untreated) contralateral preparations ($33 \pm 3\%$). Longer exposure to DA (> 90 min) was needed for complete recovery. There was no evidence that treatment with NA facilitated recovery.

In 3 experiments on vasa from guinea-pigs treated 24 h earlier with (−)-β-hydroxyphenethylguanidine (100 mg/kg, i.p.), a more potent agent than guanethidine in depleting NA stores (Fielden & Green, 1967), motor transmission persisted unimpaired.

Histochemical examination of the vasa from guanethidine or (−)-β-hydroxyphenethylguanidine-treated animals showed a total lack of catecholamine fluorescence. Catecholamine fluorescence was missing in vasa from guanethidine-treated animals even after complete recovery of motor transmission on prolonged exposure to DA.

These findings do not lend support to Furness's conclusion (1974) that the reversal of guanethidine-induced block of motor transmission in the vas deferens was dependent upon the repletion of NA-stores and therefore the transmission was adrenergic. The lack of correlation between NA-depletion and motor blockade is consistent with the non-adrenergic nature of motor transmission in this organ.

We thank Dr R. Fielden, Smith Kline & French Ltd for the gift of (−)-β-hydroxyphenethylguanidine.

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