renaline. The centre electrode was then advanced ($<60 \mu m$) to penetrate the cell. Following successful impalement, the intracellular and extracellular activity was again recorded in response to the agents.

Iontophoretic application of DLH and glutamate reduced the VRF and caused a late positive wave. Cell firing could be evoked by both agents, that to glutamate being less marked and of shorter duration than that to DLH. The VRF did not change, or increased slightly in response to noradrenaline. The pattern of firing evoked by the amino acids was not significantly different before and after motoneurone penetration. Extracellular recordings during the depolarization and firing caused by DLH and glutamate showed that the negative wave of the VRF was reduced and the typical late positive wave appeared. Noradrenaline typically caused a hyperpolarization and inhibited the depolarizations and firing evoked by DLH and glutamate.

We conclude that the act of cell penetration does not in itself qualitatively alter the response of motoneurones to extracellular iontophoresis of DLH and glutamate and that these agents decrease and 'reverse' the VRF when the cells are depolarized. Thus an increase of VRF during biogenic amine iontophoresis (Barasi & Roberts, 1977) does not necessarily indicate a neuronal depolarization but rather a hyperpolarization with action potential increase (Engberg, Flatman & Kadzielawa, 1976).

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Pharmacological study of the anococcygeus muscle of the dog

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Pharmacological study of the anococcygeus muscle of rat and cat have been carried out (Gillespie, 1972; Gillespie & McGrath, 1974). In the present experiments the anococcygeus muscle of mongrel dogs of both sexes, weighing 2-6 kg, were used. The muscle was dissected out as described for rat anococcygeus muscle (Gillespie, 1972) and suspended in oxygenated Tyrode solution (37°C). The muscle showed no spontaneous contraction. Field stimulation of the muscle (0.5 ms, supramaximal voltage) produced frequency dependent motor responses which were blocked by guanethidine $(4 \times 10^{-6} \text{ m})$ and phentolamine $(3 \times 10^{-7} \text{ M})$ but unaffected by hexamethonium, atropine, promethazine or methysergide. When the tonus of the muscle was increased by guanethidine $(1 \times 10^{-4} \text{ M})$ or carbamylcholine $(3 \times 10^{-5} \text{ M})$; field stimulation caused the muscle to relax. The degree of relaxation was frequency dependent and the responses were not blocked by propranolol (3 \times 10⁻⁶ M). Minimal and maximal contractions of the muscle were achieved by noradrenaline (10⁻⁷ to 10⁻⁵ M), tyramine $(10^{-6} \text{ to } 10^{-4} \text{ m})$, acetylcholine $(10^{-6} \text{ to } 10^{-4} \text{ m})$ M), 5-hydroxytryptamine (10⁻⁸ to 10⁻⁶ M), histamine $(10^{-7} \text{ to } 10^{-4} \text{ m})$ and by higher doses of isoprenaline (10⁻⁶ to 10⁻⁴ M). The effects of noradrenaline, tyramine and isoprenaline were blocked by phentolamine. The effects of acetylcholine, 5-hydroxytryptamine and histamine were blocked by atropine, methysergide and promethazine (but not cimetidine) respectively. Lower doses of isoprenaline (10⁻⁹ to 10⁻⁷ M) relaxed the muscle when the tonus of the muscle was raised by guanethidine. The results suggest that the response of the anococcygeus muscle of the dog to field stimulation and to pharmacological agents is similar to the responses of the anococcygeus muscle of the rat except that in the dog the muscle has also H₁ receptors and beta adrenoceptors.

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