Electrically evoked release of vasopressin from isolated neurohypophyses in sodium-free media

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Electrical stimuli applied to the stalk of the neurohypophysis evoke release of posterior pituitary hormones (Douglas & Poisner, 1964) and this effect is dependent on impulse formation (Mikiten, 1967; Ishida, 1970). It is generally assumed that impulses act by depolarizing the neurosecretory terminals and setting in motion some calcium-dependent process since elevation of extracellular potassium also evokes secretion from these preparations and the response, like that to electrical stimulation (Mikiten & Douglas, 1965) requires calcium (Douglas & Poisner, 1964). We have now obtained further support for this assumption from experiments in which electrical stimulation of the endings has been demonstrated to be an effective stimulus for hormone release under conditions which preclude impulse formation and where the effect of the stimulus is therefore referable to a direct, presumably depolarizing, action on the terminals.

In each experiment five rat pituitary glands were halved and the ten halves successively skewered on a single platinum needle-shaped electrode so that no bare electrode appeared between the pieces of gland. A small rubber stopper served to retain the glands and conceal the remaining exposed tip of the electrode. The whole assembly, resembling a shish-kebab, was then immersed in 1 ml of incubation medium and could be stimulated by current passed between the gland-bearing electrode and a second platinum electrode in the incubation medium. The arrangement is essentially similar to that described by Mikiten (1967). The glands were transferred to fresh incubation medium at 10 min intervals. After incubation for 30–60 min in Na-free Locke's solution (Na replaced by sucrose) they were stimulated (20 Hz; 1 ms duration; 5 mA) for the first 5 min of the succeeding 10 min period, the polarity of the electrodes being changed at 10 s intervals. Bioassay of the medium (Douglas & Poisner, 1964) showed that such stimulation caused a 3–8-fold increase in vasopressin output over the preceding period in ten different experiments. The addition of tetrodotoxin (2×10^{-6} M) to the Na-free medium did not prevent the response but omission of calcium did. The response was inhibited by Mg (10 mM).

We interpret these results to mean that direct depolarization of the terminals independent of sodium entry or impulse propagation provides an adequate stimulus for vasopressin release provided calcium is present.

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