after reserpine pretreatment is due to a blockade of NA uptake and storage rather than a change in the sensitivity of the postsynaptic receptor. The increased neuronal responses to iontophoretically applied NA which have been observed after iontophoretic application of imipramine and of desipramine and after destruction of adrenergic terminals with 6-hydroxydopamine (Avanzino, Ermirio & Zummo, 1971; Hoffer, Siggins & Bloom, 1971) support this suggestion.

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Microinjection study of the rôle of adrenergic transmission in the control of the secretion of antidiuretic hormone

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Particularly abundant supplies of noradrenaline-containing nerve-terminals have been demonstrated by histochemical methods in the hypothalamic nuclei concerned with antidiuretic hormone (ADH) release, namely the supraoptic nucleus (SON) and paraventricular nucleus (PVN) (Fuxe, 1965). However, comparatively little is known about the rôle of adrenergic transmission in ADH release. In a series of experiments on cats anaesthetized with chloralose, we studied the effects of the microinjection of noradrenaline (NA) and adrenoceptor blocking drugs into the SON on ADH secretion. The drug solutions, adjusted to plasma pH and osmolarity, were injected in volumes not larger than 1 μl through stereotactically placed steel cannulae.

The position of the cannulae tips was determined from frozen sections, stained with cresyl violet and luxol blue. Control injections of normal saline were made to ensure that experimental procedures had no effect on the concentrations of blood ADH.

Blood samples (4 ml) were withdrawn from an external jugular vein and extracted according to the method of Bisset, Hilton & Poisner (1967). The extracts were assayed for antidiuretic activity on rats anaesthetized with alcohol (Bisset, 1962). Arterial blood pressure was monitored during the injection sampling sequences. Noradrenaline (5–30 μg), invariably caused release of ADH. In the same cat, the response was dose dependent, although it varied considerably in size between different animals. In six experiments (in which NA (20 μg) was administered) the percentage increase of ADH in samples taken 2 min after the injection of 20 μg NA, compared with samples taken 2 min before, varied between 80% and 400%. The effects of two adrenergic blocking agents, phentolamine and propranolol, on the release of ADH by NA were studied. Pretreatment with phentolamine did not always prevent the NA-induced release of ADH. In the dose used (75 μg) phentolamine itself caused a release of ADH in four experiments out of five. The percentage increase varied between 67 and 525%.

Propanolol (30 μg), on the other hand, did not affect the basal ADH concentration, but did block the ADH release normally expected from injections of 20 μg NA.

Neither drug had any effect on the release of ADH after an injection of 1 μl hypertonic (1 M) saline.

The stimulating effect of NA on the secretion of ADH is to some extent contrary to the depression of electrical activity in antidromically identified neurosecretory cells on microelectrophoretic application of NA, as observed by Barker, Crayton & Nicoll (1971). However, previous work on cholinergic transmission using the same microinjection technique (Milton & Paterson, 1970) is in agreement with the findings of Barker *et al.* (1971). It is possible that two adrenergic pathways, one inhibitory and one excitatory, converge on the SON. This would also explain the different effects of α- and β-receptor blocking agents on the secretion of the hormone.

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Differential effects of 6-hydroxydopamine on the terminals and non-terminal axons of adrenergic neurones

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In the chick intravenous injection of 6-hydroxydopamine (6-OHDA) causes terminal adrenergic nerves to degenerate, but non-terminal axons survive (Bennett, Burnstock, Cobb & Malmfors, 1970; Bennett, 1971; Cobb & Bennett, 1971). Since 6-OHDA has to be taken up by the nerves before degeneration occurs (Bennett *et al.*, 1970), the apparently selective effect on nerve terminals could be explained by the finding that the membrane uptake mechanism is more efficient in terminal fibres than in non-terminal axons (see Hamberger, Malmfors & Stjärne, 1971). However, blockade of axoplasmic transport by vinblastine produces changes in adrenergic nerves similar to those seen after treatment with 6-OHDA (Bennett, Cobb & Malmfors, 1971). In the work described here the possibility that axoplasmic transport is involved in the differential effects of 6-OHDA has, therefore, been investigated.

Adrenergic nerves in tissues, inferior vena cava and coccygeomesenteric vein, from 2-week-old White Leghorn chicks were examined using the Falck-Hillarp fluorescence technique (Falck, 1962). Intravenous injection (six experiments) of 100 mg/kg 6-

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