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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OHDA (H44/68, Hässle, Sweden) caused, within 6 h, loss of fluorescence from many terminal fibres, but a marked increase in the fluorescence intensity of non-terminal axon bundles.

Incubation of tissue, from untreated animals, for 6 h in an oxygenated saline solution (Ginsborg, 1960) at 37°C, containing 1×10^{-4} g/ml 6-OHDA (six experiments), caused loss of fluorescence from most terminal fibres. However, there was a marked increase in fluorescence intensity at the cut ends of non-terminal axon bundles, indicating that axoplasmic transport of catecholamines was not blocked. Thus, after intravenous injection of 6-OHDA, accumulation of catecholamines could occur in non-terminal axon bundles, and thereby produce the observed increase in fluorescence intensity.

This possibility was tested by injecting six chicks intravenously with 10 mg/kg vinblastine ('Velbe', Lilly), because it has been shown that such treatment blocks axoplasmic transport of catecholamines within 1 h (Bennett *et al.*, 1971); 1 h after the vinblastine injection, the animals were given 100 mg/kg 6-OHDA (i.v.). Control groups (six animals each) received vinblastine or 6-OHDA alone, and tissues from control and treated animals (injected with vinblastine plus 6-OHDA) were examined 7 h after the first injection. There were no detectable changes in the fluorescence morphology of adrenergic nerves in tissues from animals injected with vinblastine alone. However, in tissues from animals injected with 6-OHDA alone there was a marked reduction in the number of terminal fibres detectable, while the non-terminal axon bundles showed increased fluorescence intensity. In animals treated with vinblastine and 6-OHDA the reduction in number of fluorescent terminal fibres was still apparent, but the non-terminal axon bundles also showed a marked loss of fluorescence. One day after this treatment the destruction of non-terminal axon bundles appeared to be more extensive than after treatment with 6-OHDA alone.

We conclude that 6-OHDA has no detectable effect on axoplasmic transport, at least of catecholamines. Indeed, it appears that proximodistal transport might carry 6-OHDA from the non-terminal axons into the terminal fibres and thus contribute to the differential effects of the drug.

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