The data presented here suggest that the active accumulation of mescaline into synaptosomes from the cerebral cortex is not brought about via the noradrenaline uptake mechanism. Furthermore, since the uptake of mescaline was not affected by desipramine, uptake blockade cannot explain the potentiation of neuronal responses to mescaline by desipramine.

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Depleting effects of reserpine on intracellular catecholamines in rat coeliac-mesenteric ganglion

J.D. LEVER, K.-S. LU, R. PRESLEY* & R.M. SANTER

Department of Anatomy, University College, Cardiff

Since the introduction of formol-induced fluorescence methodology for the histochemical demonstration of biogenic amines these have been studied in sympathetic ganglia not only in adrenergic post-ganglionic neurones but also in small intensely fluorescent (sif) cells (Eranko & Harkonen, 1963) present in these ganglia. Sif cells have been equated (Grillo, Jacobs & Comroe, 1974) with the small granular cells revealed by electron microscopy in sympathetic ganglia (Williams & Palay, 1969). Recently, Lever, Lu, Presley & Santer (1974) have demonstrated chromaffin-positive (CH+) small cells with a similar distribution to sif cells in a variety of sympathetic ganglia employing glutaraldehyde tissue fixation prior to dichromate treatment. Current speculation suggests that small cells in sympathetic ganglia may have an inhibitory effect on post-ganglionic transmission acting either interneuronally or in a local endocrine capacity.

The aim of the present investigation was to test the lability of sif and CH+ cells to reserpine in terms of their specific fluorophore emission and chromaffin positivity respectively in adult rat coeliac-mesenteric ganglia. Two reserpine dosage schedules were applied (a) 5 mg/kg i.p. 6 h before sacrifice and (b) 5 mg/kg i.p. at 36, 24 and 12 h before the animals were killed. Control animals were correspondingly injected with saline. After single injections of reserpine there was a statistically significant ($P < 0.001$) reduction in the % of chromaffin-positive and a statistically significant ($P < 0.001$) increase in the % of chromaffin-negative small cells compared with controls. Although after single injections of reserpine specific amine fluorophore was not detected in principal ganglionic neurones, no obvious reduction in fluorophore emission from sif cells was apparent. However, after prolonged reserpinization (3 x 5 mg/kg) not only was there a highly significant reduction in the % of chromaffin-positive small cells but fluorimetric measurements from sif cell cytoplasmic areas were significantly ($P < 0.001$) lower (by a factor of 2) than from comparable cells areas in control animals.

Our results are more definitive than those of Van Orden, Burke, Geyer & Lodoen (1970) who reported 'slight variable reduction in fluorescence intensity' from sif cells following two reserpine injections.

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The effects of nicotinic and muscarinic agonist drugs on the release of catecholamines from the isolated perfused adrenal glands of the dog

J.A.J.H. CRITCHLEY*, J. ISOBEL TIBENHAM, A. UNGAR, J. WAITE & CHRISTINE P. WEST

Department of Pharmacology, University of Edinburgh, 1 George Square, Edinburgh EH8 9JZ

Douglas & Poisner (1965) found that, in isolated perfused adrenal glands from cats, pilocarpine released almost solely adrenaline, whereas nicotine released about equal proportions of adrenaline and noradrenaline.

Working with anaesthetized animals, Critchley, Ungar & Welburn (1973) found that specific sensory stimuli in the cat caused reflex release of either mainly adrenaline or mainly noradrenaline. However, when similar sensory stimuli were applied to dogs the release occurred without a change in the ratio of adrenaline to noradrenaline. We decided to extend the work of Douglas & Poisner to the dog, using the more specific nitocinic and muscarinic agonists that are now available. We wanted to see whether cats differ from dogs in their selective responses to cholinergic agonists as well as in their physiological responses to sensory stimuli.

The glands were perfused retrogradely with oxygenated Lockes solution at 37° C through the adrenolumbar vein at a flow of 1 ml/minute. The effluent was collected for 1 min periods and the catecholamines estimated by the trihydroxyindole method (Vendsalü, 1960).

The glands were stimulated by changing the perfusate to one containing either m hydroxyphenylpropyl trimethyl ammonium iodide (10^{-8}-10^{-5} M) (Barlow & Franks, 1971) or acetyl β methyl choline (10^{-8}-10^{-6} M) as nicotinic and muscarinic agonists respectively.

In 14 tests on nine glands to which acetyl β methyl choline was given, the resting output of catecholamines was 390 ± 80 ng/min of which 22 ± 1% was noradrenaline. During stimulation the output rose by 1.4 to 8.6 fold and the increment contained 27 ± 3% noradrenaline.

In 17 tests on nine glands to which m hydroxyphenyl propyl trimethyl-ammonium iodide was given the resting output of catecholamines was 350 ± 80 ng/min of which 21 ± 2% was noradrenaline. During stimulation the output rose by 1.2 to 26 fold and the increment contained 24 ± 2% noradrenaline.

However, with cat glands we find that acetyl β methyl choline preferentially releases adrenaline while the increments with our nicotinic agonist contains a greater proportion of noradrenaline.

We conclude that in the dog, as in the cat, both nicotinic and muscarinic agonists release catecholamines from the adrenal gland. In contrast to the situation in the cat, the ratio of noradrenaline to adrenaline released in the dog changes little over a wide range of intensity of stimulation.

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