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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

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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  $(10^{-8} - 10^{-5} \text{ M})$  (Barlow & Franks, 1971) or acetyl  $\beta$  methyl choline  $(10^{-8} - 10^{-6} \text{ M})$  as nicotinic and

muscarinic agonists respectively.

In 14 tests on nine glands to which acetyl  $\beta$  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  $350\pm80$  ng/min of which  $21\pm2\%$  was noradrenaline. During stimulation the output rose by 1.2 to 26 fold and the increment contained  $24\pm2\%$  noradrenaline.

However, with cat glands we find that acetyl  $\beta$  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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