Corticotrophin production by rat adenohypophysial segments *in vitro*

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A method for the direct assessment of the adrenocorticotrophic activity of pituitary segments has been developed using the cytochemical bioassay for corticotrophin (ACTH) (Chayen, Loveridge & Daly, 1972; Alaghband-Zadeh, Daly, Bitensky & Chayen, 1974). The responsiveness of the rat adenohypophysis *in vitro* to some substances known to affect adrenocorticotrophic activity has been studied. Anterior pituitary glands were rapidly removed from freshly killed male albino Sprague-Dawley rats, carefully bisected and incubated for 2.5 h at 37°C in an artificial medium (Bradbury, Burden, Hillhouse & Jones, 1974) gassed with 95% O_2/5% CO_2. The medium was then replaced with similar medium containing the test substance. Fifteen minutes later the ACTH contents of the medium and the adenohypophysial tissue were determined separately. Lysine vasopressin, arginine vasopressin and acid hypothalamic extracts (Hiroshige, Kunita, Yoshimura & Itoh, 1968) resulted in marked, dose-related increases in both pituitary ACTH release and content. Cortisol (10-500 ng/ml) added to the final incubation medium inhibited the release but not the synthesis of ACTH induced by these substances. The method is potentially useful both for the identification and the assay of corticotrophin releasing hormone (CRH).

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The effects of glucagon on the hepatic arterial vasculature of the dog: an inhibition of the effects of vasoconstrictor agents

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Glucagon is released from the pancreas and small intestine, and its effects on the hepatic vasculature may well be of functional significance. In these experiments, the action of glucagon, and its effects on responses of the hepatic arterial vasculature to noradrenaline, angiotensin and vasopressin have been examined.

Seven female dogs (10-16 kg) were anaesthetized with chloralose (Kuhlman, Paris; 50 mg kg^{-1}) and urethane (BDH; 500 mg kg^{-1}) i.v., after induction with methohexitone sodium ('Brietal', Lilly; 7.5-10.0 mg kg^{-1}, i.v.). The hepatic artery, dissected free from its periarterial sympathetic nerves which were then divided, was cannulated close to its origin from the aorta, and perfused with blood from a cannulated femoral artery. The hepatic arterial perfusion pressure was measured close to the cannula in the hepatic artery, and blood flow in this system measured with a cannulated flow probe and electromagnetic flowmeter.

The initial control values (means ± s.e. means) were hepatic arterial mean perfusion pressure (HAPP), 119.2±5.9 mmHg and hepatic arterial blood flow (HABF) 38.8 ± 5.3 ml min^{-1} 100 g^{-1}. The calculated hepatic arterial vascular resistance (HAVR) was 3.31 ± 0.38 mmHg ml^{-1} min 100 g. *Post-mortem*, the livers weighed 324.8 ± 69.6 (mean ± s.d.) grams.

In 5 experiments, noradrenaline acid tartrate ('Levophed', Winthrop; 10 μg base, i.a.) caused a significant ($P < 0.001$; paired t-test) increase in hepatic arterial vascular resistance (HAVR) of 129.8 ± 19.7% ($n = 11$ injections). Angiotensin amide ('Hypertensin', Ciba; 0.5 μg salt, i.a.) caused a significant ($P < 0.001$) rise in HAVR of 161.1 ± 25.5% ($n = 12$), and vasopressin ('Pitressin', Parke-Davis; 0.1 unit, i.a.) caused a significant rise ($P < 0.01$) in HAVR of 98.7 ± 22.4% ($n = 9$).