Section of Endocrinology

President R I S Bayliss FRCP

Meeting February 22–23 1967
with the Society for Endocrinology

Symposium on the Investigation of Hypothalamic-pituitary-adrenal Function [*Abstracts*][1]

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Hypothalamic and Extrahypothalamic Mechanisms Controlling Adrenocorticotrophin Secretion

Studies have been performed which show that the implantation, using a stereotaxic technique, of synthetic corticosteroids at the level of the hypothalamus or of the reticular formation of the midbrain suppresses the functional activity of the pituitary-adrenal axis in the rat. The introduction of solid pellets of ACTH into the median eminence of the hypothalamus has a similar effect as evidenced by a diminution of plasma corticosterone levels. These findings suggest that corticosteroids and an excess of ACTH can both activate the receptors of a 'negative' feedback mechanism which helps control the activity of the pituitary-adrenal axis.

A method has been developed which permits the simultaneous evaluation of five neurohormonal releasing factors in crude hypothalamic extracts, namely FSH-RF, LH-RF, TSH-RF, GH-RF and CRF. This is based on the depletion of the corresponding pituitary hormones which follows the intracarotid injection of these extracts into normal recipient rats. Investigations have been carried out which suggest that distinct hypothalamic nuclei are responsible for the synthesis of the various releasing factors, which are then stored in the stalk median eminence.

[1]The papers will be published in full as a Memoir of the Society for Endocrinology

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Hypothalamic Control of Concomitant Secretion of ACTH and TSH

Several observations have been made in the past which suggest an inverse relationship in the pituitary ability to secrete acutely ACTH and TSH during exposure to stress (Brown-Grant *et al*, 1965, *J. Physiol.*, **126**, 41; Kraicer *et al.*, 1963, *Fed. Proc.* **22**, 507). None of these publications, however, could help explain whether the stress-induced shift in pituitary secretion (from TSH to ACTH) is ordered at the hypothalamus level (CRF being preferentially released rather than TRF following exposure to stress) or if it takes place at the pituitary site, as if the adenohypophyseal cells could not secrete both ACTH and TSH simultaneously at an increased rate in an acute situation. Experiments were undertaken to study the secretion of TSH in animals injected with TRF under exposure to stress or in which the ACTH responses were inhibited by pretreatment with dexamethasone and Nembutal. The results show that when the pituitary is induced to secrete TSH by TRF, it concomitantly secretes less ACTH in response to stress; conversely when the secretion of ACTH is inhibited as by pretreatment with dexamethasone and Nembutal, the pituitary secretes higher quantities of TSH in response to TRF.

In another series of experiments it has been observed that chronic exposure to stress or even mild exteroceptive stimuli inhibits or reverses the fall of plasma TSH concentration that normally follows acute exposure to stress; in these animals acute stress stimulates concomitant secretion of ACTH and TSH. Various mechanisms will be proposed to explain these observations.