8796 P

Effects of Anatomical Separation of Hypophysis from Hypothalamus in the Dog.*

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With a view of elucidating the mechanism concerned in eliciting enhanced food intake following removal of the posterior lobe of the hypophysis and hypothalamic lesions, the whole of the hypophysis was separated from the nerve and blood supply coming to it from the hypothalamus by cutting the stalk with scissors at its junction with the hypothalamus. This was successfully accomplished in 5 dogs, as evidenced by direct observation and by serial section of the tissue, without injury to the pars anterior, pars intermedia, or hypothalamus, and with variable, though slight, involvement of the pars tuberalis. In all animals there occurred a central atrophy of the infundibular process and increased cellularity in its remaining portion. The duration of the experiments ranged from three to five weeks.

A slight increase in food and water consumption occurred temporarily and showed a tendency to persist in instances, while in others there remained no deviation from the normal. There was no significant alteration in blood sugar, rectal temperature or in the general behavior of the animals either directly following operation or subsequently. These results demonstrate that in the dog, as in the monkey,[1] the blood supply from the posterior lobe artery is adequate for the gland as a whole; and also that denervation (hypothalamic) of the pars nervosa (which includes the infundibulum) does not precipitate enhanced food consumption or diabetes insipidus.

In order to harmonize these results in the dog with the interpretations of the Richter and Ranson schools relative to the cause of diabetes insipidus, one would have to assume either (1) that the ventral portion of the hypothalamus serves as a source of the antidiuretic principle as well as the pars nervosa, or (2) that our procedure in some way paralyzes a possible hypophyseal diuretic mechanism. It seems certain that the latter is not the case since

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1 Mahoney, W., and Sheehan, D., *Brain*, 1936, **59**, 61.

marked transitory diabetes insipidus (700 cc. of water per kg. body weight) occurred in four dogs in which following separation of the gland its proximal surface was cauterized. In a few days, the water intake returned to normal or only a trace of an increased consumption persisted. In one instance following cautery, typical diabetes insipidus appeared subsequent to the 10th day; thus suggesting that the essential mechanism responsible for diabetes insipidus both of the temporary and of the permanent type is inherent in the hypophysis. That the ventral portion of the hypothalamus is not involved as outlined above is indicated not only by numerous protocols from our earlier cat and dog series but also by further experiments for verification now near completion.

That the hypophysis is essential for the diabetes insipidus which follows hypothalamic lesions is also evidenced because hypophysectomy, on the 49th day after the hypothalamic lesion, eliminated abruptly a persistent marked diabetes insipidus (from 700 cc. water per kg. of body weight to normal—100 cc.±). This result might of course be due to an indirect effect on metabolism rather than to the removal of the specific mechanism causing the disturbance.

8797 C

Effect of Viosterol on the Parathyroids and Kidneys of Rats.*

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Alterations unlike those hitherto reported as following the administration of viosterol have been recently described in monkeys *(Macacus rhesus)*.[1] The parathyroids of some remained about the same, but those of others showed a marked decrease in basophilic cytoplasmic material, and the possibility was mentioned that this material might constitute a parathyroid secretion antecedent. In the kidneys, the incidence of intranuclear inclusions increased from 18.7 to 75% of individual monkeys. This was interpreted as due, either to the activation of a virus already present in very small amounts, or to the direct effect of the treatment. It was not possible to correlate the degree of loss of parathyroid basophilic ma-

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1 Cowdry, E. V., and Scott, G. H., *Arch. Path.* (in press).