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215. Portal Vessels Occlusion: A Cause for Pituitary Insufficiency in Patients with Pituitary Tumors?*

C. R. Pickardt, F. Erhardt, R. Fahlbusch, P. C. Scriba, II. Medizinische Klinik und Neurochirurgische Klinik der Universität, München, F.R.G.

Unexpectedly, only one of 13 patients with pituitary tumors and secondary hypothyroidism preoperatively failed to show an increase in TSH secretion after TRH stimulation, while the remaining 12 had TSH increments in the normal range or above normal. Therefore the question [1] was asked, whether compression of the portal vessels of the pituitary stalk and/or other suprapituitary derangements caused the observed hypothyroidism?

In all, 84 patients with hypothalamic disease and anterior pituitary tumors were so far investigated by means of the i.v. TRH stimulation test (200 μg). As expected, the TSH secretion in 9 hypothyroid patients with suprasellar disease showed a normal rise in 6 and a higher than normal response in 3 cases. This response was absent, again as expected, in 12 hypothyroid patients after operation for their anterior pituitary tumors and in 5 patients after treatment for tumors with both intra- and suprasellar extension. Surprisingly however, TSH secretion could be stimulated in 12 hypothyroid patients with anterior pituitary tumors preoperatively and in 8 of the 20 hypothyroid patients postoperatively.

From these results the following conclusions can be drawn: 1. An absence of TSH increment after TRH stimulation shows an insufficiency of thyrotropic anterior pituitary function. 2. In suprasellar disease secondary hypothyroidism is due to a lack of endogenous TRH. 3. In anterior pituitary tumors an increase of TSH secretion after TRH indicates a suprasellar cause for the secondary hypothyroidism.

One possible hypothetical explanation for the latter cases was that by compression of the pituitary stalk the tumors resulted in a disturbance of circulation in the portal system so preventing endogenous TRH from reaching the cells in the anterior pituitary. Exogenous TRH however could reach and stimulate the anterior lobe via the arterial system. The other explanation is, that there may be a functional impairement of the hypothalamus itself with an endogenous TRH lack. For the individual patient the true explanation will be decided only when the measurement of TRH levels in serum is available.

1. Pickardt, C. R., Geiger, W., Fahlbusch, R., Scriba, P. C.: Klin. Wschr. **50**, 42 (1972).

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