2.4 – 10 IU/24 h, group 3: 10 – 50 IU/24 h, group 4: >50 IU/24 h.

The results evidence a positive correlation between the functional status of the pituitary as indicated by the urinary gonadotrophin levels and the response to exogenous LH-RH as followed by plasma-levels of FSH and LH. Most of the patients with undetectable low urinary gonadotrophin levels failed to respond to LH-RH. The majority of patients with urinary gonadotrophin levels in the normal range, reacted promptly to exogenous LH-RH. Patients with primary ovarian failure and elevated gonadotrophin levels exhibit the most impressive response to exogenous LH-RH.

Further clinical studies were done to assess the therapeutic value of synthetic LH-RH in two aspects: 1. Stimulating of follicular maturation and 2. induction of ovulation in HMG stimulated ovaries. Exclusive administration of 100 to 200 μg LH-RH daily for 3 weeks did not result in any ovarian stimulation in amenorrhoic women. In a group of amenorrhoic patients in whom follicular maturation was achieved by HMG-stimulation LH-RH was administered when follicular activity was optimal as reflected by the estrogen plasma levels. Despite the fact that the plasma LH increased in 8 patients to ovulatory levels, ovulation presumably occured in only 4 patients. HMG administration normaly results in the maturation of a large number of follicles. Therefore it is obvious that those stimulated ovaries are rich in receptor sites for LH which eventually compete with those follicles which are ready for ovulation. In animal experiments it could be shown that it is not possible to release enough LH by LH-RH to saturate the receptor sites in HMG stimulated rat ovaries. This in contrast was possible with exogenous HCG. At the present we are studying the effect of LH-RH administration in patients with anovulatory cycles.

Diagnostic Use of TRH in Thyroid Disorders

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By measuring the TSH plasma levels before and 30 min after i.v. administration of synthetic TRH (short TRH test) and simultaneous estimation of thyroid hormone concentrations it is possible to get good informations about disturbances of the hypothalamo-pituitary-thyroid relationship. The TSH increment after TRH is abolished if a certain individually varying hormone concentration is exceeded or if the thyrotrophic pituitary cells are completely destroyed. Thereby the pituitary reacts rather sensitive in slight changes of hormone concentrations which might even not be detectable in blood ("subclinical" hyperthyroidism, "euthyroid" ophthalmopathy or "compensated" autonomous adenoma). The TRH test seems not to be suited for the evaluation of pituitary TSH reserve (*e.g.* in pituitary tumors), since there is quite often a normal TSH response in patients with advanced pituitary destruction and low serum levels of thyroid hormones. Furthermore the TSH peak levels after TRH are delayed in some of these cases. Thus a differentiation of secondary hypothyroidism due to hypothalamic or pituitary disorders is rather difficult by the TRH test. On the other hand, even in very slight decrease of thyroid hormones ("subclinical" primary hypothyroidism) an exaggerated TSH response to TRH can be found. In cases of overt primary hypothyroidism the initial TSH value before TRH administration is elevated. Under an antithyroid drug therapy of thyrotoxicosis a lag of weeks or even months can be observed between normalisation of serum hormone values together with clinical remission of symptoms and the normalisation of the TRH induced TSH release. The cause of this discrepancy has not yet been fully clarified.

In conclusion, the TRH test is a very useful tool for the diagnosis of overt or subclinical thyroid dysfunctions. More information can be gained than by the radioiodine uptake test, and the TRH test is much easier to perform and does not lead to greater discomfort for the patients. The test is of limited value, however, for the detection and differentiation of secondary hypothyroidism or for the control of the therapeutic effect during antithyroid drug therapy.

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