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Duplicate determinations of urinary iodine on two consecutive days were carried out on 134 patients from the Munich area. A mean value (± SD) of 36.9 ± 23.7 mcg iodine in a 24 hour urine collection was found.

In patients with non-toxic goiters a disproportionate increase in the secretion of T3 over T4 was found. This may explain the discrepancy between the clinical impression, that these patients were euthyroid, and the fact that their thyroxine levels were significantly lower than those of normal controls. From the controls (N = 23) we found the following values: T4 = 7.65 ± 1.07 mcg/100 ml, T3 = 146 ± 20 ng/100 ml and T3/T4 ratio = 19.6 ± 4.1 (ng/mcg). From the patients with non-toxic goiter (N = 43) the total T4 values were significantly lower than normal (6.19 ± 1.42 mcg per 100 ml), while the total T3 values (171 ± 41 ng/100 ml) and the T3/T4 ratios (28.8 ± 8.9 ng/mcg) were significantly (P < 0.005) higher (Horn K. et al., Z. klin. Chem. 10 (1972) 99).

Patients with non-toxic goiter (N = 49) were administered 200 mcg synthetic TRH i.v. In the larger group (N = 38) we have found, that the individual TSH response 30 min after TRH injection were within the normal range (2.73–23.6 mcU/ml, $\bar{x}$ log = 8.02 mcU/ml). Mean basal TSH levels (3.01 mcU/ml) were not higher than normal ($\bar{x}$ log = 2.81 mcU/ml), whereas the mean TSH response at 30 min (10.40 mcU/ml) was just significantly higher (P < 0.025) than normal. – The remaining 11 goiter patients had elevated individual TSH responses. The mean basal TSH level ($\bar{x}$ log = 7.75) and the mean TSH response 30 min after TRH ($\bar{x}$ log = 32.4 mcU/ml) were significantly greater (P < 0.0005) than normal. – The measured levels of T4-iodine (CPB-analysis, Horn K. et al., Z. anal. Chem. 259, 222 (1972)) in both groups of patients with nontoxic goiter did not allow the differentiation between the two groups, but were both (3.44 and 2.94 mcg/100 ml below the normal value ($\bar{x}$ log = 4.11 mcg/100 ml, P < 0.005). – In the group of goiter patients with elevated TSH responses
we assume, that the compensatory increased secretion of T3 does not render these patients euthyroid (preclinical hypothyroidism).

The dosage of thyroid hormone therapy for non-toxic goiter patients may effectively be controlled by observation of the TSH suppression before and after TRH stimulation (Pickardt C. R. et al., Klin. Wschr. 50 (1972) in print).

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