(mean±S.D.) ng/ml in control subjects, 5.09 ±2.40ng/ml in patients with hyperthyroidism and 0.52±0.28ng/ml in patients with hypothyroidism. The value was 1.19±0.37ng/ml in patients with chronic thyroiditis and 2.05 ±0.70ng/ml in those with subacute thyroiditis. Patients with simple goiter and nodular goiter had normal T_3 concentration.

The discrepancies between T_3 levels and values of T_3-RSU, T_4 and T_7 were noted in patients with hyperthyroidism and hypothyroidism under treatment. Some of patients with hyperthyroidism receiving antithyroid drugs and those with hypothyroidism taking dessicated thyroids had high levels of T_3, whereas values of T_3-RSU, T_4 and T_7 were in normal range. Other cases of hyperthyroidism under therapy had normal T_3 concentration with low values of T_3-RSU, T_4 and T_7. The discrepancy was also noticed in patients with anorexia nervosa, having significantly lowered levels of T_3 and normal values of T_3-RSU, T_4 and T_7.

Radioimmunoassay for Measurement of Triiodothyronine in Human Serum and Urine

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A radioimmunoassay (RIA) system for measurement of triiodothyronine (T_3) in human serum and urine has been developed. A specific antibody to T_3 was prepared in rabbits by immunization with a conjugate of T_3 with human serum albumin. 8-anilino-1-naphthalene sulfonic acid was used for TBG inhibitor. Normal human serum (or urine) was treated with dextran-coated charcoal and added to standard as T_3 free serum (or urine). Bound form was separated from free form by means of double antibody method. Cross-reactivity with T_4 was less than 0.01% in the T_3 RIA system. The recovery of added T_3 to serum (or urine) was 96—108%. Dilution of serum (or urine) resulted in parallel curves to that obtained for the standard T_3. The minimal detectable amount of T_3 was 12.5ng/dl when 50μl of serum was assayed. Coefficient variation for serum T_3 determination was 4.9—6.0% (within-assay) and 6.7—8.8% (between-assay) respectively. Serum concentrations of T_4 and T_3 were determined in various disorders which were divided in 6 groups ie. [I] normal T_4 and T_3 [II] increased T_4 and T_3 [III] decreased $T_4$and T_3 [IV] normal T_4 and increased T_3 [V] decreased T_4 and normal T_3 and [VI] normal T_4 decreased T_3. Untreated patients with Graves' disease showed I, II, IV, treated patients I, II, III, IV, V, hyperfunctioning nodular goiter I, II, IV, hypothalamic-pituitary tumors I, III, V, TBG deficiency III, pregnancy I, II, hydantidiform mole or chorioncarcinoma I, II, IV, and anorexia nervosa VI. There was a good correlation between serum concentration and urinary excretion of T_3 in normal subjects and patients with hyper and hypo-thyroidism. In nephrotic syndrome, however, serum T_3 level was low but urinary T_3 was normal or increased. The absolute values of T_3 concentrations in urine (or even in serum) were not

identical when different batches of anti T_3 serum were used. Heterogeneity of the anti T_3 serum which reacts with free T_3, conjugated T_3 or thyroid hormone metabolites in urine (or serum) may cause the different results of T_3 RIA.

Studies on ^{131}I-Thyroxine Binding Protein Using Single Radial Immunodiffusion and Ouchterlony Method

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In 1967, I reported the first familial throxine-binding globulin (TBG) deficiency in Japan. The evidence of TBG deficiency is based on the defect of the distribution of radioactivity due to ^{131}I-thyroxine in inter-a globulin region by electrophoresis of the serum and ^{131}I-thyroxine mixture. Recently, I have an opportunity for studying with anti-TBG rabbit serum (Behringwerke) for purpose to clarify the nature of TBG protein fraction in TBG deficient serum more exactly. Two different immunological methods are used, i.e. single radial immunodiffusion in antigen-contained gel layer and Ouchterlony method combined with radioautography. In the former procedure, the sample sera (0.1 or 0.2ml) are mixed with ^{131}I-thyroxine (20μCi, 55 μg/dl of serum) and thereafter 7.0ml of 1.2% agarose solution are poured into the mixture. After making the agar plate, 12 or 27 wells for anti-sera application are punched out with cutter.

As the result of the former method, the radioautogram showed the distribution of radioactivity around some wells into which are applied anti-sere to TBG, prealbumin, β-lipoprotein and hemopexin, corresponding to the protein precipitation rings respectively. I emphasize the probability of hemopexin to be one fraction of the thyroxine-binding proteins besides previously identified.

From the second part of these studies, I draw the conclusion that the TBG protein itself is defect in the sera of the TBG deficient patients instead of the disturbance of binding activity of TBG to ^{131}I-thyroxine, because there is the precipitation line between normal serum and anti-TBG serum, while the line between TBG deficient sera and anti-TBG serum can not be detectable.

Activities of Thyroid Stimulator in the Fractions from the Serum of Graves' Disease Eluted Through the Acid Sephadex Column

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In the present paper, activities of thyroid stimulator in the fractions from the serum of