CLINICAL EVALUATION OF ANTITHYROIDLOBULIN ANTIBODIES BY IMMUNORADIOASSAY.
Shinji Morita, Kanji Kuma, Hajime Tamai*, Noriyuki Osako*, Osamu Fukino*, Shigenobu Nagataki** Kuma Hospital, Kobe. *Department of psychosomatic Medicine, Faculty Medicine, Kyushu University, Fukuoka. *First Department of Medicine, Faculty of Medicine, University of Nagasaki, Nagasaki. Japan

Serum antithyroglobulin antibodies were measured with immunoradiometric assay(IRMA) to investigate correlation between thyroid and microsome tests and IRMA using 116 patients with Graves' disease. (1) Anti-Tg antibodies was detected by IRMA in only one of 48 patients who showed negative reactions to thyroid and microsome tests (TG, MG). (2) None of 34 patients who were TSH had anti-Tg antibodies by IRMA. (3) Anti-Tg antibodies was detected by IRMA in one out of 4 patients whose thyroid and microsome tests were positive (T=200X, MG200X). (4) Anti-Tg antibodies was detected in all 30 patients whose thyroid and microsome tests were more than 400 folds.

Conclusion: Immunoradiometric assay had no correlation with microsome test and showed high specificity to antithyroglobulin antibodies.

CLINICAL STUDIES ON REGULATION OF TSH SECRETION—Alterations after single oral dose of bromocriptine, triiodothyronine, and prednisolone. T. Mori, T. Ishihara, S. Bito, and K. Ikekubo Department of Intern. Med., and Nuclear Med., Kobe Central Municipal Hospital, Kobe

Effects of single oral 2.5 mg dose of bromocriptine on serum TSH levels of 11 patients with hyperthyroid state and normal pituitary reserve were studied. For comparisons, effects of triiodothyronine(T3) and prednisolone(PD) were also studied. Blood specimens obtained before and every one hour till 6 hours after drug administration were assayed for TSH(modified sensitive RIA),T3U, T4, T3, and free T4(Gamma Coat). Serum TSH levels(1.6±0.67 uIU/ml at 0 Hr) were progressively and significantly decreased after bromocriptine(1 Hr:81.2±0.25, 3 Hrs:60.7±0.9, 7 Hrs:50.1±18.0, respectively). T3 also showed slight but not significant decrease after bromocriptine. T4, free T4, and T3U did not show significant alterations. After 25 ug oral T3 TSH revealed progressive decrease to 111.6±5.3% at 6 Hrs. After 15 to 25 mg oral PD apparent TSH decrease compatible with 2.5 mg bromocriptine was observed (57.5±6.4% at 3 Hrs). TRH tests were performed in 2 patients under chronic bromocriptine treatment(7.5 and 5.0 mg daily). In contrast to low normal basal TSH(0.51, 0.98), excessive TSH responses (peak: 27.0, 18.8) were observed after TRH. In conclusion, bromocriptine was found to decrease TSH levels of thyroid subjects significantly, however, chronic treatment by bromocriptine were considered not so effective to reduce TSH reserve.

CLINICAL APPLICATION OF RADIORECEPTOR ASSAY FOR TSH: CHANGES OF TSH—BINDING INHIBITOR IMMUNOGLOBULINS AND PROGNOSIS OF GRAVES' DISEASE AFTER ANTITHYROID DRUG TREATMENT. Y. Iida, T. Nisaki, K. Kasagi, K. Endo, J. Konishi; K. Tokuzume. Department of Nuclear Medicine, Kyoto University School of Medicine, Kyoto

The changes in the activities of TSH-binding inhibitor immunoglobulins (TBI) and human thyroid stimulator (HTS) were studied during anti-thyroid drug therapy of Graves' disease. TBI were measured by the radioreceptor assay for TSH using 1 mg IgG and HTS was measured by the increase in cyclic AMP in the monolayer culture of human thyroid adenoma cells using 3 mg IgG.

Before treatment, TBI were positive in 8 out of 10 patients, while 9 of 10 were positive in HTS. There were little changes in TBI and HTS activities during initial 2-3 months after anti-thyroid drug treatment. The activity of TBI and HTS decreased during 4-5 months and reached normal level on 5-15 months in 3 and 2 out of 6 patients, respectively. TBI and HTS were positive in 6 and 7 out of 33 patients respectively at the time of T3 suppression test. All of the 4 patients who were positive in either TBI or HTS at the cessation of therapy relapsed.

It is concluded that the measurement of TBI and HTS is valuable as a prognostic indicator of Graves' disease.