^{3}H-PRU or ^{3}H-PRSU of this solution is measured and then calibrated in amount of corticoids in 2 ml supernatant (μg) using the standard curve of cortisol. Recovery of corticoids in 2 ml supernatant is determined by adding ^{14}C-cortisol to the plasma.

This method is based on the same principle as the method to measure blood thyroxine reported by Nakajima et al (1966).

Results: (1) Specificities, reproducibilities and sensitivities of these methods are examined with satisfactory results. (2) No significant effect with cortisone, 11-desoxycortisol, THF, THE, dexamethasone, paramethasone and triamcinolone on the ^{3}H-PRU is observed except with cortisol, hydrocortisone acetate and prednisolone. (3) Significant increase after the ACTH administration and circadian change in ^{3}H-PRU are observed. (4) Good correlation between the plasma corticoids determined by our direct method and the plasma 11-OHCS level determined by DeMoor's method is observed.

Simultaneous determination of ^{3}H-PRSU and ^{131}I-T_3RSU by our indirect method is studied with satisfactory results. No interferance between ^{3}H-PRSU and ^{131}I-T_3RSU is observed. Both blood corticoids and thyroxine level are simultaneously determined the same method as our direct method.

The advantage of these methods over the conventional method is found in their simplicity and their requirement of only a small specimen, and their capability of simultaneous determination of ^{3}H-PRSU and ^{131}I-T_3 RSU.

Clinical Studies on Cortisol Secretion and its Metabolism Using Radioactive Cortisol

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Cortisol-4-C^{14} was administered intravenously to six normal subjects, fifteen patients with thyroid dysfunction and to nine patients with liver disease. Twenty-four urine sample after the injection was collected. Cortisol secretion rate was estimated by isotope dilution method and daily excretion of major urinary metabolites of cortisol, i.e. cortols, cortolones, tetrahydrocotisol (THF), allotetrahydrocortisol (ATHF) and tetrahydrocortisone (THE) were calculated by multiplying the secretion rate by per cent dose injected of each radioactive metabolite on paper chromatogram.

In ten patients with hyperthroidism, cortisol secretion rate and urinary excretion of total glucuronide, THE, ATHF and of cortolones were remarkably increased (26.4 ± 5.7** mg/day, 17.8 ± 1.2*mg/day, 9.2 ± 2.2*mg/day, 1.67 ± 0.53***mg/day, and 5.40 ± 1.69*mg/day, respectively) as compared with those in normal subjects (16.8 ± 1.5 mg/day, 7.0 ± 1.2 mg/day, 3.2 ± 0.4 mg/day, 0.93 ± 0.33 mg/day, and 1.30 ± 0.43 mg/day, respectively). And THE/THF ratio, ATHF/THF ratio and cortolones/cortols ratio were also increased (9.8 ± 4.0*, 1.6 ± 0.3*, and 16.3 ± 6.5**, and 5.3 ± 1.3, respectively), suggesting acceleration of both 11 β-dehydrogenation and 5 α-hydrogenation in cortisol metabolism. These changes were statistically significant (* : $p<0.001$, ** : $p<0.01$, *** : $p<0.05$).

In five patients with hypothyroidism, the reverse was observed (secretion rate 8.3 ± 2.3* mg/day, total glucuronide 3.9 ± 1.6***mg/day, THE 1.12 ± 0.46*mg/day, ATHF 0.05 ± 0.02** mg/day, THE/THF 0.81 ± 0.04*, ATHF/THF 0.05 ± 0.07*, cortolones/cortols 2.28 ± 0.43**).

In all patients with liver disease (liver cirrhosis 3, chronic hepatitis 5, acute hepatitis 1), cortisol secretion rate was decreased (11.7 ± 4.1***mg/day, 10.8 ± 1.3**mg/day, and 8.6 mg/day, respectively). And was observed decreased tetrahydro (THF+ATHF+THE)/hexahydro (cortols+cortolones) ratio (1.74 ± 1.0*, 2.10 ± 0.21*, and 0.39, respectively) as compared with normal value (3.85 ± 0.94), suggesting that hexahydro metabolites of cortisol might be produced by both liver and extrahepatic tissues, whereas tetrahydro metabolites were mainly produced by liver.