subacute hepatitis which showed an extensive necrosis, deformation of lobules as well as swelling of cells in the histological pictures, only a slight decrease in the albumin metabolism. Looking at the changes in SA concentration and albumin metabolism, another case of subacute hepatitis which has recurrence three times during the course of 4 years and transformed to liver cirrhosis, finally falling into coma, it showed a fall in SA at the first and the third stage of recurrence and the decrease in ATOV at the third recurrence. On the remission by treatment, the albumin metabolism also recovered to normal level. Although the metabolism recovered to almost normal at convalescence in the third recurrent stage, on the administration of 75 mg of 6-mercaptopurine (6MP) there was a slight fall in ATOV.

Along with the fall in the ability of synthesis as shown by the fall in ATOV, it is reasonable to assume that there will also be relative gastro-intestinal losing of proteins. In order to clarify this latter problem, we studied whether or not such a protein losing takes place, by Waldman's method, using radiochromated human serum albumin (51Cr-albumin, Squib). The fecal excretion rate (FE) in 5 controls was 0.1–0.5% of the amount of SA administered and in some who showed tarry feces and positive occult blood reaction (+++) it was over 1%. However, the average of 26 cases of liver cirrhosis was 0.54 ± 0.20%, revealing hardly any significant difference from controls. There was correlation coefficient of – 0.35 between SA concentration and FE, and even in the cases showing the fall in SA concentration, FE was not less.

Next, after the intravenous injection of both 131I-albumin and 51Cr-albumin we computed the albumin metabolism and FE of 51Cr-albumin simultaneously, and the ratios of FE to TEA represented as RFE, were compared among liver diseases. As a result, in contrast to 1.24 ± 0.34 (%/g) of RFE in 6 controls, the cases of liver cirrhosis showed a significantly high value of p<0.05. Especially in 5 cases of severe liver cirrhosis which were accompanied with ascites, varix and jaundice a distinct rise (p<0.01) in RFE was observed, and in one case showing tarry feces RFE was as high as 13.5. However, there was no significant difference between mild cases of liver cirrhosis and controls. From these results it may be assumed that in severe cases of liver cirrhosis along with the decline in the albumin synthesis there arises factor of relative gastro-intestinal protein losing, and these combined factors elicit the decrease of SA and TEA.

Total Exchangeable Sodium in Relation to Extracellular Water

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Total exchangeable sodium in hypertension and various diseases related to sodium disturbance was evaluated in relation to extracellular water (ECW) and to total body water (TBW).

Materials and methods: (1) Sterile solution for intravenous injection was the mixture of the following: 3H-water 2 mCi, 22Na 20 μCi, and 51Cr-E.D.T.A. (ethylenediaminetetraacetate) 200 μCi. (2) Determination of total exchangeable sodium (TENa), ECW and TBW was carried out by isotope dilution methods. Lean body mass (LBM) was calculated form TBW. Measurement of ECW with 51Cr-E.D.T.A. was presented previously elsewhere by us. (3) 3H was assayed with liquid scintillator by the method of Weber, H., et al. Gamma rays from 51Cr and 22Na were separated by gamma ray pulse height analyzer and counted at each photopeak. (4) The observations were performed in 4 healthy subjects and 12 patients with various disease including
hypertension 7 (3 milder than K-W II of eye-ground, 4 severer than K-W III), nephrotic syndrome 2, uremia 1, Conn’s syndrome 1, and diabetes insipidus 1.

Results: (1) The mean values of body constituents in healthy subjects were TBW 62.6% per kg of body weight, ECW 26.5% per kg, and TENa 43.5 mEq per kg. (2) The ratio of ECW to TBW was 42% for healthy group. The ratio increased in nephrotic syndrome and/or uremia, which presented a striking contrast to evident decrease in diabetes insipidus (26%). (3) Plasma sodium concentration was seen almost on equal level in the materials. In respect of TENa expressed by per kg of body weight, there was little distinction between the healths and the diseases except nephrotic syndrome. (4) On the contrary, TENa expressed by per litter of ECW showed an evident distinction between the healths and the diseases: the mean value of TENa was 161 mEq/L in normal subjects, 165 mEq/L in hypertension (< K-W II), 200 mEq/L in hypertension (> K-W III), 222 mEq/L in Conn’s syndrome and 169 mEq/L in nephrotic syndrome.

Conclusions: From the observations of extracellular compositions of body fluids in hypertension and various diseases related to sodium disturbance, it seems appropriate to express TENa as a per litter of ECW. TENa expressed in terms of ECW increased in hypertension and Conn’s syndrome. However, no significant differences were seen between the healths and the diseases, when TENa was expressed by a kg of body weight.

Turnover Studies of Sodium in Animals by a Double Tracer Technique with $^{22}$Na and $^{24}$Na

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In our recent experiments using $^{22}$Na and a whole body counting technique, sodium in human body was eliminated two exponential functions with half-lives of 8 days and longer than 100 days. The latter phase may be due to sodium citrate which is fixed in skeletal system.

For the interpretation of the longer lived component, ten rats of Wistar strain were divided into two groups after intraperitoneal administration of 10 μCi of $^{22}$Na. Each group of rats was then given drinking water containing 0.5% and 0.1% NaCl respectively. Rats of the 0.5% saline group were sacrificed at 18 days following the injection, while the rats of another group were kept for 95 days. One day prior to sacrifice, each rat was injected 5 μCi of $^{21}$Na intraperitoneally, to investigate the short term and long term sodium turnover in the same animal. Activity of sodium isotopes in various tissues was assayed by gamma spectrometry.

At 24 hours after the administration, sodium content of bone was found to be higher than the other tissue, while in the later stages of both groups, sodium content in bone was evidently higher than the other tissues or plasma by the factor of approximately 4 and 40 respectively. These results suggests that the presence of the fast and slow exchanging sodium pools in bone and extracellular sodium pool.

For the determination of the total body content of stable sodium in rat, the “in vivo” activation analysis were carried out, however, the values were found to be lower than the values from the activation analysis of composite ashed samples of whole rat. This difference is probably due to the nonuniform distribution of sodium and of thermal neutron in rat at the time of activation.