National Institute of Radiological Sciences have been used for the $^{40}$K and $^{137}$Cs measurement in more than 3,000 persons.

This report is presented as an approach to studying progressive muscular dystrophy. Similar work has been done by Blahd (V.A. Center at Los Angeles), followed by Kossman (Cornell Med. Center). Our effort were directed to studying the pathogenic process of Duchenne form muscular dystrophy. A large plastic scintillator type whole body counter was used because of its higher geometric efficiency and shorter counting time. Data analysis was performed on a Burroughs-5500 digital computer.

Body potassium concentration was determined in 51 patients with neuro-muscular diseases, including 42 Duchenne form, 2 limb-girdle form, 1 myotonic form, and 6 neurogenic atrophy, who were measured a total of 119 determinations, and in 43 unaffected family members. Normal volunteers comprised 150 healthy males and 82 healthy females were measured as controls of this study.

Patients with Duchenne form dystrophy have severe depressions of body potassium concentration, and its levels usually paralleled the severity of the disease and the length of the period attacked with the disease. A similar decrease in body potassium concentration was noted in female patients with limb-girdle form, but to a lesser degree. Potassium concentration in lean body mass determined by tritiated water dilution method was also reduced in 11 selected patients with Duchenne form. 17 patients with Duchenne form exhibited a decrease in total body potassium content and body potassium concentration with the course of one year. Long-term body retention of $^{86}$Rb measured with the whole body counter showed shorter biological half life of rubidium in patients with Duchenne form. These results obtained may suggest that potassium leakage from the muscle cell might be a primary factor in the pathogenic process of the disease.

Remarkably reduced body potassium concentration were observed in about half of 28 female relatives, while slightly reduced concentrations were observed in only four of 15 male parents. In view of carrier types, total of five probable carriers and 11 of 15 possible carriers had moderate depressions of body potassium concentration. From the relationship between serum creatine phosphokinase levels and body potassium concentration, it was shown that reduced body potassium could be seen in some female relatives who had normal serum creatine phosphokinase levels. These results obtained suggest that this whole body counting method might be valuable as an indication of the gene carrier.

Thus, this method may assume a role in diagnosing progressive muscular dystrophy, even in its preclinical stage, and in predicting the inheritance of the disease.

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Studies on Sodium Metabolism in Patients with Hypertension Using $^{22}$Na and Whole Body Counter

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The sodium metabolism in 7 normal subjects, 15 hypertensive patients and 5 patients with cachexic edema (varying in age 15 to 72) have been studied for 40 to 320 days after the administration of 10 $\mu$Ci of $^{22}$Na. Among 15 hypertensive patients, 6 were in the early stage and 5 in the late stage of essential hypertension, 2 had chronic nephritis and 2 were renovascular hypertension. All patients were hospitalized and received
a diet containing 10g of salt a day or a free diet (11g to 20g of salt a day). The whole body counting was performed every day for 2 weeks by a plastic scintillator counter in a steel room.

The $^{22}$Na activities of serum samples were counted by a low background $\beta$-spectro-meter. Chemical sodium concentration of serum and urine was measured by a flame photometer. The biological half of $^{22}$Na (Tb) and the total exchangeable sodium (Nae) were calculated from these data. The effects of the high salt loading or the administration of some diuretic drugs, such as hydrochlorothiazide, acetazolamide and anti-aldosteronic drug were investigated for 1 week from 7th day after $^{22}$Na administration. Thereafter, the patients were discharged from the hospital and put on a free diet. The body counting was done every 2 weeks for 3 months, and then every month up to 11 months.

Some of the results were previously presented at the 5th annual meeting of this association. The further important findings are as follows:

1. The patients with hypertension in the late stage had a slower turnover than normal or hypertensive patients in the early stage both low and variable salt intake.

2. Tb and Nae in normal subjects under free diet ranged 5.8 to 13.2 days with an average 10.1 days, 2250 to 2400 g with an average 2310 g, respectively. In these cases, Tb is shorter and Nae is lower than those data reported in western countries.

3. The effect of acetazolamide on the elimination of $^{22}$Na in hypertensive patients remained longer than that of hydrochlorothiazide.

4. Anti-aldosteronic drug took no effect in 3 patients with essential hypertension.

5. The long term body retention curve was devided into 2 exponential components. While the Tb values were quite different each other on the 1st phase, the leveling off points reached between the range 0.3-0.5% of administered dose in all of the patients followed up to 11 months, except for 1 case (15 years old, girl). This 2nd phase has been explained to be slowly exchanging compartment in bones by several authors.

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**Studies on Copper Metabolism in Wilson's Disease**

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Abnormality of copper metabolism has been regarded as one of the most basic defects in Wilson's disease as reported by many workers. Recently, the use of the radio-isotopic copper has turned out to be very helpful in the study of the disease and there have been many reports by its use.

We have studied radiochemically on clinical cases with Wilson's disease in Japan, comparing them with those reported in American and European countries.

Six cases of Wilson's disease were studied and the control group altogether consisted of eight cases. Each case was given 1-4 mCi of $^{64}$Cu as cupric chloride by mouth. The specific activity was approximately 0.4-0.5 mCi/ mg Cu.

1) The measurement of radioactivity in blood was done mainly by a well type scintillation counter along with a low background $\beta$-ray spectrometer, particularly the latter being used for the later period of diminished activity. The control group showed its peak of radioactivity in plasma in one to three hours after $^{64}$Cu administration and the lowest level in four to six hours. In Wilson's disease, the initial peak tended to be somewhat higher and slightly delayed than that of normal, and in one of the cases the peak is much delayed.