2) The second experiment we have done was to measure the uptake of $^{64}\text{Cu}$ in certain areas of the body by the collimated scintillation counter. The areas aimed were those of the liver, head, mid-thigh and calf. There were some cases with Wilson's disease in which the uptake in the head, calf or thigh was exceedingly higher than those in controls. However, the uptake in the liver of patients with Wilson's disease was lower than controls.

3) The major portion of $^{64}\text{Cu}$ was excreted into feces, but this varied considerably from subject to subject. In the cases of Wilson's disease, the excretion of $^{64}\text{Cu}$ into urine showed the increase than normal. However, $^{64}\text{Cu}$ excreted into feces did not differ significantly from those of controls.

3) We measured the remaining radioactivity in the body after $^{64}\text{Cu}$ administration with plastic whole body counter. Of the given dose after one week, 14.5-16.5% was remained in the normal subjects, whereas in Wilson's disease, although the data were somewhat fluctuated, the remaining $^{64}\text{Cu}$ was 12.3-17.1%.

5) Recent progression of neutron activation analysis enables us to measure the trace metals in hair and nails. We used methods to measure the copper in Wilson's disease. The copper in the hair of the Japanese was less than that reported in other countries. It varied considerably even in normal subjects. The copper in the hair of Wilson's disease was increased in some but normal in others. The copper in nails also showed a similar tendency.

Calcium-47 Kinetic Studies in Human Metabolic Bone Diseases

N. Yamauchi, S. Hayami and Y. Ishizuki

The First Department of Internal Medicine, Nagoya University School of Medicine, Nagoya

There are many reports of calcium kinetic studies using bone-seeking isotopes. But the results of the kinetic study was conflicting in several aspects and an accumulation of further data was required.

Calcium$^{47}$ kinetic studies were performed in one normal subject, one patient with hypothyroidism and two patients with hyperparathyroidism.

Calcium$^{47}$ was administered intravenously from a calibrated syringe, in a single dose of 40-50 $\mu$Ci, to each subject. Thereafter calcium$^{47}$ and stable calcium of serum, urine and stool were determined daily for 6 to 11 days. Calcium$^{47}$ was counted by well-typed scintillation counter and stable calcium was determined by Clark-Collip modification of the Kramer-Tisdall's method. The analysis of data was done by the method of Haeney and Whedon.

In a normal subject the size of miscible calcium pool, E, was 37 mg. Ca/kg., bone formation rate, BFR, 8.1 mg. Ca/kg./day, fractional rate of loss of isotope from miscible calcium pool, K, 0.234±0.50. In a patient with hypothyroidism E was 35 mg. Ca/kg., BFR 6.7 mg. Ca/kg./day, K 0.201±0.030. In one patient with hyperparathyroidism E, BFR and K were 32 mg. Ca/kg., 8.3 mg. Ca/kg./day and 0.306±0.006, respectively. In another patient with hyperparathyroidism these were 59 mg. Ca/kg., 7.6 mg. Ca/kg./day, 0.348±0.011, respectively.

E was increased in a patient with hyperparathyroidism and BFR was decreased in a patient with hypothyroidism. Furthermore, K seemed to be correlated to the abnormality of calcium turnover rate in metabolic bone diseases.