Therefore, we revealed on this report the availability of the DF ^{32}P not only for the determination of MRCLS, but also for its related subjects in ferrokinetics in view of aplastic anemia.

Materials: The normal healthy volanteers who were 29 males and 8 females were studied for the determination of MRCLS. And patients with aplastic anemia reported here were 12 males and 3 females in age between 4 and 60.

Results:

1. The average DF^{32}P-MRCLS in the normal volanteers was 101 ± 12 days (in males, it was 103 ± 13 days, and in females, 96 ± 12 days).
2. In all cases with aplastic anemia, DF^{32}P-MRCLS was shortened and the average was 57 ± 13 days.
3. Red cell iron renewal rate (RCIR, mg/day/kg) can be obtained from hemoglobin iron divided by MRCLS with DF^{32}P. And effective erythropoietic rate is obtained by dividing RCIR with PIT (R/P). R/P in the patients with aplastic anemia was all decreased and the average was 48%.
4. On whole-body radioiron (^{59}Fe) distribution study, ^{59}Fe retention in bone marrow after 10 days was observed in 8 cases out of 15. And there was a correlation between ^{59}Fe retension in bone marrow and reduction of DF^{32}P-MRCLS.

Clinical Study of Reticuloendothelial Phase in Ferrokinetics in Hemolytic Disorders by Analysing ^{51}Cr- and ^{59}Fe- Red Cell, Spleen and Liver Data Using Analog Simulation Technique

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A clinical study was performed to examine the reticuloendothelial (R.E.) phase of ferrokinetics by tracing and analysing the natural process of red cell destruction, iron release for reutilization in developing red cells or its deposit as a store. The subject of this study was those cases having excess hemolysis, in which early and random destruction of red cells took place, sufficient enlargement of the spleen existed for accurate external probe and R.E. phase in ferrokinetics played an important role.

With ^{51}Cr-labelled autogeneous red cells, their circulating mass and the rate of destruction were calculated assuming the elution rate of ^{51}Cr to be 1.5 percent per day. The sites of their destruction were probed also by calculating excess count according to Lewis and Szur's method and by expressing the value in a ratio to the count over the precordium at equilibrium. In ferrokinetics, red cell incorporation rate of ^{59}Fe was accurately calculated by determining a body/venous hematocrit ratio with ^{51}Cr-red cells and ^{125}I-albumin. On external probes, the radioactivity attributable to that of red cells contained in the subjective organs was subtracted principally according to Elmlinger's formula, which was modified applying distribution data of ^{51}Cr red cell in stead of that of ^{59}Fe transferrin.

For the data analysis, a simulation technique was applied using an analog computer in order to calculate the mean transit time of iron in erythropoietic marrow and the time in R.E.S. for its release and its deposition rate there. The computed values coincided well with actual measurement ones. Simultaneous coincidence in the red cell incorpolation, the bone marrow, spleen and liver values necessitated analysis solution to be almost primary with scarce freedom.

The value obtained by this computation on iron deposition rate was substatiated well by the histological findings on the degree of stainable iron deposited in the cells with correlation coefficient of 0.898 in the spleen and 0.715 in the liver.

In hereditary spherocytosis, there was also a relationship between the amount of iron derived daily from red cell destruction and the time for R.E.S. to release iron through catabolic process. In autoimmune hemolytic anemias, it took more time in the release process for the comparable

amount of iron in a hemolytic crisis than that both in those at remission and in hereditary spherocytosis.

Thus we could successfully carried out an analysis of the data concerning the process naturally occuring in R.E. phase of ferrokinetics without doing any additionaly loading examination such as that using denaturated cells or colloidal iron particles. The result elicited those informations descrived above which had been looked over or had not been appreciated enough.

Studies on Iron Absorption in the Patients with Banti's Syndrome

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Materials and Methods:

Iron absorption was tested by using whole-body counters, measuring the percentage retention 2 weeks after the oral radioiron dose (10 μCi) containing 4 mg carrier in the form of ferrous sulfate. The subjects studied were; 15 normals, 23 iron deficiency anemia, 8 Banti's syndrome and 14 liver cirrhosis.

Results:

1. Normals; The iron absorption of normal Japanese males was $28\pm17\%$, and that of females was $30\pm15\%$. These results showed no significant difference between males and females. And the average of males and females was $29\pm15\%$.
2. Iron deficiency anemia; The iron absorption in 17 patients out of 23 (83%) was higher than normal subjects and mean was $50\pm20\%$ ranging from 6% to 86%. This is 1.7 times value for Japanese normals. Serum iron level was between 16 and 89 μg/100 m*l* with an average value of 36 μg/100 m*l*.
3. Banti's syndrome; 8 patients with Banti's syndrome were studied in our laboratory. 2 out of 8 had slightly higher absorption than normals and mean was $31\pm16\%$ ranging from 6 to 59%. Serum iron was between 16 and 51 μg/100 m*l* and average was 33 μg/100 m*l*.
4. Liver cirrhosis; The average iron absorption of 14 patients was $26\pm14\%$ ranging from 5 to 51% and serum iron level was between 29 and 124 μg/100 m*l* with an average value of 117 μg/100 m*l*.

Summary:

Although in the cases of iron deficiency anemia and Banti's syndrome serum iron level was equally decreased, iron absorption in the patients with Banti's syndrome was not increased like in iron deficiency anemia. In liver cirrhosis, iron absorption and serum iron level were both almost normal.

On this study we could prove that decreased iron absorption was one of the factors for anemia in Banti's syndrome.

Platelet Survival in Liver Disease

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Platelet survival was measured according to Aster's acid-ACD method by collecting platelets from 200 m*l* of fresh blood. A relatively large amount of ^{51}Cr (300–500 μCi) was used to label platelets. In a healthy subject the study was carried out autologously. The half life (T1/2) was 4.7 days and the recovery 53.1%. The disappearance curve was linear.

Platelet survival study was done in 6 patients with liver cirrhosis whose spleens were 11–14 cm long scintigraphically. Their platelet counts ranged from 3.4 to $10.8\times10^4/mm^3$. According to the platelet survival curves, they separated into 2 groups. In 4 patients, the studies were performed