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 occurring in R.E. phase of ferrokinetics without doing any additional loading examination such as that using denaturated cells or colloidal iron particles. The result elicited those informations described 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±17%, and that of females was 30±15%. These results showed no significant difference between males and females. And the average of males and females was 29±15%.

2. Iron deficiency anemia; The iron absorption in 17 patients out of 23 (83%) was higher than normal subjects and mean was 50±20% ranging from 6% to 86%. This is 1.7 times value for Japanese normals. Serum iron was between 16 and 51 μg/100 ml, and average was 33 μg/100 ml.

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±16%, ranging from 6 to 59%. Serum iron was between 16 and 51 μg/100 ml and average was 33 μg/100 ml.

4. Liver cirrhosis; The average iron absorption of 14 patients was 26±14% ranging from 5 to 51% and serum iron level was between 29 and 124 μg/100 ml with an average value of 117 μg/100 ml.

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 ml of fresh blood. A relatively large amount of 51Cr (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 × 10⁴/mm³. According to the platelet survival curves, they separated into 2 groups. In 4 patients, the studies were performed
autologously. The half lives (T1/2) ranged from 2.3 to 3.3 days, mean 2.8 days and the recoveries from 31.0 to 49.2%, mean 38.0%. In 2 others with probable hypersplenism, T1/2 were shortened to 1 and 2 hours when the studies were done homologously. After one of the latter was splenectomized, the T1/2 was prolonged to 2.5 days with the increase of platelet counts from 3.4 to $6.5 \times 10^4$/mm$^3$.

Our method is considered to have following advantages over the conventional way: The study can be done with autologous blood. The special equipment is not required. Only one done is necessary when it is carried out homologously. But the data on the external organ counting must be interpreted carefully when the study is done autologously because of the possible contamination by $^{31}$Cr-labeled red cells.

The results of the 4 cirrhotic patients were consistent with those reported by Kotilainer. Our method seems accurate enough for clinical and experimental applications.