Iron deficiency is caused by the increase of iron loss, decrease of iron absorption, abnormal eating habit, and etc. Iron loss is mainly caused by bleeding and the amount of blood loss is determined by using Cr-51-RBC. Iron absorption is determined by using a whole body counter measuring 14 days whole body retention after oral Fe-59 dose. Iron deficiency status is consist of simple iron deficiency (IDA), iron deficiency with complication (IDA+c), iron deficiency without clinical symptom (ID), and etc. Among these iron deficiency status, iron absorption was increased in IDA, less in IDA+c and it was between IDA and normal in ID. No significant difference was observed between normal male and female in iron absorption, although storage iron was lower in normal female than male. In general, iron absorption was mainly controlled by the amount of storage iron and affected by hematopoietic activity. Iron absorption delivers the important informations for the diagnosis of iron deficiency status and for choosing the route (oral or intravenous) of iron administration in treating the patients with IDA and iron deficiency tissue disorders.

Platelet kinetics by radioisotope labeled platelets were investigated in patients with splenomegaly. Three patients had chronic myelogenous leukemia, 2 cases of essential thrombocythemia, a group of myeloproliferative disorders with splenomegaly, 2 cases of hereditary spherocytosis, a group of hyperplastic splenomegaly, 3 cases of liver cirrhosis, 3 cases of so-called Banti’s syndrome and another patient had a congenital splenomegaly.

Platelet counts and platelet production were calculated from platelet kinetic data. In patients with severe liver disease, the splenic uptake of the first labeled platelet was significantly lower in group II than that in group I. These results indicated that mild-dose warfarin inhibited the deposition of platelets on the intracardiac thrombi and thrombocytopenia in the patients with intracardiac thrombosis which were detected by indium-111 platelet scintigraphy.

In conclusion, increased platelet pooling in the spleen were commonly observed in various disorders with splenomegaly.