days group), and five mg of chloramphenical was administered intraperitoneally once a day for thirty days. (CP thirty days group). The ferrokinetics was studied at twelve hour after last CP administration. For the studies of ferrokinetics, 1.0 μCi of ^{59}Fe-ferrous citrate in a volume of 0.25 ml of sterilized physiological saline was administered into mouse tail vein intravenously. Peripheral red blood cell count, reticulocyte count, the serum iron level, plasma iron disappearance time, and ^{59}Fe uptake of bone marrow, liver and spleen were determined in such mouse.

The results were as follow.

1) In normal mice, serum iron level was 242 γ/dl, red blood cell count was $1051\pm85\times10^4$, reticulocyte count was $28\pm15\%$, P.I.D.T. was 70 ± 10 minutes and ^{59}Fe reappearance rate was $83\pm13\%$ at 24 hours: $97\pm12\%$ at 48 hours. The uptake of ^{59}Fe in the bone marrow and spleen showed a peak at six hours after ^{59}Fe administration. The uptake of ^{59}Fe in the liver increased until six hour after ^{59}Fe injection, and then made a plateau line.

2) In CP three days group, serum iron level was 298 γ/dl, P.I.D.T. was 100 ± 30 minutes, ^{59}Fe reappearance time was $33\pm13\%$ at 24 hours; $55\pm20\%$ at 48 hours, red blood cell count was $1053\pm119\times10^4$ and reticulocyte count was $16\pm11\%$. The uptake of ^{59}Fe in the spleen was extremely decresaed, and slightly increased the uptake of liver and bone marrow.

3) In CP thirty days group, serum iron level was 360 γ/dl, P.I.D.T. was 100 ± 20 minutes, ^{59}Fe reappearance time was $67\pm6\%$ at 24 hours: $82\pm3\%$ at 48 hours, red blood cell count was $757\pm72\times10^4$, and reticulocyte count was $54\pm23\%$. The uptake of ^{59}Fe in the bone marrow, spleen and liver were decreased.

4) From these data, it was concluded that CP damged the erythroblast colonies in the spleen at first, and then bone marrow failure was followed.

Erythrokinetic Studies in Patients with Ineffective Erythropoiesis

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Erythrokinetic studies were performed in twenty-eight cases, including patients with hereditary sperocytosis (7), pernicious anemia (2), paroxysmal nocturnal hemoglobinuria (3), erythroleukemia (4), refractory anemia (5), myelofibrosis (6, primary one 5 and secodary one 1) and thalassemia (a case of β-thalassemia minor). (Figures in brackets show the number of cases.)

Simultaneous ^{59}Fe and ^{51}Cr measurements as well as morphologic examinations were studied on these patients. Plasma iron turnover and bone marrow index were used as total erythropoiesis indices and as effective erythropoiesis indices were used reticulocyte, red cell iron turnover and red cell survival (^{51}Cr) index. The erythropoiesis indices (total and effective) and bone marrow efficiency were calculated according to the formulas presented by Haurani and associates. High degree of ineffective erythropoiesis was observed in all cases of erythroleukemia and pernicious anemia and some cases of paroxysmal nocturnal hemoglobinuria, myelofibrosis, refractory anemia and thalassemia. Mean values of bone marrow efficiency in blood disorders studied were as follows: hereditary spherocytosis 82.0%, pernicious anemia 40.4 %, paroxysmal nocturnal hemoglobinuria 46.4 %, erythroleukemia 8.8%, refractory anemia 49.8%, myelofibrosis 73.3% and thalassemia minor 56.2%.

In this paper, the meanings and limitations of each erythropoiesis index, especially ferrokinetics indices, in various blood disorders were discussed on the basis of presented data. Moreover, it has been clarified in patients with ineffective erythropoiesis that significant cor-

relation between serum iron level and plasma iron disappearance (T 1/2) were lost and the percentage of bone sideroblasts showed marked increase in all cases.

The Zinc Matabolism in Rauscher Leukemic Mice

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It is widely recognized that the zinc is essential for the normal growth of plants and animals. Recently some reports, which evaluated relations between the zinc and malignant neoplasms, have been presented. We examined the zinc contents and ^{65}Zn kinetics in Rauscher leukemic mice and normal mice. For estimating the zinc contents we used the atomic absorption spectrophotometer and the ^{65}Zn-glycine complex as the tracer. The results were as follows: the zinc contents of the leukemic spleen ranged from 2.0 to 2.2 μg per 100 mg, those of the normal spleen from 3.6 to 5.5 μg per 100 mg. The zinc contents and of the leukemic liver were slightly lower than in normal mice. The spleen to liver ratios of zinc content were less than 1.0 in leukemic mice and more than 1.0 in normal ones. It might be suggested that in leukemic state the organ zinc distribution is different from the zinc distribution in normal state. The zinc content of the spleens was estimated at weekly intervals up to the leukemic phase after Rauscher virus inoculation. It decreased to one half of the normal level one week after inoculation and remained at the same level until the leukemic phase, in which it increased slightly in comparison with the preleukemic phase.

After subcutaneous injection of ^{65}Zn-glycine complex whole body radioactivities were counted on the 1st, 3rd and 7th day. The retention ratios of ^{65}Zn were higher in leukemic mice than in normal mice. The ^{65}Zn-radioactivities in spleens of leukemic mice were higher than in normal mice, but no remarkable differences were demonstrated in the livers of both groups.

The spleen to liver ratios of ^{65}Zn-radioactivities were higher in leukemic mice than in normal mice. And the increasing rate of the ratios for the first three days and the decreasing rate of the ratios for the next four days were higher in leukemic mice. These results would indicate an increased turn over of ^{65}Zn-kinetics in leukemic mice, particularly in leukemic spleens.

We have reported that Rauscher virus infects the spleen as the target organ about one week after virus inoculation. Our results in this paper would suggest that there are some relations between the role of the spleen as target organ in the leukemic virus-proliferation and the zinc metabolism in the leukemic spleens.