ing, mg of leukocyte nitrogen of each sample were determined by the micro-Kjeldahl method. CPM/mg-leukocyte-nitrogen was plotted in semi-logarithmic paper and T½ was determined. Total blood granulocyte pool (TBGP), circulating granulocyte pool (CGP) and granulocyte turnover rate (GTR) were calculated from the blood volume and differential counts of leukocytes.

The disappearance of labelled leukocytes was measured in 12 subjects. The obtained curve indicated an exponential fashion in 5 subjects and not exponential one in 4 subjects. In the other 3 subjects, however, the disappearance curve and single value for T½ could not be obtained. T½ in the group of hematologically normal subjects were from 4 to 11 hours and in a subject of leukocytosis was 16 hours.

But the T½ of CML in relapse was markedly prolonged, showing 45 hours. Both pool size and turnover rate in subject were larger than normal, while that of other cases were within normal value. No remarkable difference between T½ of in vivo and in vitro labeling method was recognized. Since the radioactivity of the leukocytes labelled with 51Cr was lower than those with DF32P, it was considered that DF32P was superior to 51Cr in measurement of life span of the leukocytes.

The Splenic Uptake of 203Hg-MHP-Treated Red Cells

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The uptake of MHP-treated red cells by the spleen was measured in normal and abnormal subjects. 203Hg-labeled MHP chromatogram was prepared with Toyo No. 51 paper and a solvent consisting of 2 parts of benzene, 2 parts of acetic acid and 1 part of water. The strip was left in the solvent for about one hour and then analysed by chromatoscanning method which gave a separation of 203Hg ion derived from MHP and free inorganic 208Hg ion, proving negligible contamination of free inorganic mercury in MHP. The Rf of labeled MHP was also shown to be similar to carrier MHP. (Rf = 0.7). Surface counting over the precordium, spleen and liver was performed by means of shielded scintillation probes with wide angled collimators after venous injection of the mixture of 0.5 ml red cells and 1.25 mg MHP (100 μCi). The spleenic scanning was also performed. The size of spleen scanned at the supine anterior view was measured by planimetry, giving the normal size to be less than 90 cm². Some subjects with normal size of spleen showed the rapid rate of splenic uptake of the MHP-treated red cells. These subjects have infection, blood diseases, liver diseases etc., which may stimulate phagocytosis of RES in the spleen.

Some subjects with splenomegaly, on the other hand, did not show the rapid increase in the rate of splenic uptake of the MHP-treated red cells. However most subjects with splenomegaly indicated the rapid uptake rate of the spleen within 30 minutes compared with the subjects with spleen of normal size.

It was suggested that there were at least two compartments in the spleen for the removal of MHP damaged red cells. These were the blood pool and sequestering pool of damaged red cells in the spleen. The presence of delayed rapid uptake of MHP-treated red cells after 30 minutes suggested the enlargement of the sequestering pool of spleen. Splenomegaly itself suggested usually the enlargement of both blood pool of spleen and the damaged red cells uptake pool, however there were certain cases with splenomegaly which accompanied no increase in RES function of the spleen.