THE STUDY OF THE RED BLOOD CELL LABELING METHOD WITH $^{11}\mathrm{Co}$ For RI-ANGIOGRAPHY

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The angiography is widely practiced in the course of vascular disease treatment. In order to practice this examination easily and non-invasively, RI-using angiography is to be taken in. By using cyclotron produced ¹¹CO-carbon monoxide gas, inhalation RI-andiography method is under exploiting in our institute. The labeling rate and blood uptake were examined comparatively between in vitro method and inhalation method in this work.

About 15mCi of ¹¹C-carbon monoxide diluted with about one liter of the air. Five volunteers inhaled this RI gas by the single breath method. The ¹¹C-carbon monoxide uptake to the pulmonary blood flow was measured with the positron camera connected with on-line computer, RI activities at the thigh and in the expiratory gas were also measured by ratemeters. The mean valus of pulmonary blood flow uptake in five healthy men was 45.7%/20sec. The activities of venous blood, which were punctured five minutes after inhalation, were counted for calculation of the labeling rate of red blood cells. 98.9% of ¹¹C-carbon monoxide activities distributed in red blood cells in average.

A middle wide U-shaped glass tube was used for in vitro labeling. For ten minutes, \$^{11}\$C-carbon monoxide gas diluted with the nitrogen gas introduced into this tube and heparinized blood, diluted with saline water, was bubled. The flow rate of this gas, which contained 100µCi \$^{11}\$C-carbon monoxide per lml nitrogen gas, was 100ml/min. After ten minutes bubling, the blood trapped 24.6% of \$^{11}\$C-carbon monoxide activity. The mean value of red blood cell labeling rate was 99.0%.

Though both method gave very high red blood cell labeling rate, the inhalation method was more easy in use. PLATELET KINETICS IN NON-STATIONARY STATE—
—MEASUREMENT AND ANALYSIS IN CYCLIC THROMBOPENIAS—
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In face of non-stationary blood-cell kinetics, interpretation of the data is complicated. We have experienced two cases of cyclic thrombocytopenia, which provided us with a typial pattern of non-stationary kinetics of platelets. The kinetics study was attempted and the data were analysed in the following way to elucidate the patho-physiology of this disorder in which cyclic occurrence of thrombopenia drew our attention to an aspect of disturbed control on blood cell production. In the first case, C.N., 58, f., with cyclic change in the platelet count(P1-C) between $0.3 - 22.0 \times 10^4$ /mml in every 20 days, 51 Crplatelet survival study (51 Cr-P1-Sv) and 75 Se-Methionine production study(75 Se-P1-Pd) were started at the increasing phase and then at decreasing phase. In the second case, Y.K., 21, f., with P1-C between 0.1- 47.4×10^4 /mml in every 25-30 days, 51 Cr-P1-Sv were started at various stages of increasing and decreasing phases but 75 Se-P1-Pd study was not performed.

In both cases, 51 Cr-P1-Sv revealed various survival rate of the labeled cells depending on the stage of the cyclic change, the survival being longer in the increasing and shorter in the decreasing phase. With obtained data of P1-C in the circulation, F(t), and the survival character of ⁵¹Cr-P1-Sv,R(t), daily production rate of platelets, U(t), and their cohort survival character,C(t), were calculated by setting and solving the convolutional formulae. A'linear and exponential model and multi-hit model were addopted as the survival character which was determined independently of cell-age population. Daily production rate, calibrated so as to fit F(t) and to satisfy R(t),fluctuated between $0 - 8.5 \times 10^4 / mml/d$ and extracellular destruction factor was in decrease phase three times that in increase phase. 75Se-P1-Pd exhibited a quite different incorporation rate of 75 Se into the circulating platelets. Calibrated date of 75Se-platelets production rate substantiated the data obtained with F(t) and R(t). Alteration in the survival character was evidenced by ⁵¹Cr-P1-Sv with the cells from the same donor successively performed along stages of decreasing and increasing phases. The extracellular destruction factor became augmented remarkably toward the end of decreasing phase but improved in the increasing phase with corresponding R(t) changes.