The Calibration of the Splenic Content of the Plasma and Blood Cells by Determining Xenon Clearance and the Mean Transit Time of $^{131}$I or $^{99m}$Tc or $^{51}$Cr-labelled Blood Elements in the Spleen

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The splenic content of the plasma and blood cells (erythrocyte, leukocyte and platelet) was measured by determining the blood flow rate, the mean transit time of each blood element and the size of the organ.

Instantaneous injection of the radiotracers was done into the celiac (splenic) artery in order to obtain corresponding radiosplenograms as well as radioprecordiograms. The splenic blood flow was measured in milliliter per minute per unit volume by a radioxenon clearance rate. In the calculation procedure, its tissue-blood partition coefficient was corrected for the hematocrit of splenic blood as well as for that of the blood entering and exiting from the spleen. The former was calculated by the plasma and erythrocyte transit time and the latter was represented by the hematocrit value of the venous blood.

The radiosplenograms which corresponded to each blood-element tracer ($^{131}$I-albumin, $^{51}$Cr-erythrocytes, $^{51}$Cr-leukocytes or platelets) were subjected to analysis by the simulation technique using an analog computer in order to calculate their respective mean transit time.

By multiplying the flow rate by the transit time, the content volume was calculated in milliliter per unit volume of the spleen. Assessing the splenic volume by scintigraphy, we eventually calibrated the total mass of the plasma and the blood cells contained in the spleen.

In hereditary spherocytosis, the plasma content per unit volume was significantly smaller, while that of the erythrocytes was remarkably greater than those in the control. This phenomenon implies remarkable hemoconcentration which provide the spleen to manifest its hyperfunctioning state.

In congestive splenomegalies associated with portal hypertension, the plasma and erythrocyte content per unit volume was significantly increased. But the plasma turnover in the splenic pool was significantly reduced in cirrhotics when compared to that in non-cirrhotics, a fact which made us speculate the existence of a different mechanism in producing the congestive state in the portal system between these two groups.

In such portal congestive splenomegalies, the total content of leukocytes and platelets in the spleen was invariably and remarkably increased. This led us to the assumption that the enlarged splenic pool plays a major role in manifesting cyto-
penia in the systemic circulation in such dyscrasias. So far as the platelet content was concerned, the amount calibrated by this method was rather small as compared to that estimated from the percent of recovery in the circulation of the labelled platelets which were intravenously administered in those cases with congestive splenomegaly as well as with idiopathic thrombocytopenic purpura. This dissociation is probably attributable to the existence of an extra-splenic marginal pool.

This method is therefore considered to be valuable since the content can be calibrated independently on either an individual variation in the counting efficiency of intrasplenic radioactivity or on the existence of an extra-splenic marginal pool of these blood cells.

Platelet Survival Studies in the Stroke-prone Spontaneously Hypertensive Rats

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Since it has been established that platelets contribute to maintain vascular integrity and are used in the formation of intravascular thrombi, the fate of blood platelets was pursued by platelet survival and turnover in the stroke-prone spontaneously hypertensive rat (SHRSP) in comparison with those in the stroke-resistant SHR (SHRSR) and in the normotensive control rat of Wistar-Kyoto (WK). SHRSP and SHRSR were established by successive selective breeding of Wistar-Kyoto rats with stroke (cerebral infarction and/or hemorrhage) and those without stroke, respectively, for many generations. Incidence of spontaneous cerebral lesions in male SHRSP over 100 days of age was more than 80%, while that in similar SHRSR less than 10%. In each group, male rats around 4 months of age were used and no stroke was yet observed throughout this experiment. However, blood pressure was already high in all rats except for WK.

Platelet half-life time in SHRSP was slightly but significantly shorter than that in any other groups of rats, irrespective of the type of platelet donors. Mean platelet consumption was also significantly increased only in SHRSP. Platelets of SHRSP injected into SHRSR showed normal survival. These data support the concept that the shortened platelet survival in SHRSP is brought about by some extracorpuscular abnormalities. Although the vascular changes in SHRSP could be the most likely explanation for the shortened platelet survival, its mechanism remains to be solved. This investigation suggests that studies of the platelet survival in hypertension may be useful to predict the development of stroke before its clinical recognition.