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, bloos 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.