Time

13.15 **0373** BLOOD PLATELET COUNT AND BLOOD PLATELET SURVIVAL IN MALE STROKE PRONE, SPONTANEOUSLY HYPERTENSIVE RATS DURING AGEING

D.G. Meuleman*, G.M.T. Vogel and A.M.L. van Delft, Organon Scientific Development Group, Oss, The Netherlands.

Blood platelets play probably an important role in the occurrence of stroke. We determined the survival of radiolabelled blood platelets, the blood platelet count and blood pressure in male stroke prone, spontaneously hypertensive rats during ageing. Our investigations revealed that 1) blood pressure starts to rise at an early age (8 weeks) reaching a steady state (approx. 250 mmHg) at 15-20 weeks of age, 2) blood platelet count is steadily decreasing from 9 weeks of age on, reaching a steady state (approx. 50% of the original platelet count) at 20 weeks of age, 3) blood platelet survival is gradually shortening from 12 weeks of age on, reaching a steady shortened half-life of 18 hours (normal value approx. 42 hours). It is concluded that the rise in blood pressure induces at first instance a moderately increased platelet consumption, which is not compensated by an increased platelet production and which is not detectable by platelet survival measurement. In a later phase, when the blood pressure is in a steady state, platelet consumption is further increased, leading to significantly shortened platelet survivals. This process is then compensated by increased platelet production leading to a steady state thrombocytopenia.

13.30 **0374** KINETIC STUDY OF THROMBOGENESIS ON STROKE-PRONE SPONTANEOUSLY HYPERTENSIVE RATS (SHRSP). THE EFFECT OF PLATELET-INHIBITING DRUG AND VITAMIN E.

A.Kuramoto, H.Okita and S.Koganemaru, Department of Medicine, Research Institute for Nuclear Medicine and Biology, Hiroshima University, Hiroshima, 734, JAPAN

The increase of platelet production was demonstrated in stroke-prone SHR(SHRSP) after 10 weeks of age, probably due to increased platelet consumption compared with those in stroke-resistant SHR(SHRSR)(Koganemaru,S., et al. Thrombos. Haemostas.(Stuttgart)38:139, 1977). We extended our study to see the effect of aging, blood pressure, platelet-inhibiting drug and vitamin E(Vit.E) on this kinetic change. SHRSP(A_1-sbF_{37-39}) and SHRSR (B_1F_{36-38}) with matched age (5 weeks, 10 weeks and 9-12 months) and sex were used. The maximum uptake of ^{75}Se-methionine(^{75}SeM) into platelets was not affected in both salt loading group and clonidine group in SHRSP. A significant decrease was observed in dipyridamole group (0.096 ± 0.020, $p<0.05$). The platelet survival time was shortened in Vit. E deficient group of SHRSR; 3.0 days at 9-10 weeks. In Vit.E sufficient group of SHRSP, the platelet survival time was corrected to normal. The maximum uptake of ^{75}SeM increased in both Vit.E deficient group of SHRSP (0.119 ± 0.010 vs 0.192 ± 0.030, $p<0.01$) and SHRSR (0.071 ±0.016 vs 0.119 ± 0.018, $p<0.01$). The increased platelet production was not affected by blood pressure but it can be suppressed by treating with dipyridamole or giving Vit.E before 10 weeks of age.

13.45 **0375** PLATELET FUNCTION AND VASCULAR RESPONSIVENESS IN SPONTANEOUSLY HYPERTENSIVE RATS (SHR)

T. Motomiya*, N. Mashimo and H. Yamazaki, Division of Cardiovascular Research, Tokyo Metropolitan Institute of Medical Science, Honkomagome, Tokyo, and Department of Internal Medicine, Tokyo Medical and Dental University, Yushima, Tokyo, Japan.

SHR has been known to develop arterial and venous thrombosis at advanced age. However cerebrovascular and cardiorespiratory disturbances induced by injection of ADP or arachidonic acid have been reported as being milder in SHR than in normal Wistar rats (NWR). In this study, utilizing 14 mature SHR (24-week of age) and 6 young SHR (6-week) and same number of age-matched NWR, platelet aggregation, ^{14}C-serotonin release and adenine nucleotides content were measured. In addition ADP and ATP contents were determined in experimentally induced pulmonary thromboembolism in SHR and NWR. Vascular responsiveness to rabbit aorta contracting substance (RCS) and serotonin was also studied. ADP-induced platelet aggregation was significantly decreased in mature SHR as compared with NWR ($P<0.$) and showed a tendency to decrease in SHR at 6-week. Serotonin release was significantly decreased in SHR as compared with NWR at 24 and 6-week ($P<0.01$). Decrease in ADP content after induction of thrombosis was smaller in SHR than NWR ($P<0.05$). Aorta smooth muscle strip contraction induced by RCS and serotonin was not different between SHR and NWR at 24 and 6 weeks of age. Decrease in platelet function in SHR even at 6-week of age when hypertension has not been established seems to represent an inherited defense mechanism against thrombus formation.