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PATIENTS WITH MILD ESSENTIAL HYPERTENSION HAVE INCREASED PLATELET SIZE AND RELEASE REACTION AND SHOW INCREASED RECEPTOR RESPONSE TO INFUSED ADRENALINE. K. Lande, I. Os, SE, Kjeldsen, A. Westheim, I. Aakesson, I. Hiernann, I. Eide and K. Gjesdal, Departments of Internal Medicine and Clinical Physiology, Ullevål Hospital, 0407 Oslo 4, Norway.

Hypertensive (n=35) and normotensive (n=44) men all 42 years old were studied. The hypertensive (HT) had larger venous platelets than the normotensive (NT) ( $7.46 \pm 0.10$  vs  $7.12 \pm 0.09 \cdot 10^{-15}$  l,  $p=0.01$ ). Plasma concentration of  $\beta$ -thromboglobulin (BTG) was increased in arterial blood in hypertensive ( $40 \pm 8$  vs  $21 \pm 2$  ug/l,  $p=0.02$ ) while the venous values were similar in the two groups. Despite similar sampling procedure, the normotensive subjects had markedly higher BTG concentration in venous compared to arterial blood ( $p<0.01$ ) at variance from the hypertensive where the arteriovenous difference in plasma BTG concentration was not present. Adrenaline was infused to 13 hypertensive and 12 normotensive subjects with dose gradually increasing to  $0.04$  ug/kg/min. Forearm blood flow was measured by strain gauge technique and relative forearm resistance calculated as mean blood pressure divided by flow. Twelve normotensive subjects (control group) received saline infusion.

Change during 40 min adrenaline infusion (mean $\pm$ SEM)

	$\Delta$ Forearm Resistance (relative)	$\Delta$ Platelet Count $10^9/l$	$\Delta$ Platelet Size $10^{-15}$ l	$\Delta$ Plasma BTG $\mu g/l$	$\Delta$ Plasma Adrenaline $ng/l$
I HT	$-39 \pm 9^1$	$31 \pm 6^1$	$0.22 \pm 0.07^2$	$18 \pm 12^2$	$373 \pm 60^2$
II NT	$-9 \pm 4$	$14 \pm 3$	$0.16 \pm 0.11$	$13 \pm 4$	$355 \pm 64^2$
III NT(saline)	$-7 \pm 5$	$-6 \pm 5$	$0.10 \pm 0.07$	$4 \pm 3$	$-17 \pm 5$

<sup>1</sup>:  $p<0.05$  compared to II

<sup>2</sup>:  $p<0.05$  compared to III

Change in forearm resistance reflects  $\beta_2$ -activation of smooth vascular cells, change in platelet count reflects splenic liberation of platelets in response to adrenergic stimulation while change in BTG may reflect platelet release upon stimulation of  $\alpha_2$ -receptors. Thus, middle aged men with essential hypertension show increased sensitivity to adrenaline infusion in vascular smooth muscle, spleen and platelets.

## PRENATAL AND NEONATAL HAEMOSTASIS

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THE ABSORPTION, EXCRETION AND TRANSPLACENTAL TRANSPORT OF VITAMIN K IN THE PERINATAL PERIOD. S. Suzuki, Dept. Obstet. & Gyn., School Med., Hokkaido Univ., Sapporo, Japan

It is a well-known fact that hemorrhages are observed in wholly breastfed infants beyond the neonatal period. In order to clarify vitamin K (VK)-deficiency, it is necessary to follow-up the absorption and excretion of VK<sub>2</sub>. 1. To 128 cases of newborns. i) The activity of VK-dependent factors (II, VII, X) were determined by Hepaplastin test (HPT). ii) Using Latex-test, PIVKA-II was tested. We found values of HPT (Y) and PIVKA-II (X) to be inversely proportional in the relation.  $Y=61.9 - 6.7 X$  ( $r=-0.3$ ). 2. These 15 cases of hypoprothrombinemia, VK<sub>2</sub> 6mg, VK<sub>2</sub> 2mg were given, and plasma VK<sub>2</sub>-concentration was measured by gas chromatography. After 3 hours VK<sub>2</sub> 6mg concentration was 1030ng/ml; VK<sub>2</sub> 2mg, was 224ng/ml. This clearly shows a dose-response relation. 3. VK<sub>2</sub> transplacental transport was also proved by using umbilical venous blood after Cesarean section. (Before Cesarean section, VK<sub>2</sub> 60mg was given.) In umbilical venous blood, relatively high doses of VK<sub>2</sub> (50 - 120ng/ml) were demonstrated. Additionally, the  $\gamma$ -carboxyglutamic acid-concentration in the urine of newborn, who received VK<sub>2</sub>-syrup was higher than those who did not receive it.

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THE VITAMIN K DEFICIENCY IN INFANCY IN JAPAN -- THE SECOND NATION-WIDE SURVEY. T.Nagao (1), Y.Hanawa, K.Sawada, I.Tsukimoto (2), I.Ikeda, M.Komazawa, K.Shiraki, S.Shirahata, Y.Tsuji, T.Terao, E.Matsuyama, M.Maki, S.Mikami, B.Murata, K.Motohara, K.Yamada and Y.Yamamoto. Kanagawa Children's Med. Ctr. Yokohama (1), Univ. of Toho, Tokyo, Japan (2).

Questionnaires were sent to 1,218 hospitals with more than 200 beds, in order to know the incidence of hemorrhagic disease due to vitamin K deficiency in infancy beyond 2 weeks after birth, during 4 and a half years, i.e. from January 1981 to June 1985. Out of the 534 cases reported, 407 had no obvious reasons for vitamin K deficiency: "idiopathic vitamin K deficiency in infancy". Other 68 cases had bleeding episodes due to vitamin K deficiency associated with hepatobiliary lesions (e.g. congenital bile duct atresia), chronic diarrhea, long term antibiotic therapy and so on: "secondary vitamin K deficiency in infancy". The third group consisting of 59 cases was so called "near miss" type, in which hemorrhagic tendency was discovered at the time of mass screening tests for vitamin K deficiency or by chance without any clinical hemorrhage. In the idiopathic group, 345 cases (84.8%) developed their bleeding episodes between 21 and 59 days of age, and 368 cases (90.4%) were wholly breast-fed. Intracranial hemorrhage was seen in 338 cases (83.0%) of this group. In most cases of this series (97.3%), no vitamin K was supplemented after birth. Administration of vitamin K is an urgent routine procedure during the first one or two months of life for all newborn babies, although the incidence of the idiopathic vitamin K deficiency in infancy has not decreased significantly compared to the results of the first nation-wide survey (Jan. 1978 - Dec. 1980). This study was sponsored by the Ministry of Health and Welfare of Japan.