

**Thursday, July 16, 1981**

## **Poster Presentations**

# **Atherosclerosis – II**

**11:00–12:30 h**

**Grand Ballroom Lobby Boards 262–273**

### **0764**

DIETARY INDUCED CHANGES IN ARTERIAL THROMBUS FORMATION IS INDEPENDENT FROM THROMBOXANE-PROSTACYCLIN BALANCE. G. Hornstra (1), E. Haddeman (2) and J.A. Don (2). Department of Biochemistry, Limburg University, Maastricht, the Netherlands (1) and Unilever Research, Vlaardingen, the Netherlands (2).

The time needed for the complete thrombotic obstruction of a plastic aorta prosthesis appeared a reliable parameter for the arterial thrombosis tendency of rats. We recently demonstrated that in this model arterial thrombus formation is regulated primarily by prostaglandin-like substances. Therefore we used this model to test the hypothesis that the arterial thrombosis tendency depends on the ratio between prothrombotic thromboxane  $A_2$  ( $TxA_2$ ) produced by activated blood platelets and antithrombotic prostacyclin ( $PGI_2$ ) formed by vascular tissue. Groups of 24 rats were fed diets containing 5 energy % (en%) of either sunflowerseed oil (SO, control) or hydrogenated coconut oil (essential fatty acid deficient). Four other groups received the control diet, enriched with 45 en% linseed oil, cod-liver oil or hydrogenated coconut oil. After 8–10 weeks' feeding, arterial thrombosis tendency was measured in 50% of all animals. The other animals were used for quantifying the  $TxA_2$  production of collagen-activated blood platelets and the formation of  $PGI_2$  by freshly punched-out pieces of aorta.  $TxA_2$  was measured as MDA (spectrophotometry) or HHT (GC/MS).  $PGI_2$  measurements were performed with the platelet aggregation bioassay, using a standard curve obtained with synthetic  $PGI_2$ . Control measurements were carried out using tissue of the same blood vessel pretreated with indomethacin. The various diets induced highly different arterial thrombosis tendencies. Platelet  $TxA_2$ - and vascular  $PGI_2$ -productions were significantly affected by the diet in a strikingly similar way: both were positively correlated with the arterial thrombosis tendency and the  $TxA_2/PGI_2$ -ratio remained essentially unchanged. Consequently, no correlation existed between this ratio and arterial thrombosis tendency. From this study it is suggested that the potency of platelets to produce  $TxA_2$  is of greater importance for arterial thrombus formation than the ability of the vessel wall to generate  $PGI_2$ .

### **0765**

PLASMA LIPOPROTEIN LEVELS IN THYROID DISEASES IN RELATION TO CORONARY HEART DISEASE. B. Lipinski, F. Azizi, I. Lipinska, S. Naidu and J.E. Mannix. Vascular Laboratory, Departments of Research and Medicine, St. Elizabeth's Hospital, Tufts University School of Medicine, Boston, MA. 02135.

Plasma high density lipoprotein (HDL), low density lipoprotein (LDL), very low density lipoprotein (VLDL) and total cholesterol (TC) were determined in 30 patients with thyroid dysfunction and in 58 healthy subjects. The lipoproteins were separated by means of polyacrylamide gel electrophoresis, quantitated by densitometric scanning and the results expressed as a percent of total. Percentage distribution of lipoproteins in patients with hyperthyroidism was not different from that observed in healthy subjects. In patients with hypothyroidism HDL, VLDL, LDL and TC were 17%, 16%, 66% and 320 mg/dl, and in healthy subjects were 34%, 8%, 60% and 206 mg/dl respectively. When lipoproteins were analysed in relation to the parameters of thyroid function, it was found that only HDL % values correlated well with serum thyrotropin (TSH) concentration ( $r=0.62$ ) and with the free triiodothyronine ( $T_3$ ) index in hypothyroid patients ( $r=0.54$ ). In patients who developed hypothyroidism after radioactive ablation of the thyroid, HDL % became subnormal as early as 2 months after treatment. Replacement doses of l-thyroxine ( $T_4$ ) normalized plasma HDL in patients with hypothyroidism. In view of these findings, it is suggested that low levels of HDL and increased content of VLDL in plasma may play a role in the development of coronary artery disease in patients with thyroid dysfunction.