1161 RETENTION OF PLATELETS BY FOREIGN SURFACES AND ITS MODIFICATION BY CHEMICAL SUBSTANCES

H.D. Reuter and H. Hielscher, Medizinische Universitätsklinik Köln-Lindenthal, 5 Köln 41 F.R.Germany

For investigation of platelet retention from streaming citrated (3.8%) blood by foreign surfaces a test system has been developed consisting of a peristaltic pump by which anticoagulated blood is passed continuously through silicone tubing connected with glass bead and charcoal filters. Maximum retention in glass bead filters as well as in filters packed with charcoal particles was observed when citrated blood was passed through dry filters (mean + S.D.: 30.3+10.5% and 37.4+10.5% at a passage time of 10 min.). By prior coating the filter particles with saline or 20% albumin retention was reduced to 24.8 +12.9% (n.s.) and 14.8+3.9% (p 0.01), respectively in glass bead filters and to 22.9 +5.9% (p 0,01) and 13.9+8.2% (p< 0.01), respectively in charcoal filters. Coating of the glass beads with 0.6% fibrinogen and 80 µM/l ADP significantly (p <0.01) increased retention while addition of 0.25 mg/ml of RA 233 to citrated blood significantly (p < 0.01) reduced retention in charcoal filters (67.1+15.8%, 42.3+12.1% and 10.2+6.7%). Further the kinetics of retention, the rate of hemolysis and the effect of passage on platelet metabolism was investigated.

1162 ANTITHROMBOTIC ACTIVITY OF POLYDEOXYRIBONUCLEOTIDES OF MAMMALIAN ORIGIN(LABORATORY CODE: FRACTION P) IN EXPERIMENTAL ANIMALS

R.Niada, M.Mantovani, G.Prino, R.Pescador and G.F.Nardi, Crinos Biological Research Laboratories, Villa Guardia (Como), Italy

Fraction P(FP)is a polydeoxyribonucleotidic substance of mammalian origin which was found able to activate the fibrinolytic system of some experimental animals.We have investigated the possible antithrombotic activity of FP in three different experimental models. In the collagen-induced thrombosis of the rabbit femoral vein,pretreatment with FP i.v.(50, 100 or 200 mg/kg) reduced the thrombus dry weight by 42% (P<0.005), 50% (P<0.001) and 72% (P<0.001),respectively;pretreatment with FP per os(12.5, 25 or 50 mg/kg) decreased the thrombus dry weight by 22% (n.s.), 46% (P<0.001) and 69% (P<0.001), respectively. In the electrically induced thrombosis of rat carotid artery, pretreatment with FP i.v. (37.5, 75 or 150 mg/kg)reduced the fall in arterial surface temperature by 20%(P<0.005), 62% (P<0.025)and 86% (P<0.001),respectively.In the hamster cheek pouch model, venular thrombosis induced by iontophoresis of ADP was inhibited by 85%(P<0.025)when the animals were pretreated with FP i.v. (2 mg/kg) or by 95% (P<0.025) when FP was given per os (1 mg/kg). These antithrombotic effects lasted longer than the activation of fibrinolysis measured in ex vivo studies in the same animal species. This would suggest that a more complex mechanism(possibly including vascular factors at local level)could be responsible for the in vivo effect of FP as an inhibitor of thrombus formation.

1163 THE EFFECT OF ACETYLSALICYLIC ACID ON THE PROLIFERATION OF CULTIVATED AORTIC WALL CELLS

J.Mey, H. Schulte and W.H. Hauss, Institut für Arteriosklerosеforschung an der Universität Münster, West Germany

In former studies we showed that risk factors induce an acceleration of the proliferation of the arterial wall cells. Furthermore we examined the influence of acetylsalicylic acid (ASA) upon the proliferation of arterial wall cells of normal animals and of animals which had been damaged by risk factors. We received the following results:

1. ASA given to the cell culture inhibits the proliferation of aortic smooth muscle cells (ASMC), endothelial cells and adventitial cells of minipigs,
2. ASA given to the culture of ASMC of rats which had been damaged by arterial hypertension, by staphylolysine or by atherogenic diet reduces their increased proliferation rate nearly to normal.
3. ASA-treatment of rats which had been damaged by injection of staphylolysine reduces the increased proliferation rate of ASMC of these rats nearly to normal.
4. It is remarkable that the induced activation (by risk factors) and the induced inhibition (by ASA) of the cell growth persisted in the subcultures.

This behaviour is explained by the assumption that the arterial wall has different cell clones, characterized by different proliferation rates: the faster proliferating clones are activated by risk factors and the slowlier proliferating clones by ASA.
These results are relevant in prevention and therapy of arteriosclerosis.