1185 STUDIES ON FIBRINOPEPTIDE A IN THROMBOTIC DISORDERS

T.Murakoshi*, T.Seya, Y.Oguma, M.Yamauchi, H.Nagata and H.Hasegawa
First Department of Medicine, Hokkaido University School of Medicine, Sapporo, JAPAN

Fibrinopeptide A (FPA) was measured in human plasma by a radioimmunoassay kit (IMCO). In 14 normal individuals this assay revealed a mean FPA level of 1.5 ng/ml. 121 patients were divided into 6 groups by the levels of fibrinogen (FBG) and FDP as follows. A: patients with elevated FBG and elevated FDP levels (FPA: av.9.4 ng/ml). B: patients with elevated FBG and normal FDP levels (FPA: av.3.7 ng/ml). C: patients with normal FBG and elevated FDP levels (FPA: av.3.9 ng/ml). D: patients with normal FBG and normal FDP levels (FPA: av.5.3 ng/ml). E: patients with reduced FBG and elevated FDP levels (FPA: av. 4.1 ng/ml). F: patients with reduced FBG and normal FDP levels (FPA: av.4.8 ng/ml). It is noteworthy that elevated FPA levels are found in group D.
Considering FPA levels from clinical disorders, elevated FPA levels were found in patients suffering from DIC and other thrombotic disorders. In clinical cases, comparing with other indicators which represented hypercoagulable state, FPA levels changed most rapidly, and after injection of heparin in these patients, they were reduced rapidly into the normal range. Additionary, it appears possible to determine the structure of thrombi from the correlation between FPA and β-thromboglobulin. These results suggest that FPA is most sensitive indicator of thrombin action, and provides direct information concerning the effectiveness of therapy in preventing fibrin formation.

1186 STUDIES ON CERTAIN ASPECTS OF THE DYNAMICS OF CHOLESTEROL DEPOSITION IN RABBIT ARTERIAL TISSUE.

S. Mukherjee and C. Mukherjee, Department of Applied Chemistry, University of Calcutta, India

Rabbits were maintained on a 1% cholesterol diet for 30, 60 and 90 days and isotope labeling of aorta and serum was attained by feeding 100 mc of 4 - ^{14}C - cholesterol during the last 4 days before sacrifice of each group. Labeled aorta and low-density lipoproteins (LDL) isolated from sera of the various diet regimens were employed for in vitro study of rates of flux of free and esterified cholesterol by incubating respectively with LDL and aorta of a rabbit maintained for similar periods on above diet but given no radiocholesterol. Influx of free and ester cholesterol into thoracic aorta of hypercholesterolemic rabbit were respectively 6 and 11 times that obtained with tissues isolated from normocholesterolemic animals. With progressive ingestion of hypercholesterolemic diet, a gradual increase in rate of influx with concomitant lowering of efflux rate was noticed for both free and ester cholesterol. Studies on percentage clearance of labeled cholesterol from aorta of hypercholesterolemic rabbits into serum LDL indicate that 25 to 30 percent of radioactivity of free cholesterol is removed by LDL as against 2 to 4.5 percent of ester cholesterol radioactivity. Experiments carried out with partially de-lipidated LDL of sera hypercholesterolemic rabbits reveal that efflux of cholesterol from aorta is markedly increased.

1187 CRITICAL EVALUATION OF BLOOD PLATELET KINETICS IN RAT ARTERIAL THROMBOSIS

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

Blood platelet survival is shortened under several thrombotic conditions. We studied platelet kinetics during arterial thrombosis induced by indwelling intra-aortic cannulas in rats. The alteration of platelet behaviour by mechanical and pharmacological interventions was investigated. In our model platelet consumption is strongly increased, leading to a long-lasting, steady state thrombocytopenia but with a normal platelet turnover. Termination of the thrombotic stimulus readily reduces the increased platelet consumption, leading to restoration of normal platelet counts. Pharmacological interference with treatment of either PGE_1 or PGI_2 decreases platelet consumption and elevates the platelet count, whereas treatment with sulfinpyrazone or Org 3382 does not improve significantly platelet disappearance but partially normalizes the platelet count.
We conclude that differences in platelet disappearance in this rat model are measured more sensitively by the difference between platelet production and -consumption (as expressed by the platelet count) than by platelet consumption alone as measured by disappearance of radiolabelled platelets).