PLENARY II

Platelets

THE ARRANGEMENT OF GLYCOLIPIDS IN THE HUMAN PLATELET PLASMA MEMBRANE. P.K. Schick and K.A. MacDonald. The Medical College of Pennsylvania, Philadelphia, Pennsylvania, U.S.A.

Glycolipids present on the platelet surface were investigated with an external labeling technique. Washed platelets were incubated with galactose oxidase followed by reduction and labeling with ^3H -sodium borohydride. Neither galactose oxidase nor sodium borohydride caused platelet leakage or release of platelet serotonin during the experimental procedure. Glycolipids were extracted, purified, separated by thin-layer chromatography, and quantitated by the fluorometric assay of sphingosine using fluorescamine. Platelet glycolipids represented 0.4% total platelet lipids. The following four platelet neutral glycolipids were identified and expressed as % of total platelet glycolipids: 41% trihexosylceramide, 29% globoside, 26% lactosylceramide, and 4% cerebroside. Only globoside and to a lesser extent trihexosylceramide were heavily labeled by the galactose oxidase technique. The study indicates that globoside and trihexosylceramide are located on the platelet surface. Glycolipids present on the platelet surface conceivably play an important role in platelet physiological activities.

MEMBRANE GLYCOPROTEIN LOSS FROM CIRCULATING PLATELETS: INHIBITION BY DIPYRIDAMOLE AND ASPIRIN. J. N. George and P. C. Lewis. University of Texas Medical School, San Antonio, Texas, U.S.A.

Platelet senescence during circulation has been suggested by evidence for the decreased size and hemostatic effectiveness of older platelets. We previously reported that when platelets are labeled simultaneously with $[^{125}\text{I}]$ -diazodiodosulfanilic acid (I), which reacts with surface exposed plasma membrane glycoproteins, and ^{51}Cr , which reacts with soluble cytoplasmic material, the disappearance of I is more rapid than Cr (J Lab Clin Med, 88:247, 1976). We postulated that platelet surface material may be lost during reversible contact interactions in the process of hemostasis. To test this hypothesis we studied the survival of doubly labeled platelets in 5 rabbits treated with dipyridamole (D) (5 mg/kg IV at 0.5, 3 and 23 hours after infusion of labeled platelets) and aspirin (ASA) (25 mg/kg IV at 0.5 hours after infusion) in comparison with 13 control rabbits (untreated or given the drug diluent fluids in the same regimen). The diluent fluids had no effect. D and ASA had no effect on Cr disappearance but prolonged the disappearance rate of I so that it was equal to Cr. In control rabbits, the $T_{1/2}$ of Cr (33.3 ± 7.9 (SD) hours) was greater than I (19.5 ± 3.6 , $p < 0.001$). In treated rabbits, the $T_{1/2}$ of Cr (36.0 ± 5.3) was not different from that of I (30.8 ± 8.1 , $p > 0.2$). The $T_{1/2}$ of both Cr and I in the treated rabbits were greater than that of I in the control rabbits ($p < 0.001$) but not different from the $T_{1/2}$ of Cr in the control rabbits ($p > 0.5$). We conclude that platelet surface glycoprotein loss is a continuous process in the normal circulation, possibly related to the changes of platelet senescence. The effect of D and ASA suggests that the membrane glycoprotein loss may occur during hemostatic encounters.