ABSENCE OF THE 145000 MOLECULAR WEIGHT, SOLUBLE PLATELET GLYCOPROTEIN - LACK OF PLATELET AGGLUTINATION. N.O.Solum, I.Hagen, M.Peterka and T.Gjemdal. Institute for Thrombosis Research, University of Oslo, Rikshospitalet, Oslo, Norway.

The agglutination of human platelets by bovine factor VIII related protein has been studied in relation to the presence or absence of the 145000 molecular weight, soluble platelet glycoprotein (GPS) using the presence of a granule-located glycoprotein as a positive control. GPS is found in the soluble fraction after subcellular fractionation of platelet homogenates on sucrose density gradients, and is probably identical to the soluble membrane protein glycocalicin. The granule and the soluble glycoproteins separate into two bands on SDS-polyacrylamide gels both in reduced and unreduced samples when the gels contain urea and EDTA and are stained with periodic acid-Schiff reagent (PAS). A concomitant loss, or specific elution, of GPS and loss of agglutination was observed when platelets were stored in Tris-buffered saline, pH 7.4 (TS) at 4°C for 7 days, or when freshly prepared platelets were frozen and thawed when suspended in TS. When suspended in an EDTA-containing buffer during freezing and thawing, the resulting platelet ghosts still contained GPS and still agglutinated. The soluble fraction obtained by subcellular fractionation of giant platelets (Bernard-Soulier) did not show any PAS bands, whereas soluble fraction of these platelets prepared by freezing and thawing in TS clearly showed the presence of the granule protein but not the GPS band. The data indicate an association between GPS and the agglutination phenomenon.

INVITED SYMPOSIUM XIV

Physiological and Clinical Implications of Fibrinogen Biochemistry.

DISORDERS OF FIBRINOGEN AND FACTOR XIII - INTRODUCTION. H. L. Nossel. Department of Medicine College of Physicians and Surgeons, Columbia University, New York, N. Y.

Transformations of the fibrinogen molecule are amongst the best biochemically defined reactions of the hemostatic mechanism. The exact peptide bonds cleaved by thrombin are known and the initial sites of plasmin attack are defined. The gamma chain cross-links produced by Factor XIIIa are characterized, and work is proceeding on the identification of the alpha-chain cross-links.

Physiologically fibrinogen participates in a number of platelet functions including adhesion to collagen and foreign surfaces and ADP-induced aggregation. Polymerizing fibrin aggregates platelets. Fibrinogen not only acts as a substrate for thrombin but is a powerful reversible inhibitor of thrombin activity.

Congenitally abnormal fibrinogen may be associated with a thrombotic tendency, a hemorrhagic tendency or be asymptomatic. Acquired molecular changes occur in thromboembolic disease, intravascular coagulation and possibly in liver disease. Assays for molecular alteration find increasing clinical application. In the present session the following topics will be discussed:

1. Relationship between fibrinogen and platelets
2. Fibrin polymerization - biochemistry
 - physiological and clinical significance
3. Cross-linking
 - biochemistry
 - physiological and clinical significance