Three radioactive peaks (approx. MW 97.000, 120.000 and 150.000) were seen on subsequent SDS polyacrylamide gel electrophoresis of control platelets. Treatment of prelabeled platelets with thrombin did not reveal any changes in the "specific" radioactivity of the three peaks, indicating that no labeled fragments had been split off.

Treatment with thrombin before iodination under conditions where the release reaction occurred, resulted in a drastic reduction in the incorporation of radioactivity. When the release reaction was inhibited by antimycin and 2-deoxyglucose before thrombin treatment and subsequent iodination, very little or no effect could be observed on the incorporation of radioactivity.

Aggregation of these platelet suspensions with bovine factor VIII, and ristocetin plus human ristocetin cofactor indicate that platelets which have undergone the release reaction aggregate poorly with these agents. The membrane conformation necessary for iodination of the three membrane polypeptides and the ability of the platelets to aggregate in the factor VIII-dependant systems thus seem to be altered during thrombin-induced release reaction, possibly because of alterations in the membrane structure.

*D. R. Phillips, C. S. P. Jenkins, D. Meyer, M.-J. Larrieu and E. F. Luscher* (Theodor Kocher Institute, Bern, Switzerland, and Institut de Pathologie Cellulaire, Hôpital de Bicêtre, 94270 Le Kremlin-Bicêtre, France): **Platelet Membrane Glycoproteins and their Possible Relationship to the ADP Mechanism of Platelet Aggregation. (451)**

Platelets isolated from patients with Glanzmann's Thrombasthenia release in the presence of thrombin and other stimuli but fail to respond to ADP. Since the intial interaction between the platelet and ADP is at the membrane surface, it would appear that this surface lacks the necessary receptor for ADP. The surface structure of normal and thrombasthenic platelets was compared using the lactoperoxidase iodination technique. Iodination of normal platelets results in the labelling of four glycoproteins, I, IIa IIb and III, with relative ratios of 1 : 1 : 1 : 3 plus other non-characterised polypeptides. Thrombasthenic platelets similarly treated revealed a drastically altered expression of the glycoproteins on the membrane. The relative ratios (1.5 : 1 : 0.4 : 0.5) revealed the decrease of glycoprotein IIb and the marked reduction of glycoprotein III. Arguments and data will be presented which point to the possibility that glycoprotein IIb is involved in ADP-induced aggregation.

*S. Levy-Toledano, R. Bredoux, F. Rendu, G. Tobelem, L. Degos and J. P. Caen* (Hôpital Saint-Louis, 2, pl. du Dr. Fournier, F 75475 Paris Cedex 10, France): **Specificity of an Acquired Antiplatelet Antibody Occuring in a Polytransfused Thrombasthenic Patient. (452)**

An IgG antibody which developed in a polytransfused thrombasthenic patient reacted in complement fixation with platelets from 350 normal individuals but not with platelets from 8 other thrombasthenic patients. It agglutinates human platelets in PRP but not thrombasthenic platelets. This agglutination increased if the PRP was preincubated at 37° C or if the platelets were isolated before the addition of the antibody. Dog but not rabbit platelets are agglutinated by the patient plasma. Neither adenosine, nor PGE1 inhibit this agglutination which is slightly reduced by acetylsalicilic acid and disappears with EDTA and EGTA (3,8 mM).

Its activity is reduced or abolished after incubation with control platelet membranes but not with those obtained from thrombasthenic or rabbit platelets.

It does not inhibit the ADP-induced shape change of normal platelets, and it prevents all the ADP mediated platelet aggregations but not those induced by bovine factor VIII and ristocetin.

This antibody seemed to be directed against a molecule absent or structurally modified in thrombasthenic platelets which would be involved in platelet aggregation and more especially in ADP mediated platelet aggregation.