ADP AND RETENTION OF PLATELETS IN GLASS BEAD COLUMNS. J. Dale, Institute for Thrombosis Research, Oslo, Norway.

Platelet retention is known to be dependent upon ADP, thought to be derived either from red cells by hemolysis in the glass bead columns or from the platelets themselves. In order to study this, the following experiments were done in blood from 8 healthy subjects and 11 patients with prosthetic heart valves. The whole blood content of adenine nucleotides was measured, the platelet retention in whole blood was determined by Hellem's modified method, the degree of hemolysis provoked by passage of whole blood and EDTA-anticoagulated blood was estimated, and finally the liberation of adenine nucleotides from EDTA-blood was measured. The whole blood content of ADP was similar in the two groups of subjects, as was the degree of hemolysis caused by passage of blood through the columns, while platelet retention was low in blood from the ball-valve patients. The adenine nucleotides were liberated in the same proportions as hemoglobin, and ADP appeared in plasma in mean concentrations of 0.10 μM in both groups of subjects after passage of EDTA-blood through the columns. The results indicate that ADP in amounts necessary to induce platelet retention is derived from red cells. The reduced retention in ball-valve patients in spite of normal ADP-liberation is probably a result of trauma to the platelets inflicted by the prosthetic valve.

HEPARIN NEUTRALIZING ACTIVITY (HNA) IN CLINICAL PRACTICE. A. Hurler Birk-Jensen, M. Moriau, C. Col-De Beys, M. Vanderlinden and R. Masure. Haemostasis and Thrombosis Research Unit, University of Louvain, Belgium.

HNA is measured in several pathological conditions by a biological assay. PFP, platelets and serum are compared.

In PFP, HNA/ml is high in thrombocytosis, in acute DIC and in some thrombotic states; it is normal in most cases of thrombocytopenia. When expressed as units/ 10^9 platelets, PFP's HNA is low in thrombocytopenia from hepatic origin. HNA/ml of PFP decreases during treatment with sulotidil; these last results are related to platelet survival time.

Serum HNA, more than platelet HNA, reflects the antiheparin capacity of the blood and could be used to estimate the minimal amount of heparin required in therapy.

HEPARIN COFACTOR DURING EARLY STAGE OF COAGULATION IN VITRO AND DURING BLEEDING IN VIVO. A. Hurler Birk-Jensen, M. Moriau, C. Col-De Beys, C. Gillet and R. Masure. Haemostasis and Thrombosis Research Unit, University of Louvain, Belgium.

Heparin cofactor activity is measured with the amidolytic method according to Ødegård (1975) during in vitro coagulation and during bleeding.

Heparin cofactor activity decreases differently during coagulation process of PRP or PFP. It is suggested this is due to heparin neutralization by PF4 and/or to thrombin formation. Antithrombin III level is not modified.

During bleeding, the same phenomenon is observed when the amount of heparin in the buffer is low enough. Decrease in heparin cofactor activity is already observed on a sample collected less than 90 seconds after the puncture, before any detectable thrombin formation.

Decrease in heparin cofactor activity during bleeding is studied in pathological conditions. It is more marked in some hypercoagulable states.