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 performed.

1. Blood from 8 healthy subjects and 11 patients with prosthetic heart valves was measured.
2. The whole blood content of adenine nucleotides was measured, and finally the liberation of adenosine nucleotides from EDTA-blood was measured. The whole blood content of ADP was similar in the two groups of subjects, as also was the degree of hemolysis caused by passage of blood through the columns, while platelet retention was low in blood from the Patients. The adenosine 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 full-volume 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. Harlet Nisk-Jensen, N. Horgau, G. Col-de Brey, M. Vanderlindem and R. Masure. Hemostasis 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 sulindac; these last results are related to platelet survival time.

Serum HNA, more than platelet HNA, reflects the antihemia 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. Harlet Nisk-Jensen, N. Horgau, G. Col-de Brey, C. Gillet and R. Masure. Hemostasis and Thrombosis Research Unit, University of Louvain, Belgium.

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

Heparin cofactor activity decreases during coagulation process of PFP or PFP.

It is suggested this is due to heparin neutralization by PFP 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.