THE EFFECT OF ENHANCED FIBRINOLYTIC ACTIVITY ON THE RED CELL MEMBRANE TRANSPORT. S. B. Ulutin, N. B. Emekli and T. U. Yardimici. University of Istanbul, Cerrahpasa School of Medicine, Istanbul, Turkey.

In dogs and in human subjects, using hepatic vein catheterization before and after the activation of the fibrinolytic system, the blood samples were obtained and the red cell amino acid transport was investigated. The time course accumulation of radioactive histidine in isolated red cells was followed together with the measurements of the fibrinolytic activity.

A decrease in the active transport of histidine was observed in the red cells after the stimulation of the fibrinolytic system. Also a correlation between the decrease of active transport and the increase of fibrin-fibrinogen degradation products was seen.

PLATELET ANTITHROMBIN III IN ATHEROSCLEROSIS. A. Z. Aktulga and O. N. Ulutin. University of Istanbul, Cerrahpasa School of Medicine, Istanbul, Turkey.

The presence of antithrombin III in the platelets and its secretion from the platelets was shown. In normals the antithrombin III was 13.71 ± 1.5 μg per 10^9 platelets, but in the cases of atherosclerosis with hypercoagulability this value decreased to 8.43 ± 1.07 μg per 10^9 platelets.

MORPHOLOGIC PLATELET CHANGES IN VITRO AND DURING THROMBUS FORMATION IN THE RAT. R. Wiedemann, K. Breddin, H. Grun and W. Weichert. Department of Angiology, Medical Center, University Frankfurt a.M., Germany.

Using high power Nomarski optics the shape change of thrombocytes can be shown. Platelets appear in their native shape as flat discs, less than 25% show pseudopodes. Depending on the time after blood sampling and on incubation temperature platelets in PRP or citrated blood swell and form tentacles. The addition of ADP to PRP induces the formation of aggregates. Single platelets form large vesicles rupturing and releasing granulated material. The remaining platelet material fuses. Bencyclan affects platelet morphology by inducing a spherical transformation, which is paralleled by the inhibition of platelet adhesiveness, spreading and aggregation. Observations in small mesenteric vessels of the rat show platelets in their native shape under stasis conditions. Vascular lesions are produced with a focused laserbeam (Hadron 513 biolaser). Immediately after the lesion platelets stick to the site of the microburn. Within seconds these platelets swell and form protrusions. After 3 - 10 min the vessel is occluded by a thrombus of platelets, which undergo further swelling. Later the thrombus is partially or completely swept away and the vessel is recanalized. Irreversible fusion of platelets is rarely observed. These morphologic platelet changes differ markedly from those observed during in vitro aggregation. Injection of a new antithrombotic substance (Bay G 6575) diminishes the adhesion of platelets on the vessel lesion. The morphologic changes of single platelets (primary shape change) probably represent basic processes in hemostasis and thrombus formation.