PLATELET FUNCTION CHANGES DURING HEMODIALYSIS. Robert D. Levin, Peter Ivanovich and Hau C. Kwaan. Departments of Medicine, Northwestern University Medical School and Veterans Administration Lakeside Hospital, Chicago, Illinois.

Platelet function during hemodialysis was studied in 27 patients during 62 dialyses on a Cordis-Dow Hollow Fiber Artificial Kidney CDAK-4 with anticoagulation by porcine mucosal heparin. Platelet counts fell from $2.08 \pm 0.52 \times 10^9/l$ (Mean $\pm$ S.D.) prior to dialysis to $1.74 \pm 0.49 \times 10^9/l$ after 1/2 hour of dialysis ($P < 0.01$), and remained at depressed levels throughout the dialysis period. Platelet adhesiveness in arterial blood did not significantly vary during dialysis. By aggregometry, the fraction of aggregable platelets decreased from $69 \pm 13\%$ prior to dialysis to $51 \pm 15\%$ after 1/2 hour ($P < 0.001$) and afterwards remained depressed. The ratio of aggregation after $1.2 \mu M$ ADP to that after $4.7 \mu M$ ADP elevated from $87 \pm 15\%$ prior to dialysis to $93 \pm 7\%$ after 1/2 hour ($P < 0.05$). Platelets from the venous return limb of the hemodialyzer were poorly aggregable to $50 \mu M$ ADP or $10 \mu M$ epinephrine.

The findings are compatible with an enhanced reactivity of platelets and the concomitant adherence of aggregable platelets upon the dialyzer fibers, leaving an increased fraction of poorly functioning platelets in the circulation. The enhanced reactivity is apparently induced by heparin, increased levels of ionized calcium, and other factors.

INTERACTION OF HUMAN PLATELETS WITH SUBENDOTHELIUM: IMPLICATIONS FOR MEASUREMENT OF DEFECTS IN PLATELET FUNCTION. V.T. Turitto, H.J. Weiss and D.S. Cohen. Roosevelt Hospital, Department of Medicine, New York, New York, USA.

An in vitro perfusion technique introduced by Baumgartner has been used to investigate the interaction of human blood platelets with subendothelium of rabbit aortas. Platelet contact, adhesion (spreading) and the formation of microthrombi were measured directly by use of a morphometric technique for a variety of exposure times (1 to 40 min) and a physiological range of flow rates (shear rates varying from 50 to 830 sec^{-1}). A theory accounting for platelet transport through the blood and the platelet reactivity at the vessel surface indicated platelet transport to be the controlling influence on platelet attachment. Under low shear conditions platelet attachment is unaltered by moderate changes in platelet reactivity. The results suggest that high shear rates (greater than 800 sec^{-1}) are necessary for a sensitive measurement of defects in platelet attachment and that experimental devices which employ low shear conditions are limited in measuring such defects.

ADENOSINE DIPHOSPHATE INDUCED AGGREGATION AND RELEASE REACTION IN HEPARINIZED PLATELET RICH PLASMA. S. Heptinstall and G. P. Mulley. University Department of Medicine, General Hospital, Nottingham, England.

Preparations of heparinized platelet rich plasma (PRP) from 54 different volunteers were examined to determine the extent of platelet aggregation and release reaction both in the absence and presence of citrate. Platelet aggregation was studied in fresh untreated samples of PRP using a range of concentrations of ADP. To study release reaction platelets in a portion of each preparation were labelled with 3H -5-hydroxytryptamine. Released radioactivity was measured after stirring with ADP or with ADP and citrate.

Even in the absence of citrate release was considerable in 26 of the preparations. There was a good correlation between extent of aggregation and extent of release reaction. When second phase aggregation occurred release was extensive, when release was low or absent the higher concentrations of ADP were required to bring about "irreversible" aggregation. Whenever citrate was present release reaction was enhanced. Enhanced release reaction was also observed in PRP in which the bulk of plasma calcium had been exchanged for sodium by ion exchange chromatography.

It is concluded that ADP induced release reaction can occur in heparinized PRP but that it is enhanced by reducing the concentration of extracellular ionised calcium.