PLATELET FUNCTION CHANGES DURING HEMODIALYSIS. Robert D. Levin, Peter Ivanovich and Hsu C. Hsu.
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Platelet function during hemodialysis was studied in 27 patients during 62 dialyses on a Corr-Dis-Dow Hollow Fiber Artificial Kidney CDAK-6 with anticoagulation by porcine mucosal heparin.

Platelet aggregation was significantly depressed during dialysis (P<0.01). The platelet count was reduced from 1,08 ± 0.37 x 10^9/L (Mean ± S.D.) prior to dialysis to 1.14 ± 0.49 x 10^9/L after 6 hours of dialysis (P<0.01). Platelet aggregation in citrated blood did not significantly vary during dialysis. Platelet aggregation was significantly lower in citrated blood (P<0.05) compared to blood without anticoagulant.

The findings are compatible with an enhanced reactivity of platelets and the concomitant adherence of aggregate 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.

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An in vitro perfusion technique developed by Rümpf and Partner has been used to investigate the interaction of human blood platelets with subendothelium of rabbit aorta. Platelet attachment, adherence, aggregation, and the formation of microthrombi were measured directly by a quantitative photographic technique in vivo (40 to 90 min) and a physiological time course of shear 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 that platelet transport to the subendothelial surface could be the controlling influence on platelet attachment. Under low shear conditions, platelet attachment is mediated by aggregating 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.

ADRENOGR INHIBITED AGGREGATION AND RELEASE REACTION IN HYPERVISSERED PLATELET RICH PLASMA. S. Bentinse and H. P. Nollw.
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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 Na-2-iodothymidine. 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 "irreversibility" 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 an exchange chelatomorph.

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