Time

13.15 **0993** INCREASED LEVELS OF LOW AFFINITY PLATELET FACTOR 4 IN PLASMA AND URINE OF PATIENTS WITH CHRONIC RENAL FAILURE.

S. Niewiarowski*, J. Guzzo, A.K. Rao, I. Berman and P. James, Temple University Health Sciences Center, Philadelphia, Pa.

Low affinity platelet factor 4 ($LA\text{-}PF_4$) is a specific platelet secretory protein immunologically related to ß-thromboglobulin and to platelet basic protein that stimulates cell growth. The level of $LA\text{-}PF_4$ antigen was determined by specific radioimmunoassay in platelet poor plasma (PPP) and platelet rich plasma (PRP) of 17 normal individuals (NI), 9 patients on hemodialysis (HD) and 18 patients with chronic renal failure (CRF) who were not dialysed. Eight patients with glomerular filtration rate (GFR) 5-20 ml/min were included to group 1 and 10 patients with GFR 30-50 ml/min were included to group 2. Levels of $LA\text{-}PF_4$ in ng/ml PPP were 31.9 ± 2.8 in NI, 291.8 ± 26.3 in HD, 149.4 ± 45.2 in group 1 of CRF and 103.7 ± 11.1 in group 2 of CRF. Differences between patients and NI were significant at $p < 0.05$. After 3 hours of hemodialysis $LA\text{-}PF_4$ increased to 505.0 ± 92.3 ($P < 0.05$), however, there was no changes in PPP level across the artifical kidney at one hour. The levels of $LA\text{-}PF_4$ in PRP of patients with CRF and HD did not differ from NI. Mean $LA\text{-}PF_4$ excretion in urine (ng/day/100mg creatinine) was 57.7 ± 6.4 (range 33.3-75.7) in 6 NI with GFR >60 ml/min and 1461.5 ± 674.6 (range 105.3-7041.6) in 10 CRF patients. In conclusion, elevation of $LA\text{-}PF_4$ in PPP of patients with CRF and its increased urinary excretion suggest stimulation of platelet secretion and/or decreased metabolism of $LA\text{-}PF_4$ in the kidney.

13.30 **0994** EVIDENCE FOR DEPENDANCE OF BETA_THROMBOGLOBULIN LEVELS ON RENAL FUNCTION

D.Deppermann, K.Andrassy*, H.Seelig, E.Ritz, D.Post, Medizinische Universitätsklinik Heidelberg. (FRG)

Beta-Thromboglobulin (TG) is a thrombocytic protein which is released into the circulation upon lysis of thrombocytes. Its serum concentration is thought to be a sensitive indicator of thrombocyte consumption. Although a relation of TG platelet consumption could be observed by us in patients with normal renal function, no such relation was found in pat. with renal failure (RF). Therefore we examined whether TG, similar to beta-2-microglobulin (BMG), behaves like a tubular protein. - Patients and methods - 90 pat. with RF were examined. None of the pat. had diseases with known platelet consumption or medication which interferes with platelets. TG and BMG were measured by RIA. - Results - There was a highly significant correlation between TG, serum creat. (r=0,78) and Ccr (r=0,73) and BMG. The normal range ($\bar{x}$+SD) of TG was 33,9 + 9,7 ng/ml and values consistantly above 50 ng/ml were observed at Ccr < 50 ml/1,73 m². TG was found in tubular proteinuria. - Conclusion - TG is a tubular protein the concentration of which rises in patients with renal failure. This finding invalidates the use of TG for detection of thrombosis in patients with impaired renal function.

13.45 **0995** β-THROMBOGLOBULIN IN PATIENTS WITH CHRONIC RENAL FAILURE: EFFECT OF HEMODIALYSIS

D. Green*, S. Santhanam, F. A. Krumlovsky, and F. del Greco, Department of Medicine, Northwestern University Medical School, Chicago, Illinois, U.S.A.

β-thromboglobulin (β-TG), a protein located in the α-granules of platelets, is released into the plasma when platelets are disrupted. Since plasma β-TG is cleared by the kidney, we measured β-TG levels in normal subjects and and in patients with chronic renal failure, using a radioimmunoassay kit (Amersham Corp.). In 24 controls, mean values were 27 + 12 (S.D.) ng ml^{-1} and in 24 patients, 123 + 41 ng ml^{-1}, $p<0.001$. Because hemodialysis may induce platelet damage, we examined β-TG levels in patients before and after dialysis. Although platelet counts were unchanged, plasma β-TG levels rose in all but 2 patients, with an average increase of 30 ng ml^{-1}. That the increase in β-TG was due to platelet disruption was confirmed by (1) no change in β-TG in 3 patients having peritoneal dialysis, and (2) studies of a patient with radiation nephritis and severe thrombocytopenia (18,000 per cu mm) secondary to chemotherapy. β-TG was 12 ng ml^{-1} and did not increase after hemodialysis. We conclude that plasma β-TG is significantly elevated in patients with chronic renal failure, and that measurement of this protein provides a sensitive indicator of platelet disruption by hemodialysis.