1071 CHANGES IN FIBRINMONOMER AND FIBRIN STRUCTURE IN PATIENTS WITH RENAL FAILURE

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Procedure: 54 patients subdivided in 3 groups were investigated: 1) 10 patients with acute renal failure. 2) 36 patients with chronic renal failure (serum creatinin >5mg%). 3) 8 patients before and one hour after hemodialysis.

Fibrinmonomer and fibrin structure were studied by SDS-polyacrylamidgelelectrophoresis according to Weber and Osborne. In addition factor XIII activity was determined immunologically according to the method of Bohn

and Haupt.

Results: 1) Patients with acute renal failure showed a defective structure of fibrin with loss of polymerisation of the α -chains but normal γ -dimers. In these patients factor XIII activity was lower than 60%. 2) In 21 out of 36 patients with chronic renal failure, fibrin polymerisation was incomplete though no factor XIII deficiency could be determined. 3) The changes in fibrin polymerisation could be demonstrated before as well as after hemodialysis.

Conclusions: 1) In acute renal failure, factor XIII deficiency induces an incomplete α -chain polymerisation of fibrin. 2) In chronic renal failure, the deficient α -chain polymerisation may be caused by an inhibitor which is not dialysable.

1072 INVESTIGATION ON CARBOHYDRATE RESIDUES INVOLVED IN THE BLOOD COAGULATION MECHANISM USING LECTINS

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Decline in the activity of blood coagulation factors of citrated pooled human plasma(in%) after incubation with lectins (final concentration lmg/ml,15 min, at room temperature):

| after incubation wit | h lec | tins (II | nai co | oncent. | ration | 1mg/m | 1,15 | min. at room temperature/: |
|----------------------------------|-------|----------|--------|---------|--------|-------|------|---|
| Lectin: | F-V | F-VIII | F-IX | F-XI | F-XII | F-VII | F-X | Lectin specificity |
| Concanavalin A(ConA) | 70% | 20% | 20% | 30% | 30% | - | - | D-glucose, D-mannose |
| Lens Culinaris | - | 40% | - | 40% | 40% | - | 10 | ", |
| Castor Bean 120,000 daltons | 4 | 10% | - | 10% | 10% | - | - | D-galactose, D-galactosamine |
| Castor Bean 120,000+60,000 d. | - | 40% | - | 40% | 35% | - | - | D-galactose, D-galactosamine |
| Wheat Germ (WGL) | 55% | 60% | 30% | 20% | 85% | - | 15 | tri-N-Acetyl-D-glucosamine (Ac-D-gluc) |

The inhibitory influence of lectins could be prevented by adding specific carbohydrates. Taking the specificity of lectins into account, one could assume that carbohydrate residue Ac-D-gluc. has a prominent influence on clotting activity of FXII and VIII.F-V-activity also seems to be strongly connected with glucosyl or mannosyl residues. Also PTT, as global test for the intrinsic clotting system was remarkably prolonged(6x)with WGL(not shown on the table).It is striking that FVII activity is not inhibited by the lectins examined. This raises the question, whether carboh.resid.take part in extrinsic clotting at all.

1073 HEMOSTASIS ACTIVATING FACTOR AND VON WILLEBRAND'S SYNDROME

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Small amounts of subcutaneous tissue extracts containing a "hemostasis activating factor" (HaF) induce pseudopode formation and sphering in freshly drawn citrate blood and they markedly enhance platelet retention in glass bead columns. If HaF (50 $\mu l/ml)$ was added to freshly drawn citrate blood together with a fresh cryoprecipitate (50 $\mu l/ml)$ from normal PPP, retention was further increased. Cryoprecipitate alone (50 $\mu l/ml)$ did not induce morphologic platelet changes or enhance platelet retention. In 15 patients with v.Willebrand syndrome (VWS) spontaneous morphologic platelet changes after blood sampling did not differ from normal controls. But two different types of VWS patients could be distinguished in the retention test: In the first group low retention was increased to normal after the addition of HaF. In a second group a very small or missing increase of platelet retention was found with HaF alone; but retention was "normalised" by adding small amounts of cryoprecipitate together with HaF.VWF may be essential for the rapid stimulation of platelets by HaF in primary hemostasis.