ANTITHROMBIN III ACTIVITY, MEASURED WITH A CHROMOGENIC SUBSTRATE, IN PATIENTS WITH HEPATIC CIRRHOSIS, WITH PROSTHETIC HEART VALVES AND DURING PARENTERAL ADMINISTRATION OF MEDROXYPROGESTERONE ACETATE AS A CONTRACEPTIVE AGENT. G. Baele, E. Matthys, G. De Cock, M. Thiery and F. Barbier. University of Ghent Departments of Medicine and Obstetrics, Ghent, Belgium.

Antithrombin III (AT III) activity was assayed on heat defibrinated plasma using a synthetic chromogenic substrate, benzoyl-Phe-Val-Arg-p. nitroanilide. AT III activity in 30 normal subjects averaged 95 ± 18 (± 1 SD). As AT III is synthesized in the liver, we measured its activity in 72 samples from 44 patients with hepatic cirrhosis. The mean activity (49 ± 23) was significantly lower than in the control group. AT III activity was also measured in 32 patients with prosthetic heart valves receiving sodium warfarin therapy. The mean activity in this group (90 ± 23) fell in the normal range. It also did not differ significantly from the mean activity (95 ± 29) in a similar (matched for sex and age) group of 32 subjects, also treated with sodium warfarin but for other reasons than bearing a prosthetic heart valve. As low AT III levels have been reported in women using a combined oral contraceptive, we also measured AT III in 19 women receiving trimonthly injections of 150 mg of medroxyprogesterone acetate as contraception. AT III levels in this group of women (102 ± 23) were found to be normal. So the oestrogen in the combined contraceptive may be responsible for the reported fall in serum AT III activity.

COMPLEX FORMATION OF FIBRIN MONOMERS STUDIED BY GEL FILTRATION AND POLYACRYLAMIDE GEL ELECTROPHORESIS. R. Hafer, M. Baumgärtner, R. v.Hugo and H. Graeff. I. Frauenklinik der Universität, München, Germany.

Estimation and characterization of thrombin mediated products of fibrinogen (soluble fibrin monomer complexes=SFMC) can be achieved by gel filtration and PAA-gel electrophoresis. In order to gain further information about complex formation in vitro tests were performed. Fibrin monomers were prepared from fibrin dissolved in 3 M KBr after action of thrombin (des-AB-fibrin) and reptilase (des-A-fibrin) on fibrinogen and added to plasma. In a certain range of fibrin concentration (up to 3 % for des-AB-fibrin and up to 15 % for des-A-fibrin) soluble complexes are formed with fibrinogen in a 1:1 molar ratio. Further increase of fibrin percentage results in a partial precipitation of complexes. Additionally, a marked increase of SFMC (up to 60 %) is observed with des-A-fibrin. This latter reaction indicates complex formation with a higher ratio of fibrinogen involved. An influence of temperature on complex forming could be observed. Gel filtration at 37°C reveals a shift in the elution profile, indicating dissociation of high molecular weight complexes (5 million daltons) to lower molecular structures. The dissociation behaviour of SFMC from plasma samples of patients with hypercoagulability is similar. However, crosslinked fibrinoligomers showed no temperature dependant dissociation behaviour. It is concluded that SFMC exist during the state of hypercoagulability in vivo predominantly as a dimeric fibrin-fibrinogen complex.

SPONTANEOUS PLATELET AGGREGATION AND SULFINPYRAZONE INFLUENCE IN PATIENTS WITH DISEASES OF THE KIDNEYS AND HYPERTENSION. W.Kirch, H.Köhler, I.Med. Univ.Klinik, Mainz, W.Germany

In some diseases, particularly in diseases of the vessels, there is a spontaneous aggregation of platelets. So it was of interest, whether in patients with diseases of the kidneys and essential hypertension there is a frequent spontaneous aggregation as well. In addition, the effect of the antithrombotic substance sulfinpyrazone on platelet aggregation, on plasmatoc coagulation and on clinical parameters, for instance on proteinuria, was to be investigated. In 47 patients with diseases of the kidneys or essential hypertension spontaneous platelet aggregation, thrombin time, partial thromboplastin time, fibrinogen and factor VIII were studied. Patients with spontaneous platelet aggregation were given 800 mg sulfinpyrazone daily for 1 to 6 months. In 15 of 16 patients with chronic glomerulonephritis (serum creatinine 0,7-2,0 mg%) and 13 of 14 patients with a chronic pyelonephritis (serum creatinine 0,8-1,7 mg%) spontaneous platelet aggregation was found as well. So in almost all patients with chronic glomerulonephritis and pyelonephritis a spontaneous aggregation was seen. The frequency of spontaneous platelet aggregation in patients with essential hypertension is likely to depend on the stage of hypertension. Spontaneous platelet aggregation did not return to normal in any of our patients on sulfinpyrazone and the substance had no influence on proteinuria, haematuria and leukocyturia in patients with diseases of the kidneys.