dissecting aneurysm, plasma fibrinogen exhibited marked resistance to normal α-chain proteolysis by Arvin. The patient showed a moderate haemostatic defect acquired from the time of aortic dissection: bleeding was stopped with EACA. Tests were consistent with the presence in plasma of high and low molecular weight FDP, thus deficient haemostasis could be explained by defective fibrin polymerization and X-linking.

The α-chain resistance to Arvin was a coincidental finding. Does this behaviour represent a defect and, if so, is it congenital, or acquired representing a previously unencountered phenomenon associated with DIC? The data show Arvin to be a valuable agent for the rapid isolation and investigation of fibrinogen in normal and pathological plasmas.

A. R. Williams (Depts. of Anatomy and Medical Biophysics, University of Manchester, Manchester, England): **Intravascular Acoustic Microstreaming as an Initiator of Mural Thrombi in Vivo. (375)**

Acoustic microstreaming (a rapidly rotating small-scale eddy of blood) is developed inside blood vessels when a discrete portion of the vessel wall is driven to oscillate at an ultrasonic frequency. The wall of the intact vessel was set into motion by the external application of a steel probe (a 115 μm radius wire oscillating transversely at 20 kH_z or the 52.5 μm diameter tip of an exponentially tapered velocity transformer oscillating in its longitudinal mode at 80 kH_z). The intravascular microstreaming field subjects the vascular endothelium immediately adjacent to the probe tip to large hydrodynamic shear stresses which renders that surface thrombogenic. Simultaneously, platelets are traumatized by the same microstreaming field and are bomarded against the vessel wall where they appear to participate in the formation of mural thrombi. Small blood vessels are completely occluded by platelet plugs which may be stabilized by the formation of fibrin.

This technique produces discrete localized intravascular thrombi by the non-invasive application of hydrodynamic shear stress in the absence of any other thermal, chemical or electrical trauma. It therefore provides a novel *in vivo* model to investigate the effect of biological and pharmacological variables on thrombus growth and development, and the nature of the repair processes.

Jean M. Thomson, L. Poller, G. Green and I. W. Dymock (Departments of Haematology and Medicine, Withington Hospital, University Hospital of South Manchester, Manchester M20 8LR, Great Britain): **The Value of a Prothrombin Complex Concentrate in the Coagulation Defect of Liver Disease. (376)**

A prothrombin complex concentrate rich in factor VII has been used in the management of the clotting defect in thirteen patients with liver disease. Adequate correction of coagulation was achieved immediately after the infusion in all cases. Within four hours there was some deterioration and by twenty-four hours the results approximated to pre-infusion values. Liver biopsy procedures were performed without haemorrhagic complication in the immediate post-infusion period. There was no evidence of induced intravascular coagulation.

Other prothrombin complex concentrates have proved disappointing both in their failure to correct the clotting defect and their production of DIC. From our results the use of this factor VII rich concentrate appears to be the treatment of choice in patients with liver disease requiring temporary correction of the coagulation defect.

R. Zimmermann, U. Fauser, K. Andrassy and H. Bleiler (Med. Univ. Klinik, W.-Germany, 69 Heidelberg, Bergheimerstr. 58): **Hemorrhagic Diathesis in Amanita Phalloides Poisoning. (377)**

In human amanita phalloides poisoning the severity of the disease as well as the different hospitalisation time after toxin uptake allow no clear interpretation of blood clotting analysis. Therefore the coagulation changes were examined in a standardized animal model in relation to the given dose. Due to the main responsibility of amanitin for the

clinical picture Gamma-Methyl-Amanitin (0,05–0,08 mg/kg) was administered intravenously in 20 beagle dogs. Depending on the given dose three different groups could be observed: *Group I* (amanitin > 0,07 mg/kg): Death in hypoglycemic shock 20–40 hours after poisoning. No manifest hemorrhagic diathesis. Histologically no fibrin clots. Coagulation analysis: DIC (presence of fibrinmonomers) with predominant fibrinolysis, increased level of FDP (> 120 ug/ml). Within 24 hours decrease of coagulation factors, Fibrinogen, F. II, V, VIII, XIII (< 10%) and platelets (< 30,000). *Group II* (amanitin 0,05–0,07 mg/kg): *A.* 70% of the animals survived with signs of hemorrhagic diathesis. Histologically no fibrin clots. Coagulation analysis: Decrease of clotting factors and of platelets to about 50% with nadir after 48 h. Maximal fibrinolysis after 36 h. Normalisation after 120 h. *B.* Protracted decrease of the clotting factors and platelets to 7000 after 60 h. Marked fibrinolysis with death in hemorrhagic shock. Histologically fibrin clots. Comment: In relation to the dose a different reaction of the clotting system can be stated: I. In cases of massive poisoning an endotoxinshock-like picture could be shown. Predominant fibrinolysis. II. With lower dose of amanitin a moderate consumption coagulopathy with moderate, secondary fibrinolysis (A), or (B) predominant consumption coagulopathy with marked fibrinolysis and death in hemorrhagic shock.

W. A. Andes, R. B. Lindberg, D. D. McEuen and J. P. Baron (Institute of Surgical Research, Fort Sam Houston, San Antonio, Texas 78234, U.S.A.): **Inhibition of Fibrinolysis in Burn Wound Infection. (378)**

Infection is the leading cause of death following thermal injury. Various indices of fibrinolysis have been found to be disturbed in such patients. This study reports findings in a lethal *Pseudomonas aeruginosa* burn wound infection in rats, uninfected but burned controls, and preliminary studies in their human counterparts. Sequential studies revealed that as the infected animals neared their demise their plasminogen levels (caseinolytic) fell ($p < 0.01$), serum antiplasmin (method of von Kaulla) but not antiactivator activity rose ($p < 0.01$), euglobulin lysis times were very prolonged, fibrin-related antigen titers (staphylococcal clumping) were lower and fibrinogen concentrations were slightly higher than in the uninfected-burned controls. Alpha$_2$-acute phase globulins but not α_1-macroglobulins (by. K. Ganrot) were 18 times higher in the infected than in uninfected rats. The bacteria did not induce antiplasmin activity when cultured in serum. 3,5-di-iodosalicylic acid Na abolished the antiplasmin activity. Burned patients had no unusual antiplasmin activity on the day of burning but developed high levels coincident with lowered plasminogens.

It is possible that such changes in plasmin activity have importance in infections in burns, or other conditions, through impairment of fibrinolysis and microcirculatory flow.

K. Korsan-Bengtsen, B. Hallgren and A.-C. Teger-Nilsson (Department of Internal Medicine II, Department of Clinical Chemistry, Sahlgren's Hospital, and Astra Nutrition AB, Göteborg, Sweden): **Effects of Dietary α-Tocopherol and Polyunsaturated Fats on the Fatty Acid Composition of Platelet Phospholipids and on Blood Coagulation. (379)**

The study group was 40 male post myocardial infarction patients 47–57 years old. All the participants were investigated two times with two weeks interval after which they were randomly divided into four groups with 10 subjects in each. Group 1 was given alpha-tocopherol 300 mg/day, group 2 was given alphatocopherol 300 mg/day and a diet containing extra polyunsaturated fats, group 3 was given extra polyunsaturated fats but no extra alpha-tocopherol and group 4 served as a control group – thus continued their ordinary diet. After three months all participants were again investigated twice with two weeks interval.

On the values from all 40 subjects before the start of the dietary regimens linear regression analyses showed that there was a significant correlation between the content of the fatty acid 18 : 0 in the serin cephalin fraction and recalcification time in platelet