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

rich plasma (RPRP), and a negative correlation between 20 : 4 and RPRP. There was also a correlation between the ratio 18 : 0/20 : 4 and RPRP and a negative correlation between 18 : 0/20 : 4 and platelet factor 3 activity in plasma.

In group 2 there was a significant decrease in 18 : 0 and an increase in 20 : 4 in the serin cephalin fraction from platelets after the diet period compared to preexperimental values. Russel's viper venom clotting time (RVV) decreased significantly in group 1. There was a significant correlation between the decrease in RVV and the increase in plasma alpha-tocopherol.

L. E. McCoy, D. T. H. Liu and V. Y. Wu (Wayne State University, School of Medicine, 540 E. Canfield, Detroit, Michigan, 48201, U.S.A.): **Thromboplastin and Platelet Factor 3: Protein and Phospholipid Components Required for Procoagulant Activity. (380)**

Characteristics of bovine brain and lung thromboplastins and platelet factor 3 are compared, emphasizing protein and phospholipid moieties involved in procoagulant activity. Isolated tissue and platelet procoagulant lipoproteins were separated into lipid and protein fractions by repeated ethanolic extraction. Lipid composition and quantitation was ascertained by thin layer chromatography. The protein-free lipid mixture functioned as a partial thromboplastin. Protein fractions were purified by deoxycholate solubilization, gel filtration, and chromatography on diethylaminoethyl cellulose. Comparative biochemical properties were determined by gel filtration, polyacrylamide gel electrophoresis, sedimentation velocity and by carbohydrate and amino acid composition. Purified protein components were devoid of enzymatic activity. They were also free of procoagulant activity on prothrombin or autoprothrombin III (Factor X) unless they were combined with one or more phosphatides of ethanolamine (PE), choline (PC), serine (PS), or inositol (PI). The proteins of thromboplastin appear to have homologous biochemical characteristics and phospholipid requirements for expression of procoagulant activity, while differences in platelet factor 3 activity relate to differences in both protein and phospholipid requirements.

(Supported by USPHS, NIH Grant HL 14142).

L.-O. Andersson and H. Sandberg (AB KABI, Stockholm, Sweden): **Thromboplastic Effects of Human Plasma Lipoproteins. (381)**

Lipoprotein fractions from human plasma was prepared by ultracentrifugal flotation. Additions of those fractions to plasma containing various amounts of platelets showed that in platelet-poor and platelet-free plasma there was a clear clot-promoting effect of the additions. In platelet-rich plasma this effect was negligible. Measurements on the thromboplastine and Stypven clotting times showed that the high density lipoprotein fraction affected both the prothrombin and the Factor X activation steps whereas the low density lipoproteins only influenced the prothrombin activation step. Addition of antibodies against high density lipoproteins to platelet-free plasma caused a prolongation of the thromboplastin time.

The relation between lipoprotein structure, phospholipid content and thromboplastic effects is dicussed.

T. W. Barrowcliffe, J. M. C. Gutteridge, J. Stocks and T. L. Dormandy (Whittington Hospital, London N19. National Institute for Biological Standards and Control, London NW3): **Effect of Lipid Oxidation Products on Blood Coagulation. (382)**

Pure fatty acids in buffered aqueous suspension were allowed to autoxidise for up to 4 days. At intervals, lipid-free extracts were tested for effect on coagulation. 1-day extracts accelerated recalcification and RVV times, but products after 2–4 days oxidation showed progressive inhibition in these tests, and prolonged the P. T. and P. T. T. Addition of extracts to phospholipid altered its activity, and preparations of phospholipid extracted with an without antioxidant had different activity. In the absence of phospholipid,