After passage of the current topical application of ADP or ATP for three minutes results in the formation of white platelet thrombi.

Microprojection of the arterial segment performed on a series of LDR's (Light Depending Resistances) allows registration of potential variations due to white thrombus formation.

The magnitude of these potentials is linearly related to the thrombus mass.

Computerization of these data is possible after A–D conversion of the signals and appropriate programmation.

E. Nemesánszky, T. Riesz and J. M. Jákó (Ist. Departement of Medicine Postgraduate Medical School, 1135 Budapest, Hungary): **Disturbance of Haemostasis in Rats Caused by Partial Liver-Necrosis.** **(368)**

A partial liver-necrosis was induced in albino rats by ligation of the left hepatic lobe and the changes of the haemostatic system were investigated in the course of sterile autolytic process caused. In the test animals, changes characteristic to diffuse intravascular coagulation (DIC) developed after surgery. This process proved to be reversible and undervent spontaneous remission within 48 h. The alterations observed in the blood clotting screening tests rapidly normalized and the fibrin precipitate, demonstrable in the kidney by histological method were only detectable until the second day following surgery. Even after the normalization of haemostasis, fibrinogen products (FDP) were detectable by immunological methods for several days.

I. Mahn, C. Reuter, H. Merkel and G. Müller-Berghaus (Zentrum für Innere Medizin, Klinikstr. 36, 63 Gießen, Germany): **The Elimination of Labelled Fibrinogen in Galactosamine-Induced Experimental Hepatitis in Rabbits.** **(369)**

The intravenous injection of D-galactosamine-HCl into animals induces a liver disease resembling virus hepatitis in man in its histological and clinico-phatological features. In a previous study disseminated intravascular coagulation was demonstrated by tracing fibrin-rich microclots in the renal glomerular capillaries, especially if the fibrinolytic system was inhibited by EACA (Thromb. Res. *1*, 473, 1972). In order to differentiate between disturbance of synthesis and disseminated intravascular coagulation, investigations with ^{125}I-fibrinogen were performed in rabbits treated with D-galactosamine (1 g/kg) and EACA (0.5 g/kg × hr). In rabbits infused with galactosamine and EACA the elimination of ^{125}I-fibrinogen was increased in comparison to the control animals treated with EACA or isotonic saline only. If heparin (750 u/kg × hr) was infused additionally to galactosamine and EACA, the accelerated decay of labelled fibrinogen was prevented. The occurrence of ^{125}I-activity in organs was pronounced in animals exhibiting microclot formation. These experiments indicate that due to a diminished synthesis of coagulation factors in this model of hepatitis disseminated intravascular coagulation may contribute to the coagulation defect.

(Supported by the Deutsche Forschungsgemeinschaft, Bad Godesberg, Germany.)

D. R. B. Jones, J. D. Cash, Rosemary Owens, R. G. Dalton and C. V. Ruckley (Department of Clinical Surgery and Regional Blood Transfusion Service, Edinburgh): **Serum Fibrin/Fibrinogen Degradation Products (FDP) after Experimental Pulmonary Embolism in Dogs.** **(370)**

In previous clinical studies we reported elevated serum FDP values after pulmonary embolism. We detected high values in only 50% of patients and the elevation was transitory. In order to determine the exact time and magnitude of the FDP elevation, and whether the increase was consistent, experimental pulmonary emboli were produced in dogs.

Under pentobarbitone anaesthesia emboli of autologous clot were injected via the femoral vein. FDP, platelets and fibrinogen were estimated on frequent samples taken via a jugular vein catheter. The FDP were measured by the Tanned Red Cell Haemagglutination Inhibition Immunoassay with reagents specific for dog fibrinogen.

In 5 control animals FDP values rose gradually during 8–12 hours of anaesthesia. In 11 dogs receiving 3 ml/kg of clot the mean FDP rose from 9 ug/ml at rest to 142 ug/ml (Range 9–1600 ug/ml). The peak of FDP was reached in 30–60 minutes and remained significantly above control values for up to 7 hours. These data support our view thaf FDP measurement is valuable in the diagnosis of venous thrombo-embolic disease.

F. De Clerck, J. Vermijlen, G. Hornstra and R. Reneman (Medical Faculty Maastricht, Unilever Research the Netherlands, Medical Fac. Leuven, Belgium): **Modification of Venous Stasis Thrombosis by Platelet Active Drugs and by Heparin. (371)**

In rats, stasis thrombosis of a renal vein was produced by the occlusion of a vascular segment after the induction of systemic hypercoagulability by intravenous injection of ellagic acid. The development of thrombosis depended upon the dose of ellagic acid, the duration of circulation before occlusion of the vascular segment and the duration of stasis. Morphologically the thrombus consisted mainly of red blood cells with occasional foci of platelet aggregates. Prostaglandin E1, at doses which reduced platelet retention by glass beads (6 μg/min/300 g I.V.) only slightly reduced thrombus size. The platelet release inhibitor indomethacin (50 mg/kg I.V.) had no effect on thrombus size. Heparin (10 and 20 u/kg I.V.) produced a dose-related reduction of venous thrombosis. The platelet-active drug VK 774 at 10 to 50 mg/kg I.V. also reduced the thrombus size. Binding to ellagic acid or induction of coagulation changes are proposed as a possible mechanism of action. It is concluded that the participation of platelet function in the development of venous stasis thrombosis is small.

F. K. Beller, J. Ribhegge and E. H. Schmidt (Department of Obstetrics and Gynecology, D-44 Muenster, Westring 11, W.-Germany): **Influence of Steroids on Endotoxin Induced Disseminated Intravascular Coagulation. (372)**

We have previously demonstrated that stilbestrol augments disseminated intravascular coagulation (Theiss, W., Beller, F. K., Amer. J. Obstetr. Gynec., *115*, 795, 1973). This was demonstrated by using a standardized endotoxin infusion model in rats. The experiments were repeated systematically on castrated females. Different groups of about 20 rats got daily injections of ethinyl oestradiol, progesteron or a combination of both over a period of three weeks. It was shown that ethinyl-oestradiol augmented DIC in a dose range of 30 μg, whereas pretreatment with oestrogen in smaller doses or with progesteron or both was not so effective. In contrast to these findings pretreatment with corticosteroids in a large dose range prevented glomerular fibrin deposits completely.

E. H. Schmidt, L. Fleitmann, F. Graubner and F. K. Beller (Department of Obstetrics and Gynecology, D-44 Muenster, Westring 11, W.-Germany): **Influence of Volume Expanders on Endotoxin Induced Glomerular Fibrin Deposition. (373)**

The continous infusion of endotoxin in virgin rats is followed by glomerular fibrin deposition. The same effect could be reached by a single injection of endotoxin followed by an infusion of normal saline. (Theiss, W. and Beller, F. K., J. Lab. Clin. Med. *3*, 431–442, 1973). This experimental model was used to compare a variety of fluid solutions. Low and high molecular dextrane did not result in glomerular fibrin deposition but depressed the plasma fibrinogen level strongly. The infusion of gelatine produced glomerular fibrin deposition, which was significantly less pronounced than after infusion of normal saline. It is suggested that dextrane prevents glomerular deposition by interference with plasma fibrinogen.

R. S. Lane, O. H. Baugh and P. T. Flute (St. George's Hospital, Blackshaw Road, Tooting, London S. W. 17 G. B.): **Fibrinogen α-Chain Resistance to Arvin (Ancrod) in DIC. (374)**

In high concentrations, Arvin (Ancrod) splits fibrinogen α-chains into fragments of 39,000 and 31,000 molecular weight. In a patient with mild chronic DIC, secondary to a