Monday, July 13, 1981

**Oral Presentations** 

# Thrombosis, Clinical — I

08:00-09:30 h

# Thrombosis, Clinical – II

09:45-11:00 h

Cinema 1

### 0035 08:15 h

STUDIES ON COAGULATION PHYSIOLOGY IN PERIOPERATIVE THROMBO-EMBOLISM PROPHYLAXIS IN SURGERY. K. Koppenhagen, A. Häring, H. Zühlke, A. Wiechmann, M. Matthes, J. Hardleck, R. Rieser, E. Tank, K. Breddin. Dept. of Radiology, Nuclear Medicine and Surgery Klinikum Steglitz, Free University of Berlin. Dept. of Angiology, University of Frankfurt.

In a prospective and randomized double-blind study in 630 general surgery patients with different thromboembolism prophylaxis (130 patients with 5000 and 122 with 2500 I.U. of heparin, 128 with a fixed combination of 2500 I.U. of heparin and 0.5 mg dihydroergotamine ((2500-heparin-DHE)), 124 with 0.5 mg DHE and 126 with a placebo, 3 x daily) the influence of the medication on blood coagulation was examined by repeated pre-and post-operative determinations of blood-and coagulation parameters (activated partial thromboplastin time, heparin-plasma level, AT III, thromboplastin time, fibrinogen, thrombin time, thrombocyte aggregation test, β-thromboglobulin, Hb, erythrocytes, hematocrit, thrombocytes and differential blood picture). The frequency of postoperative thromboses and pulmonary embolisms was investigated by means of the radiofibrinogen test and lung scintigraphy. The results show that the lowest thromboembolism rate occurs in that patient collective in which there is in addition to the heparin application a simultaneous hemostatic effect of DHE. Side effects of the prophylaxis and influences on blood coagulation are dependent on the amount of heparin administered both in the single-drug low-dose treatment and in the combination therapy. The lowest rate of side effects was found in the group receiving 3x2500 I.U. of heparin and in the combination group 2500-heparin-DHE, but the combination treatment additionally affords the same thromboembolism protection as the routinely applied 3x5000 I.E. of low dose heparin.

### 0034

08:00 h

RELATIONSHIP BENWEEN FIBRINGEN PROTEOLYSIS, PLATELET RELEASE AND FIBRIN DEPOSITION FOLLOWING TOTAL HIP REPLACE-MENT. D.A. Lane, T.G. Allen-Mersh, H. Ireland, S. Wolff, S. Jennings and F. Grant. Charing Cross Hospital and Medical School, London W6 8RF, Great Britain.

It has been postulated that fibrin deposition is controlled by the relative competition of thrombin and plasmin for fibrin I. We have tested this hypothesis by measuring haemostatic activation products in the plasma of patients undergoing hip replacement. Thrombin sensitive fra, ment fibrinopeptide A (FpA), plasmin sensitive fragment B $\beta$ 1-42 and  $\beta$  thromboglobulin ( $\beta$ TG) have been measured before and after operation (n=26). The incidence of venous thrombosis was assessed by phlebography and I125 fibrinogen scanning. The mean preoperative FpA, BA1-42 and ATG concentrations were 1.80, 4.22 and 0.99 pmol/ml respectively. The mean FpA and ATG levels rose to 3.07 and 1.56 pmol/ml respectively on the sixth day after operation but the mean Ba1-42 level rose more rapidly to a maximum of 10.9 pmol/ml on the fourth day after operation. In those patients who developed a thrombosis (n=9), the The those patients who developed a thrombosis (l=9), the mean FpA level was higher on the first day after operation (2.87 pmol/ml) than in those patients (n=17) in whom thrombosis did not occur (1.66 pmol/ml). Conversly, the mean B $\beta$ 1-42 level was lower (4.76 pmol/ml) in those who developed a thrombus than in those without thrombus (6.79 pmol/ml). When the postoperative day 1 results were expressed as a ratio of FpA/B(1-42, the corresponding mean values of this ratio for the two groups of patients were 0.74 and 0.37. These results demonstrate that (a) there is an activation of the coagulation and fibrinolytic systems, and of platelets following hip replacement surgery (b) thrombus formation following major surgery is characterised by an increased action of thrombin on fibrinogen and a reduced action of plasmin on either fibrinogen or fibrin I (c) assays for FpA and BA1-42 may be useful for the detection of developing thrombi.

#### 0036

08:30 h

POSTOPERATIVE HYPERCOAGULABILITY, DETECTION AND MEASURE-MENT USING A MODIFIED RECALCIFICATION TIME SYSTEM, EFFECTS OF LOW DOSE HEPARIN AND TYPES OF SURGERY. P.B. Lundquist and J. Swedenborg, Depts of Surgery and Expr. Surgery, Karolinska Hospital, 104 01 Stockholm, Sweden.

The purpose of the study was to demonstrate postoperative hypercoagulability and its possible prevention by low dose heparin (LDH). Healthy volunteers (with LDH), patients undergoing cholecystectomy (with & without LDH) and patients undergoing arterial reconstructive surgery with synthetic grafts (with LDH), were tested. All were tested 1, 3 & 5 hrs after LDH.

Overall coagulability was determined by using platelet free plasma and a modified recalcification time system with a nefelometer to detect first fibrin formation. Recalcification times were measured before  $(T_0)$  and after  $(T_A)$  plasma activation against glass, in vitro. Heparin Tevels were determined with the Factor Xa inhibition test using a chromogenic substrate (Coatest R, Kabi).

chromogenic substrate (Coatest\*, Kabi).  $T_0$  and  $T_A$  were prolonged after LDH in healthy volunteers. Cholecystectomy caused shortening of  $T_0$  and  $T_A$ . This could be prevented by LDH, raising  $T_0$  to level recorded after LDH in normals. Patients receiving synthetic arrerial grafts showed no prolongation of  $T_0$  and  $T_A$  after surgery with LDH, but rather a shortening. These patients showed hypercoagulability in spite of LDH.

It is concluded that postoperative hypercoagulability can be traced with the presented method and counteracted by LDH in patients undergoing cholecystectomy but not in patients receiving synthetic arterial grafts. All patients on LDH had similar heparin levels, determined with the Factor Xa inhibition test. Vascular surgery with synthetic grafting and Cholecystectomy seem to induce two different kinds of hypercoagulability, where the former is thought to be induced by the foreign surface. Subcutaneous heparin therapy (LDH) results in poor inhibition of surface induced coagulation, in vivo.