

Poster
Board
P4-1270700 PLASMIN ACTIVITY, PLASMINOGEN LEVELS AND CONCENTRATIONS AND
FUNCTIONAL ACTIVITIES OF ANTIPLASMINS DURING SEPTICEMIA

M.J.Gallimore,*A.O.Aasen, K.Lyngaas, M.Larsbraaten, E.Amundsen and N.Smith
Eriksen. Institute for Surgical Research, Rikshospitalet, and Department
of Anesthesia, Akershus Central Hospital, Oslo, Norway.

Recent studies have indicated that the fibrinolytic system becomes activated during endotoxemia in animals and septicemia in man. In the present study we have used chromogenic peptide substrate assays to measure plasmin activity and "immediate" and "total" antiplasmin activities in plasma samples from normal subjects and patients with septicemia. Plasma concentrations of plasminogen (Pg), α_2 antiplasmin (α_2 AP), α_2 -macroglobulin (α_2 M) and α_1 -antitrypsin (α_1 AT) were also determined. In samples from patients with fatal sepsis low levels of Pg, α_2 AP and α_2 M were found together with reduced "immediate" and "total" antiplasmin activities. Plasmin activity and α_1 AT levels were higher than normal. Three patients who recovered following septicemia had higher Pg and α_2 AP concentrations than those who died and functional antiplasmin activities were within the normal ranges. Our results confirm that the fibrinolytic system becomes activated during septicemia and in sepsis of fatal outcome a marked consumption of Pg and α_2 AP occurs. In our limited number of recovery patients the fibrinolytic system appeared to have been less activated than in those who died.

Aggregation and Other Reactions of Platelets

Level 5 - Terrace (Red Side)

Free Poster Session 11.30 - 12.45

P5-053 0701 A STUDY OF INTRAVASCULAR PLATELET AGGREGATION IN THE RAT

G. G. Duncan and G. M. Smith, School of Pharmacy, Robert Gordon's Institute of
Technology, Schoolhill, Aberdeen AB9 1FR

Intravascular platelet aggregation can be studied by measuring the fall in the circulating platelet count induced by aggregating agents in anaesthetized animals. The Technicon Auto-counter was modified and connected via a double cannula to an anaesthetized rat to give a continuous count of the number of circulating platelets (1). Adenosine diphosphate (ADP), Collagen, Arachidonic acid (AA) and 5-Hydroxytryptamine (5-HT) were given at 15 minute intervals over a period of 2-3 hours. Aspirin (10 mg/Kg IV) and Indomethacin (1-8 mg/Kg IV) partially inhibited collagen-induced aggregation and Indomethacin (2 mg/Kg IV) completely inhibited AA-induced aggregation. Adenosine (0.25 mg/min) inhibited the ADP-induced aggregation but did not inhibit aggregation produced by collagen or the residual response to collagen that remains after the addition of indomethacin.

Reproducible responses to ADP and collagen were obtained but responses to AA and 5-HT were not reliable. Collagen-induced aggregation is thought to be mediated by the liberation of ADP, 5-HT and the formation of prostaglandin (PG) endoperoxides and thromboxane A₂. This study has shown that collagen-induced aggregation is reduced by inhibition of PG synthesis but the involvement of ADP or 5-HT could not be shown.

Reference (1) G. M. Smith and F. Freuler. *Bibl. Anat.* No.12, 229-234, (1973).