## Action of Heparin and other Anticoagulants

## Poster Board P6-092

Level 6 - Green Side (Westminster Buffet)

and adjacent Terrace

Free Poster Session 11.30 - 12.45

THROMBELASTOGRAPHIC EFFECTS OF DEXTRAN 70 ON HUMAN BLOOD: H. Kwaan, S. Wright DL. Zuckerman\*, J. A. Caprini and P. J. Vagher, Department of Surger Evanston Hoppital and Department of Medicine, Northwestern Univ. Med. School and V.A. Lakesida Medical Center, Chicago, Ill. U.S.A. 0758 THROMBELASTOGRAPHIC EFFECTS OF DEXTRAN 70 ON HUMAN BLOOD: H. Kwaan, S. Wrigh Medical Center, Chicago, Ill. U.S.A.

The effects of dextran was studied by thrombelastography (TEG) because it can graph cally depict fibrin formation and dissolution and platelet-fibrin interaction during close ting. Dextran 70 in concentrations of 1-8% were added to samples of native whole blood (WB), recalcified whole blood (RWB), platelet-rich plasma (PRP) and platelet-poor plasma (PPP) using buffered saline as control. Dextran produced an 18% reduction in clot stiff ness (MA) with native and celite activated WB. The same native WB showed a 23% prolong  $\mathfrak{g}$  tion of clotting (R) and a 24% decrease in the rate of clot formation  $(\alpha)$ , while the celite activated WB R and  $\alpha$  were reduced by 17% and 19% respectively. However, using R we found only a 10% reduction in MA as a result of dextran addition, suggesting that cite trating and recalcification diminished the dextran effects. With PRP (platelets >560,0 $oldsymbol{\sigma}$ 0 dextran did not show any significant reduction in the measured parameters, while PPP ga $\frac{g}{dt}$ the greatest response with a 36% decrease in MA. Results were similar if PRP was obtain ed from subjects who had taken aspirin. This indicates the main action of dextran at the content of the fibrin network rather than on platelets or the platelet-fibrin interaction though this effect is best seen when platelet concentrations are low. Clot lysts by urgo kinase or streptokinase was accelerated in the presence of dextran but was reduced by creased platelet concentrations. These results suggest a direct effect of dextran on brin formation and may explain the antithrombotic effect of dextran in that defective fibrin formation occurs.

O759 EFFECT OF DEXTRAN ON THE POSTTRAUMATIC FIBRINOLYSIS INHIBITION

G. Carlin, G. Karlström, J. Modig and T. Saldeen, Institut of Forensic Medicine and the Department of Anaesthesiology and Orthopaedic Surgery, University of Uppsala, Sweden Increased fibrinolysis inhibition activity (FIA) in the blood is seen in patients with Market and the property of the ed from subjects who had taken aspirin. This indicates the main action of dextran ef

## 0759 EFFECT OF DEXTRAN ON THE POSTTRAUMATIC FIBRINOLYSIS INHIBITION P6-093

Increased fibrinolysis inhibition activity (FIA) in the blood is seen in patients with postoperative thromboembolic complications and has been suggested to be of importance in the pathogenesis of these conditions. Infusion of dextran (500 ml 6 %) to patients 3 days after total hip replacement surgery, when FIA was maximal, strongly decreased FIA of plasma measured by a clot lysis assay. Plasma antiplasmin (chromogenic substrate assay) and immunologically determined levels of the primary fibrinolysis inhibitor (PFI,  $\alpha_2$ -antiplasmin),  $lpha_2$ -macroglobulin,  $lpha_1$ -antitrypsin and plasminogen were not changed after the infusion of dextran. The effect of dextran on plasminogen activation by urokinase and fibrin degradation was studied in clotted plasma. The presence of dextran accelerated both the fibrinolysis and the uptake of labelled plasmin on fibrin. This effect of del ran was not seen in experiments where the clot was made of purified fibrinogen (containing plasminogen) instead of plasma. The FIA-decreasing effect of dextran is also dependent on the presence of physiological plasmin inhibitors and fibrin. The results indicate that the activation of plasminogen is enhanced by dextran provided that fibrin and inhibitors are present.