FIBRIN DEGRADATION PRODUCTS AND VASCULAR PERMEABILITY. Bengt Gerdin, Herman Högstorp, Olle Lindquist, Tom Saldeen and Erik Svensjö, Dept. of Forens. Med., Univ. of Uppsala and Dept. of Exp. Med., Pharmacia AB, Uppsala, Sweden.

Increased vascular permeability plays an important role in the pathogenesis of the delayed microembolism syndrome. Fibrin degradation products (FDP) may play a role for this permeability disturbance. Fractions of lymph from the cannulated right lymphatic duct in dogs with induced microembolism syndrome and lysate from fibrin clots obtained by gel chromatography were used. The effect on vascular permeability was determined in the hamster cheek pouch and in the dorsal skin of the rat. Increased permeability was determined by leakage of fluorescein labelled dextran in the first model and by use of isotope labelled albumin in the second model. Lymph from the lymphatic duct and fractions of lysate from fibrin clots caused an increased vascular permeability of the same character in both models, the effect being partly due to high molecular weight products and partly due to low molecular weight products. The effect of high molecular weight products may possibly be due to their continous cleavage releasing low molecular weight vasoactive FDP. The effect of FDP on vascular permeability was enhanced by pretreatment with the β-adrenergic inhibitor propranolol and inhibited by the β_2-adrenergic stimulator terbutaline. Bredykinin and PGE_1 both increased macromolecular leakage in the hamster cheek pouch. This increase was also counteracted by terbutaline. The FDP effect on permeability might be due to contraction of the endothelial cells.

WRIGHT-SCHULTE LECTURE

HEPARIN AND VENOUS THROMBO-EMBOLISM. V.V.Kakkar, Thrombosis Research Unit, King's College Hospital Medical School, London.

Venous thromboembolism represents a serious hazard in patients confined to bed in hospital. Though the search for an effective method of prophylaxis has been going on for nearly the last 90 years, a method which is effective in the total elimination of this condition has yet to be developed. One promising approach is the use of low-dose heparin given subcutaneously.

In this review: (a) the rationale for low-dose heparin will be considered in the context of present concepts of pathogenesis of venous thrombosis; (b) factors which govern the kinetics of clearance of heparin from the circulation will be compared following its intravenous and subcutaneous administration; (c) the recently published studies, where the efficacy of this form of prophylaxis against deep vein thrombosis and pulmonary embolism has been assessed, will be analysed, and (d) further possible developments in heparin prophylaxis including the use of semi-synthetic heparin analogue will be discussed.