appeared within two hours after infusion, but whole blood clotting times were still shortened after 12 hours in two experiments. Control studies using cryoprecipitate were satisfactory. Plasmas of three of six hemophilic dogs showed weak inhibition of the small active factor VIII fraction in vitro. The inhibitory effect was present in these hemophilic plasmas after heating to 56° C for 1 hour. The rapid disappearance of activity after transfusion is not completely explained by this inhibitory effect, as rapid in vivo disappearance was also observed where no inhibition was detectable. It appears that use of small active factor VIII does not offer a practical means for intravenous, subcutaneous, or intramuscular administration.

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V. Noordhoek Hegt (Gaubius Institute, Health Research Organization TNO, Herenstraat 5d, Leiden, The Netherlands): **Endothelial and Smooth Muscle Cells as Antagonists in Vascular Fibrinoysis.** **(327)**

Endothelial plasminogen activator activity in different types of human blood vessels obtained from fifty necropsies and thirty-five biopsies was detected and localized by means of plasminogen-rich fibrin slides. Great differences in endothelial activator activity were found along and across (vasa vasorum) the wall of the human vascular system.

The same blood vessels were simultaneously investigated by a modified fibrin slide technique using plasminogen-free fibrin slides covered by plasmin to detect and localize inhibition of fibrinolysis in the vascular wall. The great variation in plasmin inhibition in different vessels revealed by this "fibrin slide sandwich technique" appeared to be closely associated with the localization and number of smooth muscle cells present in the walls of the vascular system. Strong plasmin inhibition was generally found at sites which showed no activator activity with the regular fibrin slide technique, while areas with a high endothelial fibrinolytic activity mostly revealed no inhibitory capacity.

These results indicate that much of the variation in endothelial fibrinolytic activity on fibrin slides is due to inhibitory effects from the surrounding smooth muscle cells rather than to variability in the plasminogen activator content of the endothelium itself.

L. Donner and D. Šafránková (University Hospital, Prague, U nemocnice 2, Czechoslovakia): **The Plasminogen Activator Content of the Arterial Wall in Different Arteries.** **(328)**

Six hundred and sixty specimens of thoracic, abdominal aortas, cerebral, carotid, vertebral, pulmonary, coronary, kidney and mesenteric arteries from 110 fresh cadavers were examined for their plasminogen activator content by means of the histochemical method of Todd. The plasminogen activator in pulmonary arteries, kidney and splenic arteries obtained during surgery was estimated. Differences between the fibrinolytic activities, generally found in the adventitia, are given.

Intact arterial endothelium, in contrast to the venous endothelium, is only slightly or not at all active. Endothelium of thoracic aortas detached by scraping and repeatedly washed was tested on fibrin plates with streptokinase. Under these conditions arterial endothelium showed fibrinolytic activity suggesting that endothelium contains pro-activator. The fibrinolytic activity of arteries adventitia with atheromatous plaques examined by the histochemical method was higher than in normal arteries.

R. C. Franz, N. Hugo and C. R. Jansen (The University of Pretoria and The Atomic Energy Board. Pretoria, Republic of South Africa): **The Histochemical Assay of Plasminogen Activator in the Venous Wall – An Inter-Ethnic Study.** **(329)**

Despite intensive research in the field of blood coagulation and fibrinolysis facilitated by several studies emanating from this continent, the results still afford no satisfactory explanation for the well established observation of the relative rarity of clinically manifest venous thrombo-embolic disease in the Black African.