ON THE NATURE OF FIBRINOLYTIC ACTIVITY OF TISSUES. L. Donner, P. Klener, L. Vodáková. University Charles School of Medicine, Prague, Czechoslovakia.

Plasminogen activator of the vessel wall of thoracic, abdominal aortas, coronary, renal and carotid arteries was examined by means of the Todd's histochemical method.

Activator was removed from sections by treatment for one hour with kalium thiocyanate. After saline wash possible proactivator was converted with streptokinase or urokinase solution and reacted again with the histochemical method. Sections treated with thiocyanate showed no or very low fibrinolytic activity. After streptokinase or urokinase treatment a local fibrinolysis appeared again. It is suggested that thiocyanate despite its power to remove activator is unable to extract proactivator from tissue. No differences were found in different types of arteries examined neither in atherosclerotic or normal arteries.

INHIBITORY EFFECT OF APROTININ ON THE ACTIVATION PROCESS OF CONTACT FACTORS AND PLATELET AGGREGATION. I. Yamaguchi, Y. Yano, T. Kitahara, M. Yatabe, M. Ukita, M. Fujimaki and K. Fukutake. Tokyo Medical College, Tokyo, Japan.

It has been reported by several investigators that aprotinin demonstrates inhibitory action on the process of intrinsic blood thromboplastin generation. In this paper the authors will present the facts that on the hydrolytic action of plasma kallikrein, and on the activation process of plasma prekallikrein as well, the inhibitory action of aprotinin is indicated by the use of chromozym PK assay and the latter process might be caused by the inhibition of factor XII activated with kaolin, and as to platelet aggregation induced by thrombin, aprotinin also plays the inhibitory effect which is more sensitive than the antithrombin activity of aprotinin measured by thrombin clotting time. However, the inhibitory effect of aprotinin on platelets might be caused not only by antithrombic action of this enzyme inhibitor, but also other influence of the inhibitor on release mechanism of the platelets, because epinephrine induced platelet aggregation can be disturbed by the addition of this inhibitor. In summary it might be suggested in this study that the clinical use of aprotinin preparation might be one way for anticoagulant therapy on thrombotic diseases associated with intrinsic hypercoagulability of plasma factors and platelets.

FIBRINOLYTIC ACTIVITY OF GEL FILTERED PLATELET. M. Watada, M. Nakagawa, A. Nishizawa, and H. Ijichi. Kyoto Prefectural University of Medicine, Kyoto, Japan.

Too many factors in the plasma has made it complicated to investigate the functions of platelets, and gel filtration of the platelet rich plasma was proved to be the beneficial method to separate platelets undamaged from plasma protein. Avoiding the influences of the surrounding plasma factors, this gel filtered platelets (GFP) were considered to be applicable for the various platelet research. Using this GFP, the fibrinolytic activities were measured by means of the plasminogen free fibrin plates and of the caseinolytic techniques on the separated platelets and disrupted platelets. The inhibitors like alpha-1 antitrypsin or alpha-2 macroglobulin were also assayed. The lytic area on the fibrin plate was observed when GFP was placed while platelet rich plasma did not lyse, however the lytic area was increased in diameter when GFP was placed with plasmin, comparing with plasmin or GFP alone. In case of the ultrasonically disrupted GFP, it showed the greater lytic area. Caseinolytic activity of GFP supported also the above mentioned results. These observation proved that the platelet has fibrinolytic activity which seems to be masked by inhibitors in the plasma circumstance.