Pandolfi's modification of the "fibrinolytic autograph" method of Todd was selected to assess the histochemical localization and the degree of activator activity in the venous wall of the upper and lower limbs in a White control group (18) and in a group of 20 Black patients.

Both ethnic groups showed greater fibrinolytic activity in the veins of the upper limb - a difference which appeared to satisfy the test of actuarial cross examination (P < 0.00001). Of particular interest was the significantly greater fibrinolytic activity in the lower limb of the African patients as compared with the lower limb veins of the White group (P < 0.05).

The increased fibrinolytic activity in the lower limb veins in Black Africans may be an additional anti-thrombogenic component in the haemostatic profile of this particular ethnic group.


It has been shown that phenformin plus ethyloestrenol causes a significant increase in the fibrinolytic activity of the vessel wall in most patients with recurrent idiopathic venous thrombosis and with an initially low fibrinolytic activity in the vein walls. At the same time a marked decrease in the frequency of thrombotic episodes occurred (Nilson et al. 1974). Due to side effects of phenformin attempts have been made to administrate ethyloestrenol alone to patients with an abnormally low plasminogen activator content of the vessel wall and/or a decreased capacity to release plasminogen activator from the vein walls on venous occlusion of the arms. Ethyloestrenol in a dose of 8 mg/d has been given to 34 patients for 3 to 6 months and to 6 patients for 12 months. Ethyloestrenol caused a significant increase in the fibrinolytic activity in 30 of the 34 patients treated for 3–6 months and in 5 of those 6 treated for 12 months. Ethyloestrenol in a dose of 4 mg/d had no effect.


A method for purification of pig heart tissue activator is described. The method is based on affinity adsorption on fibrin and on hydrophobic interaction chromatography. Acetone dry powder from pig heart tissue was extracted with potassium acetate buffer and fractionated with ammonium sulfate essentially as described by Bachmann et al. (Biochemistry 3, 1578, 1964). A fraction obtained between 14 and 36% saturation with ammonium sulfate was dissolved in phosphate buffer pH 7.0 and adsorbed with fibrin prepared from fibrinogen and thrombin free from plasminogen (about 0.7 g fibrin was used for a preparation prepared from 1 kg heart tissue). The fibrin was collected after adsorption for 2 hours and eluted with 2 M KSCN at pH 7. Practically all activity was precipitated by isoelectric precipitation at pH 4.2. Finally the activator was subjected to hydrophobic interaction chromatography on phenylethyl-Sepharose. The activity was eluted with a KSCN-gradient. The specific activity of the active fraction was about 23,000 Ploug units/mg. The over all yield was about 25%. One strong and three weak fractions were obtained on SDS-polyacrylamide gel electrophoresis. In pure systems tissue activator activates plasminogen at a very low rate. In presence of fibrin the rate of activation increases markedly. Even fibrinogen effects the activation but to a much smaller extent.

D. Ogston, B. Bennett and M. Mackie (Department of Medicine, University of Aberdeen, Aberdeen, Scotland): Studies on Circulating Plasminogen Activator. (382)

A plasminogen activator was prepared in crude form from postmortem occlusion plasma by gel filtration of the plasma on Sephadex G-200 in 0.005 M phosphate buffer (pH 7.5) containing 0.15 M NaCl followed by further gel filtration of the fractions with maximal activator activity on Sephadex G-200 in 0.005 M phosphate buffer containing 1 M NaCl. The preparations were free of plasminogen, α₁-antitrypsin, α₁-macroglobulin, Cl inactivator