
Under fibrinolytic therapy, the levels of fibrinogen and of fibrin(ogen) degradation products (FDP) grossly influence most of the laboratory tests usually performed for clinical control of fibrinolytic treatment. Although these interferences are basically known, there is a lack of some true clinical conditions. Therefore, fibrinogen determination according to Claus (1957), thrombin time, and thromboplastin time were used for routine monitoring of 29 patients undergoing therapy with streptokinase for up to 6 days, and the results of these tests were correlated with true clottable fibrinogen as determined according to Ratnoff and Menz (1961) and with FDP as determined according to Horsley et al. (1966).

Fibrinogen determination by the Clauss method yields poor results; for immediate clinical control, however, they were sufficient, when values above 80 mg/dl were obtained (r=0.60 when compared to actual fibrinogen concentration). Values below 80 mg/dl were completely unreliable and more closely reflected FDP (r=0.62) than actual fibrinogen levels (r<0.20). The Clauss assay is entirely insufficient, when exact fibrinogen determinations are required as for rheological or pharmacological considerations. Quite in contrast, fibrinogen levels measured according to Ratnoff and Menz were independent of FDP levels.

After bi-logarithmic transformation, the thrombin time closely correlated to the FDP level (r=0.56); it thus is a valuable test for quickly estimating FDP. Since FDP also greatly influence thromboplastin time determinations (r=0.75), they must duly be considered, when overlapping oral anticoagulation is planned towards the end of fibrinolytic therapy.

PLATELET FUNCTION, ULTRASTRUCTURE AND MANAGEMENT IN GLANDERMAN'S THROMBOANGIATHIA, J.M. Lusher, W.L. Parnaby, J. Pollock, and A.J. Waryk. Wayne State University School of Medicine, Detroit, Michigan and University of Mississippi Medical Center, Jackson, Mississippi, U.S.A.

This exhibit will demonstrate laboratory abnormalities found in longitudinal studies on six patients with Glanderman’s thrombopathy. All have had mucocutaneous bleeding (predominantly epistaxis) since infancy. In addition to prolonged bleeding times, absent clot retraction and little or no platelet retention in a glass bead column, all have defective platelet factor 3 release with KCl and their platelets do not aggregate with ADP, epinephrine or collagen. The platelets of all six do aggregate with ristocetin, however. Transmission electron microscopy (TEM) of thin sectioned platelets revealed normal ultrastructure. However, in the TEM platelet function test (Thromb.Research., Suppl. 42:321-344, 1970) there was difficulty in capturing the thrombocytopenic platelets even on the forewax surface. These platelets which did adhere to the former exhibited normal percentages of dendritic and spread forms although pseudopod detail was unusual. Inspection by scanning electron microscopy (SEM) of surfaces of platelets responding to contact activation and captured on companion forewax overlays revealed surface membrane features consistent with inhibition of membrane activation. Platelets were swollen with convoluted membranes while pseudopod outgrowth from discrete regions was not prominent. Suppression of membrane activation was also noted by SEM of platelets obtained by cyt centrifugation of buffy coat preparations. Upon exposure to collagen platelet adhesion and morphologic features of aggregation were abnormal under our test conditions. In addition to local measures, recommended management of severe bleeding episodes is infusion of single donor platelet concentrates, obtained from an HLA matched donor by platelethpheresis.

CHANGES IN EUGLOBULIN LYSIS FOLLOWING INTERMITTENT PNEUMATIC Calf COMPRESSION, Mabel M. Stevenson, Paul R. Rohr, Ann G. Davison, Edward F. Byers, Gordon Hopkins, and Thomas J. Parnay, West Virginia University, Morgantown, WV.

Intermittent pneumatic compression of the calf has proven beneficial as prophylaxis for calf vein thrombosis. To define a possible role for the fibrinolytic system, two studies were performed.

Two groups of normal male volunteers aged 18-42 years were rested for one half hour; a control blood sample was drawn and one arm or both legs were pumped for one half hour using a pneumatic pump inflated intermittently to 50mm Hg (90 sec on, 90 sec off) filling time 60 sec.). A second blood sample was obtained. In group one (arm pumped; antecubital vein sample, same side) the euglobulin lysis time decreased 2½ (19) to 147 min, (p<0.05). In group two (both legs pumped) simultaneous sample from femoral and antecubital veins there was a 25% reduction in euglobulin lysis (230 to 172 min, (p<0.01) in femoral vein blood and an 18% decrease in antecubital vein blood (229 to 186 min, (p<0.04). As expected the effect was most pronounced in the local venous effluent and less noticeable in the systemic circulation due to dilution.

The possibility that mechanical compression of the calf veins produces chemical changes that might exert a beneficial effect in preventing venous calf thrombosis is consistent with the data.