LABORATORY CONTROL OF FIBRINOLYTIC THERAPY. W. Theiss, A. Wirtzfeld, E. Sauer, L. Lutitsky, A. Kriessmann. I. Medizinische Klinik der Technischen Universität München, Munich, Germany.

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 information on their influence under true clinical conditions. Therefore, fibrinogen determination according to Clauss (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 Menzie (1961) and with FDP as determined according to Merskey et al. (1966).

Fibrinogen determination by the Clauss method yielded 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 ($r=0.63$) 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 Menzie were independent of FDP levels.

After bilogarithmic transformation, the thrombin time closely correlated to the FDP level ($r=0.86$); it thus is a valuable test for quickly estimating FDP. Since FDP also greatly influence thromboplastin time determinations ($r=0.79$), they must duly be considered, when overlapping oral anticoagulation is planned towards the end of fibrinolytic therapy.

PLATELET FUNCTION, ULTRASTRUCTURE AND MANAGEMENT IN GLANZMANN'S THROMBASTHENIA. J.M.Lusher, M.I.Barnhart, J.Pullen and A.I.Warrier. 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 Glanzmann's thrombasthenia. All have had mucous membrane 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 six have defective platelet factor 3 release with Kaolin 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.Diath.Haemorrh., Suppl. 42:321-344, 1970) there was difficulty in capturing the thrombasthenic platelets even on the formvar surface. Those platelets which did adhere to the formvar 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 formvar coverslips 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 cyto-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 plateletpheresis.

CHANGES IN EUGLOBULIN LYSIS FOLLOWING INTERMITTENT PNEUMATIC CALF COMPRESSION. Mabel M. Stevenson, Paul R. Rohr, Ann G. Davidson, Edward F. Byars, Gordon Hopkins, and Thomas J. Tarnay. 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 pumped for one half hour using a pneumatic boot 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 23% (191 to 147 min., $n=13$, $p=.05$). 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., $n=16$, $p=.01$) in femoral vein blood and an 18% decrease in antecubital vein blood (229 to 188 min., $n=16$, $p=.04$). 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.