THROMBOTIC MANIFESTATIONS OF ESSENTIAL THROMBOCYTHEMIA. Shafkat Hussain, Joel Schwartz, Sa[illegible]or Friedman and Segundina Chua, Coney Island Hospital, Brooklyn, New York.

The course of essential thrombocythemia (ET) has been observed in ten patients, aged 46-83. Eight have been followed for 1 to 8.5 years. In contrast to previous recorded experience with ET, arterial thrombosis was much more common than hemorrhage, with peripheral arterial or cerebrovascular disease dominating the clinical presentation. Six patients had incipient gangrene of toes, and three patients developed occlusion of tibial and larger arteries. Hemorrhage was seen only in two patients in surgical wounds. Absent or markedly decreased epinephrine-induced platelet aggregation was found in all patients studied at initial diagnosis. Epinephrine-induced aggregation improved with platelet count lowering and was impaired again with increase in platelet count. Collagen-induced aggregation was normal in all but one and ADP-induced aggregation was variable. Leukocyte alkaline phosphatase, serum vitamin $B_{12}$ and unbound $B_{12}$ binding capacity determinations were not of differential diagnostic significance. A prolonged remission was easily achieved with busulfan in all patients except one who died of a cerebrovascular thrombosis. The six patients with incipient gangrene showed marked improvement during therapy. Relapses in five patients after 2-87 months responded well to another course of busulfan therapy. No patient has shown evolution to another myeloproliferative disorder. Essential ET should be considered in the differential diagnosis of occlusive arterial disease even in the presence of atherosclerosis.

FACTOR VIII RESPONSE TO VENOUS CONGESTION IN PATIENTS WITH VON WILLEBRAND'S DISEASE. M.MORFINI,G.LONGO,P.ROSSI FERRINI and A.SERI.Haematology Department, Florence,Italy.

Venous congestion(v.c.) is known to increase plasma levels of antihemophilic factor activity(VIII AHF) and fibrinolytic activity in man. In 20 normal subjects and 15 patients with VonWillebrand's disease(VWD),before and after v.c. on forearm the VIII AHF and related antigen(VIII AGN),the euglobulin lysis time,the haematocrit and platelet's count were performed.Ethanol-precipitate (v/v 3% at -3°C) is prepared from each sample.Two-dimensional immunoelectrophoresis and gel filtration through siliconized glass column(K15/30 Pharmacia) packed with Sepharose 6B, was performed by applyng ethanol-precipitate.
In normal subjects a significant increase of VIII AHF has been observed after v.c.(F test $p<0.001$) and of VIII AGN(F test $p<0.05$).In VWD patients only VIII AHF increase was found(F test $p<0.01$) but in patients with very low VIII AHF level,the increase did not occur.The anodal migration of AGN on crossed electrophoresis and elution pattern of AHF and AGN did not show any significant variations after v.c. in normal subjects as in VWD patients. A likely mechanism of VIII AHF and VIII AGN increase may be a release from endothelial cells of vascular wall during venous congestion.

EFFECT OF CRYOPRECIPITATE INFUSION ON FACTOR VIII RESPONSE TO VENOUS CONGESTION IN PATIENTS WITH VON WILLEBRAND'S DISEASE. M.MORFINI,G.LONGO,P.ROSSI FERRINI and A.SERI. Haematology Department,Florence,Italy.

In patients with Von Willebrand's disease(VWD) venous congestion(v.c.) is unable to increase the VIIIAHF and VIIIAGN level as in normal subjects. To evaluate the effect of rise in VIIIAHF and VIIIAGN levels, 5 patients with VWD received single infusion of lyophilised cryoprecipitate(15 U./Kg) for treatment of bleeding episodes. Two v.c., at 15min and at 300min after the completion of infusion, one on each arm, was performed. VIIIAHF plasma level, raised by infusion,did not show further increase after v.c. at 15min. This finding may suggest that increased levels after v.c. in normal subjects is not achieved by activation of plasma VIIIAHF. In contrast at 300min a significant increase of VIIIAHF was obtained(F test $p<0.05$). On the other hand VIIIAGN high plasma level immediately after infusion showed a significant rise(F test $p<0.05$) after v.c. at 15min but much less at 300min when VIIIAGN is reduced. The divergence on response of VIIIAGN and VIIIAHF to v.c. at different times after infusion may support the mechanism of VIIIAGN conversion to VIIIAHF in VWD. As the plasma VIIIAHF activation appears to be unlikely,the endothelial cells may be responsable for release of AGN and AHF i.e. for conversion of AGN to AHF.