CIRCULATING PLATELET AGGREGATES AND INCREASED PLATELET TURNOVER IN HYPERTENSIVE PATIENTS. R. Abbate, G.F. Gensini, M. Marinoni and A. Lagi. University of Florence Medical School, Florence, Italy.

Cerebrovascular disorders and particularly TIA, frequently due to circulating platelet aggregates, represent a most frequent complication of high blood pressure. 28 patients affected by [illegible] of different aetiology have been investigated for the presence of circulating platelet aggregates according to Wu and Hoak (1974). In 23 patients we observed circulating platelet aggregates unrelated to the aetiology of high blood pressure. These patients showed also an increased number of megathrombocytes (Garg et al. 1971) so indicating the probable formation of irreversible aggregates. These patients usually did not show a plasma aggregating activity investigated by Wu and Hoak method on cross-matches of patient's PPP with control's PRP. The appearance of circulating platelet aggregates is related to blood pressure values, and decreases after the blood pressure has returned to normal values and after anti-aggregating treatment.

ON THE ACTIVATION OF FACTOR X-ACTIVATOR OF THE RUSSELL'S VIPER-VENOM BY CALCIUM. B. B. Nath and Swapan Chattopadhyay. Department of Chemistry, Visva-Bharati, Santiniketan, India.

The relationship between Ca^{2+} ion and factor X-activator of Russell's-viper-venom (RVV) seems to point to some sort of stoichiometry as evidenced from the composition and stability studies using an approximate concept of the Job's method of continuous variation. The concept of the method, used in co-ordination chemistry to ascertain the metal-ligand interacting ratio has been used, in order to interpret the results. The clotting time was measured by varying Ca^{2+} ion and venom concentrations respectively and 'log of clotting time' was plotted against concentration in each case, and the minimum in the curve was taken to represent the maximum formation of the complex of Ca^{2+} ion with factor X-activator of RVV. The plot of clotting time against the ratios of venom and Ca^{2+} ion concentrations also pointed to an interacting ratio. At higher concentrations either of Ca^{2+} ion or of venom, longer clotting time points respectively to the salt-effect and the denaturising effect of the venom on plasma-proteins. Accordingly it is concluded that Ca^{2+} ion activates the factor X-activator in definite ratio before the activated metal-enzyme complex (M-E), acts on its substrate to produce E-M-S or $E\overset{M}{<}S$ for the formation of the products.

FACTOR VIII RELATED PROPERTIES IN PLATELETS OF PATIENTS WITH VON WILLEBRAND'S DISEASE (VWD) Z.M.Ruggeri, R.Bader, T.Barbui and P.M.Mannucci. Hemophilia & Thrombosis Ctr.Univ.Milano, Italy

In 10 normal subjects washed human platelets (Pl)contained FVIII related antigen(VIIIR:AG) as measured by immunoradiometric assay (IRMA)and electroimmunodiffusion(EID);and ristocetin cofactor (VIIIR:RCo)as assayed by a washed platelet method.The observed values were:VIIIR:AG(IRMA) 0.11-0.24u/mg Pl protein;VIIIR:AG(EID)0.11-0.30u/mg; VIIIR:RCo 0.06-0.21u/mg.In 10 pts with severe homozygous VWD,VIIIR:AG was unmeasurable in 7 and extremely low ($1x10^{-3}$-$0.6x10^{-3}$u/mg)in 3 using the very sensitive IRMA;VIII RCo was always unmeasurable.In 12 pts with "classical" dominant VWD characterized by very low plasma levels of VIIIR:AG(0.03-0.09u/ml)and VIIIR:RCo(<0.05u/ml),FVIII related properties were normal in Pl and the mobility of Pl VIIIR:AG on bidimensional immunolectrophoresis was not different from that of normal controls.In 7 pts showing a faster mobility of plasma VIIIR:AG, the same abnormality was found in Pl.Pl VIIIR:AG level was within the normal range when assayed by EID whereas IRMA gave lower values both in plasma and in Pl.Pl VIII R:RCo was lower than in normal subjects and pts with "classical"VWD without electrophoretic variant.These findings show that severe VWD is the expression of a marked reduction of VIII synthesis fully expressed both in Pl and in plasma.In"classical"VWD the plasma defects are not reflected in Pl,which show normal levels of FVIII-related properties accompanied by normal electrophoretic mobility of VIIIR:AG;this suggests a defective transfer from Pl to plasma.Patients with abnormal mobility are the expression of a qualitative alteration of the FVIII molecole functionally defective both in Pl and in plasma.