CIRCULATING PLATELET AGGREGATES AND INCREASED PLATELET THROMBUS IN HYPERTENSIVE PATIENTS.
Cerebrovascular disorders and particularly TIA, frequently due to circulating platelet aggregates, represent a major frequent complication of high blood pressure. 28 patients affected by these circulating platelet aggregates according to Wu and Srouve (1974). In 23 patients we observed circulating platelet aggregates unrelated to the antihypertensive activity of high blood pressure. These patients showed an increased number of megakaryocytes (Gang et al. 1971) as indicating the probable formation of irreversibly aggregated platelets. These patients usually did not show plasma aggregating activity investigated by Wu and Srouve method on cross-matches of patients' PPP with control's FFP. 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.

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 interesting ratio. At higher concentrations of Ca$^{2+}$ ion or of venom, longer clotting time points respectively to the salt-effect and the denaturing 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 (A-E), acts on its substrate to produce E-A or E$^3$ for the formation of the products.

FACTOR VIII RELATED PROPERTIES IN PATIENTS WITH WYLLIE-BARNARD'S DISEASE (VWD) G.M. Baggeri, M. Baden, T. Nishin, and P. M. Marchese. Hemophilia & Thrombosis Ctr, Unil, Milano, Italy.
In 10 normal subjects washed human platelets (PI) contained a VIIIR$a$ antigen (VIIIIR$a$) as measured by immunoneurographic assay (IDA) and electrommunoassay (EMD) and viscoelastic co-factor (VIIIIR$\beta$) as assayed by a washed platelet method. The observed values were: VIIIIR$a$ (IDR) 0.10-0.20 ng/ml, VIIIIR$a$ (EMD) 0.10-0.30 ng/ml, and VIIIIR$\beta$ (EMD) 0.05-0.07 ng/ml. In 20 pts with severe homozygous VWD, VIIIIR$a$ was unmeasurable in 7 and extremely low (1x10$^{-3}$ to 0.6x10$^{-8}$ ng/ml) in 3 using the very sensitive EMD. VIIIIR$\beta$ was always unmeasurable. In 12 pts with "classical" dominant VWD characterized by very low plasma levels of VIIIIR$a$ (0.02-0.05 ng/ml) and VIIIIR$\beta$ (0.04 ng/ml) and VIIIIR$a$ related properties were normal in PI and the mobility of PI VIIIIR$\beta$ on bidimensional immunoblotting was not different from that of normal controls. In 7 pts showing a faster mobility of plasma VIIIIR$\beta$, the same abnormality was found in PI. The plasma factor level was within the normal range when assayed by EMD whereas IBA gave lower values both in plasma and in PI, PI VIIIIR$\beta$ was lower than in normal subjects and PI with "classical"VWD without electrophoretic variant. These findings show that severe VWD is the expression of a marked reduction of VIII synthsis fully expressed both in PI and in plasma. In "classical"VWD the plasma defects are not reflected in PI, which show normal levels of VIIIIR-a associated properties accompanied by normal electrophoretic mobility of VIIIIR$\beta$; this suggests a defective transfer from PI to plasma. Patients with abnormal mobility are the expression of a qualitative alteration of the VIIIIR$a$ molecule functionally defective both in PI and in plasma.