CIRCULATING PLATELET AGGREGATES AND INCREASED PLATELET TURBULENCE IN HYPERTENSIVE PATIENTS.
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Cerebrovascular disorders and particularly TIA, frequently due to circulating platelet aggregates, represent a major frequent complication of high blood pressure. 28 patients affected by circulating platelet aggregates according to Wu and Hook (1974). In 23 patients we observed circulating platelet aggregates unrelated to the agranulocyte of high blood pressure. These patients showed also an increased number of megakaryocytes (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 Hook method on cross-matches of patient's PPP with control's PPP. 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 Ca²⁺ 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 Ca²⁺ and venom concentrations respectively and 'log of clotting time' was plotted against concentration in each case, and the minima in the curves was taken to represent the maximum formation of the complex of Ca²⁺ ion with factor X-activator of RVV. The plot of clotting time against the ratio of venom and Ca²⁺ ion concentrations also pointed to an interesting ratio. At higher concentrations of Ca²⁺ ion or of venom, longer clotting times points respectively to the salt-effect and the denaturing effect of the venom on the plasma-proteins. Accordingly it is concluded that Ca²⁺ ion activates the factor X-activator in definite ratio before the activated metal-enzyme complex (M-X), acts on its substrate to produce Fb-3.5 for the formation of the products.

FACTOR VIII RELATED PROPERTIES IN PATIENTS WITH VON WILLEBRAND'S DISEASE (VWD) E.M. Brugeiri, E. Badert, I. Barbuti and P.M. Mannucci, Hemophilia A Thrombosis Ctr., Univ., Milano, Italy.
In 10 normal subjects washed human platelets (Pl) contained VIlI related antigen (VIII:R:Ag) as measured by immunoradiometric assay (IRMA) and electroimmunoassay (EIA) and ristocetin cofactor (VIII:Rc:Ag) as assayed by a washed platelet method. The observed values were: VIII:R:Ag (IrBM) 0.10-0.45 %, VIII:Rc:Ag (IrBM) 0.04-0.37 µg/ml. In 10 pts with varying degrees of hemostatic impairment, the VIII:Rc:Ag (IrBM) was unmeasurable in 7 and extremely low (1x10⁻⁸-4x10⁻¹⁸/mg) in 3 using the very sensitive IRMA. In 10 pts with "classical" dominant VWD, VIII:R:Ag (IrBM) was always unmeasurable. In 12 pts with "classical" dominant VWD, characterized by very low plasma levels of VIII:R:Ag (0.04-0.09 µg/ml) and VIII:Rc:Ag (0.06-0.10 µg/ml), VIII related properties were normal in PI and the mobility of PI VIII:Rc:Ag on bidimensional immunoelectrophoresis was not different from that of normal controls. In 7 pts showing a faster mobility of plasma VIII:Rc:Ag, the same abnormality was found in PI.PI VIII:Rc:Ag level was within the normal range when assayed by IRMA whereas IRMA gave lower values both in plasma and in PI, PI VIII:Rc:Ag 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 synthesis fully expressed both in PI and in plasma. In "classical" VWD the plasma defects are not reflected in PI, which show normal levels of VIII related properties accompanied by normal electrofophoretic mobility of VIII:R:Ag; this suggests a defective transfer from PI to plasma. Patients with abnormal mobility are the expression of a qualitative alteration of the VIII molecule functionally defective both in PI and in plasma.