MEASUREMENT OF THE ELECTRICAL IMPEDANCE OF BLOOD DURING CLOTTING. W. Theiss and A. Ulmer.

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Characteristic changes of the electrical impedance are observed during blood clotting, the measurement of which can be used as a screening test for hemorrhagic diatheses (L. Arnhold, W. Theiss, J. Tiemann: Deutsche Medizinische Wochenschrift 100:1657-1663, 1975). Large scale use of the test was hampered by the technically delicate experimental set-up of a matched pair of precision measuring cells arranged in a bridge circuit and by the necessity to perform measurements immediately on native blood samples.

Comparative measurements on blood samples of 30 healthy volunteers in a conventional bridge circuit with a pair of measuring cells and in a modified circuit with a single measuring cell revealed good results also with the latter method. Experiments with recalcification of citrated whole blood showed satisfactory correlation with measurements carried out on native whole blood.

This newly developed straight forward procedure of measuring in a single cell and the possibility to work with citrated blood are two major advances in making the measurement of the electrical impedance of clotting blood amenable for use on a larger scale in further research work and eventual routine use.

AGGREGABILITY OF MAMMALIAN AND AVIAN THROMBOCYTES WITH SPECIAL RESPECT TO THE EFFECT OF VARIOUS ANTITHYMOCYTE AND ANTILYMPHOCYTE GLOBULINS. E.A. Suba and G. Csákö. Naval Medical Research Institute, Bethesda, Maryland, U.S.A.

Although interest in platelets is rapidly growing, systematic comparative studies on platelet aggregation are still limited. Here, the aggregation in citrated platelet (thrombocyte)-rich plasmas (PRP) from 18 mammalian (man; chimpanzee; baboon; cynomolgus, rhesus, pigtailed, stump-tailed, and squirrel monkeys; dog; cat; cow; goat; sheep; horse; rabbit; rat; hamster; guinea pig) and one avian species (chicken) was measured by Born's turbidimetric method with agents at final concentrations as given below. Despite the quantitative differences, ADP ($5 \mu\text{M}$), acid-soluble calf skin collagen ($120 \mu\text{g/ml}$) or bovine thrombin (0.5 U/ml) aggregated in all mammalian PRPs. In contrast, epinephrine ($25 \mu\text{g/ml}$) caused aggregation in canine, feline and some primate PRPs, whereas ristocetin (1.5 mg/ml) did in PRPs of certain primates. Chicken thrombocytes were slightly reactive only to collagen and a 4-fold dose of thrombin. Because platelets share antigens with lymphocytes, it was not surprising that all unabsorbed antithymocyte (horse anti-human; rabbit anti-rat, -mouse, -chicken ATGs) and antilymphocyte globulins (rabbit anti-rhesus, -rat, -mouse, -hamster, -guinea pig ALGs) tested exhibited platelet aggregating activity. The cross-reactivity of ATGs or ALGs always followed the phylogenetic order qualitatively, but the quantitative relations (extent of aggregation) frequently differed from that pattern. As an explanation, variations in susceptibility to aggregation among various species can be suggested. In parallel studies, goat antirhesus and rabbit antibaboon platelet sera revealed wide cross-reactions between mammalian species, however, like anti-mammalian ATGs or ALGs, not for the chicken.

HEMOSTASIS DISORDER UNDER PENICILLIN G THERAPY. K. Andrassy, M. Scherz, E. Ritz, E. Walter, R. Zimmermann, W. Vömel, H. Storch, B. Hasper. Med. Univ.-Klinik Heidelberg and Boehringer GmbH., Mannheim, F.R.G.

Systemic administration of penicillin G (P) has been shown to cause a coagulation disorder (Andrassy et al., Lancet II, 1039, 1976). In this study time course, dose effect relationship and effect on viscosity were investigated. - 20 pat. with normal GFR received P either intermittently (i.i.) ($2 \times 20 \text{ mio U/d}$ as 1h short inf.) or by constant drip inf. (c.i.) (40 mio/d); Analysis: Bleeding time, (B.T.); platelet aggreg. test; coagulation factors (AT III, fXa); rotation/capil. viscosimetry of blood; serum P conc. (diff. test). An increase in B.T. and dimin. collagen resp. ristocetin ind. platelet aggreg. was usually found 6 h and consistently 24 h after P infusion. After stopping P, the abnormalities were demonstrable for 4 days (i.i.) and for 8 days (c.i.). Serum P levels (preinfusion) were $0.5-8 \mu\text{g/ml}$ (i.i.) and $40-80 \mu\text{g/ml}$ (c.i.) 8 days after stopping P, reactive thrombocytosis was noted in the c.i. group. Blood viscosity, if anything, increased; therefore the prolongation of B.T. is not accounted for by abnormalities of blood viscosity. In additional pat., $2 \times 5 \text{ mio P}$ were without noticeable effect on hemostasis, whereas definite abnormalities were demonstrable with 20 mio P/day . In the plasmatic system, increased AT III and diminished f. Xa were found irrespectively of the mode of administration. Minor bleeding (epistaxis, macrohematuria, wound bleeding) was observed in 5/20 patients.