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function were normal. Plasmatic antithrombin III activity was normal whereas its serum activity was increased. The bleeding time was moderately prolonged and clot retraction impaired. The thromboelastogram showed reduction of the "ma." and plasma polymerization was deficient. These last two abnormalities were partially corrected by transfusions of fresh plasma. Aggregation of fibrin monomers was also deficient whereas fibrinopeptide release was quantitatively normal. The abnormal fibrinogen has increased immuno-electrophoretic mobility and an abnormal sedimentation pattern.

Her son had a markedly prolonged thrombin time with bovine thrombin whereas with the human enzyme the test was moderately abnormal. Plasma polymerization was absent and sedimentation cooefficient increased. Reptilase time was normal. The abnormal fibrinogen is tentatively designated fibrinogen Buenos Aires, since its possible identity

with other abnormal fibringen has not been excluded.

C. Soria, J. Soria, M. Samama, E. Poirot and C. Kling (Laboratoires d'Hématologie et de Biochimie (Prof. Caen et Rousselet) Hôpital Lariboisière, Paris, Service du Professeur Bousser (Prof. Ag. Samama), Hôtel Dieu, Paris. Centre de Transfusion, Metz): Role of Trisodium Citrate in the Unclottability by Thrombin of Fibrinogen Metz. (225)

In a case of homozygous dysfibringenemia, the whole blood clotting time was moderately prolonged, while the thrombin clotting time was infinite, whatever dose or nature of thrombin used. Besides, the bleeding syndrome in this case was very weak.

We observed also that only after trisodium citrate addition to purified fibrinogen, the abnormal fibrinogen became unclottable by thrombin even after addition of calcium

chloride, since without trisodium citrate thrombin time was only prolonged.

By immunoelectrophoresis and by isofocusing in the presence or in the absence of trisodium citrate, we therefore undertook to show that trisodium citrate reacts more strongly with the abnormal fibringen than with normal one. Thus, trisodium citrate conferring a negative charge to the pathological molecule, the abnormal fibringen became resistant to clotting with thrombin. Protamine sulfate, by positiving the charges of fibringen, partially corrects the defect in fibrin formation.

N. B. Bosch and C. Arocha-Piñango (Banco Municipal de Sangre and IVIC, Caracas, Venezuela): An Abnormal Fibrinogen in a Venezuelan Family. (226)

An apparently new fibrinogen abnormality, inherited as an autosomal dominant trait, was found in 4 members of a family affected with moderate bleeding. Prolongation of plasma prothrombin, thrombin and Reptilase times and normal concentration of fibrinogen were observed. Normal values of platelet aggregation, FDP and antithrombin III levels were obtained. The k value of the thromboelastogram was slightly increased. Factor XIII was 50 to 60% (Beringwerke F XIII kit) and 100% by radial immunodiffusion. Photometric study of thrombin induced polymerization of fibrin in plasma or purified fibrinogen and the aggregation of monomers in plasma measured by a two stage method, were abnormal. Marked prolongation of the clotting time of fibrin monomers were observed at pH over 7.4. Alterations of polymerization curves and plasma thrombin time were accentuated by raising molarity and ameliorated by Ca ions, increasing thrombin concentration and frozen storage. Normal plasma was slightly inhibited by patient's plasma in these tests.

Normal electrophoretic mobility of the abnormal fibrinogen was shown on cellulose acetate, polyacrylamide and inmunoelectrophoresis. Ouchterlony inmunodiffusion revealed non-identity pattern with normal fibrinogen.

L. Tranqui, V. J. Marder, M. Suscillon, A. Z. Budzynski and G. Hudry (CEN-Grenoble, DRF, Hématologie, BP 85, 38041-Grenoble, France): Electron Microscopic Studies of the Molecular Shape of Fibrinogen Molecule. (227)

Electron microscopic studies of fibrinogen have demonstrated many variations in shape and size, with a corresponding number of opinions regarding its transformation to fibrin.