M. L. Scrobohaci, N. Stăncioiu, N. Stefănescu and S. Idu (The Center of Cardiology – Bucarest, Romania): **Platelet Factor XIII in Hyperlipidemias. (355)**

The total platelet factor XIII – T. P. F. XIII (after destroying by triton) and the released one – R. P. F. XIII (during thrombin induced platelet aggregation) were quantificated in IIa, IIb, IV types of hyperlipemia and myeloproliferative disorders subjects compared to normal control subjects.

T. P. F. XIII was increased in IIb and IV, and normal in IIa types of hyperlipemia while it was decreased in myeloproliferative disorders. R. P. F. XIII was increased only in IIb, "nul" in IIa, IV types of hyperlipemia, myeloproliferative disorders and control subjects too.

Both T. P. F. and R. P. F. XIII increased values in IIb parallel the increased platelet aggregation in these subjects. On the contrary the lack of R. P. F. XIII in IIa and IV types is concordant with normal platelet aggregation revealed in our subjects too. In myeloproliferative disorders when the platelets are "pool-vide" the T. P. F. and R. P. F. XIII is much decreased. These data supported the enhance thrombogenic tendency in IIb type hyperlipemia by platelet increased activity and decreased fibrinolysis due partially to enhanced fibrin stabilization. Other factors incriminated in the decrease of fibrinolysis and increased platelet aggregation, not discussed here, may be responsible for the type IV as thrombogenic as IIb and may counter balance our disordant data in these more prevalent types. The myeloproliferative disorders data are consensual with the tendency to bleeding disorders.

T. Mandalaki, C. Dimitriadou and C. Louizou (2nd Reg. Blood Transfusion Centre Athens Hospital Vasileus Pavlos – Athens – Greece): **Platelet Function in Various Haemorrhagic Disorders. (356)**

Platelet aggregation by ADP, collagen, thrombin and ristocetin was studied systematically both in citrate platelet rich plasma and isolated platelets by density gradient using albumine (according to Nicholls and Hampton) in various cases of congenital haemorrhagic diathesis, namely v. Willebrand disease, Glanzmann disease (thrombasthenia), Thrombopathy (PF_3 defect), Factor XIII deficiency and in unclassified hereditary haemorrhagic disorders as well as in acquired bleeding tendency. According to the platelet abnormalities found during this study a classification of "Thrombopathies" observed in Greece is attempted.

J. I. Haft and Y. S. Arkel (St. Michael's Hospital, Newark, New Jersey, U.S.A.): **Effect of Emotional Stress on Platelet Function in Humans. (357)**

Emotional stress is one of the important risk factors associated with coronary atherosclerosis and acute myocardial infarction. The initial event in the formation of an atherosclerotic plaque may be a mural platelet aggregate forming on the endothelial surface of an artery; and the final event in myocardial infarction may be the plugging of a narrowed area of a coronary vessel by an inappropriately forming intravascular platelet aggregate. To study the effect of emotional stress on platelet function, 14 normal interns and residents were studied immediately before, immediately after, and 7 days after presenting a case before a large critical group of attending physicians. Response of platelet rich plasma to ADP and epinephrine was determined using a Chronolog aggregometer. In eleven subjects the rate of first phase aggregation, the percent total aggregation and the length of the plateau phase with epinephrine indicated significantly inhibited platelet function immediately after stress as compared to the baseline study 7 days later. Eight subjects responded similarly to ADP. In 4 subjects similarly decreased function occurred in the prestress (anticipation) period. It is concluded that emotional stress has a profound effect on platelet function, an effect that may suggest that platelets aggregate intravascularly during stress, deaggregate, and then are relatively insensitive to aggregation immediately after the stress period.