G. Arapakis, J. Andriopoulos, A. G. Papayannis and C. Gardikas (Evangelismos Medical Center, Athens 140, Greece): **The Effect of Hypoglycaemia on Platelet Count, Platelet Adhesiveness and Bleeding Time.** **(358)**

The effect of hypoglycaemia (HG) on platelet count (PC), platelet adhesiveness (PAd) (Salzman's method) and bleeding time (Ivy's method) was studied in 30 healthy volunteers (12 males and 18 females), aged 17 to 55 years. Insulin cryst. 0.2 u/Kg b.w. was given i.v. and the platelet tests were performed immediately before insulin administration and during maximum HG. In 7 subjects PC and PAd were repeated immediately after the HG was reverted and 2h and 24 h later. It was found that PC and PAd were significantly increased during HG ($p < 0.001$); especially PC increased from $m = 251 \pm 95X10^3$ to $m = 288 \pm 95X10^3$, and PAd from $m = 48 \pm 13\%$ to $m = 72 \pm 11\%$. These increased values persisted for 2h after HG was reverted by glucose infusion and returned to pre-insulin levels 24 h later. On the other hand the bleeding time showed a significant shortening from $m = 3.2 \pm 0.9$ min to $m = 2.1 \pm 0.8$ min during HG ($p < 0.001$). There was no correlation between the changes of the various parameters studied.

F. E. Preston, W. R. Timperley, B. C. O'Malley and J. D. Ward (The Royal Infirmary, Sheffield S6 3DA, England): **Intravascular Thrombosis and Platelet Function in Diabetic Neuropathy.** **(359)**

Peripheral neuropathy is a serious complication of Diabetes Mellitus. The precise pathogenesis is unknown. Histological examination of biopsy material obtained from patients with diabetic neuropathy has revealed intravascular fibrin in vessels supplying the sural nerve in 10 out of 25 cases. This finding prompted us to study platelet function in this same group of patients.

Blood samples were obtained from fifteen patients. After preparation of platelet-rich plasma, platelet aggregation was measured following the addition of various concentrations of ADP, collagen and adrenaline. The responses were assessed photoelectrically in a temperature-controlled, constantly stirred, non-glass system. In addition the samples were tested for spontaneous platelet aggregation.

Spontaneous platelet aggregation was detected in one-third of samples studied, but in none of the controls. Enhanced ADP-induced aggregation was observed in 53% of samples and enhanced adrenaline-induced aggregation in 50%. Similar results were obtained with collagen.

The data supports the view that enhanced platelet aggregation and intravascular thrombosis is of considerable importance in the pathogenesis of diabetic neuropathy.

E. Pogliani, E. Cofrancesco, A. Della Volpe, M. Cortellaro, G. Masera and E. E. Polli (Medical Clinic I^ and Pediatric Clinic (°) University of Milan – Italy): **Assay of Anti-platelet Antibodies in Immune Thrombocytopenic Patients by Sedimentation Pattern Test.** **(360)**

15 sera from never transfused thrombocytopenic children (idiopathic thrombocytopenic purpura, drug induced and post-viral thrombocytopenia) were performed in a micro-technique according to the modified sedimentation pattern test of Myllyla (1). Eighter per cent of these sera, tested against a pannel of at least five healthy donors, gave a positive result at different dilutions (from $^1/_4$ to $^1/_{16}$). This quantitative platelet agglutination assay, compared with platelet aggregometry and 14 C-serotonin release, seems to be a sensitive, simple and reliable method to detect antiplatelet antibodies. Therefore it could have clinical applicability to the study of patients affected by immune thrombocytopenia.

(1) Myllyla, G.: Aggregation of human blood platelet by immune complexes in the sedimentation pattern test.