Time
15.15 **0796** PLATELET FUNCTION TESTS IN RELATION TO RETINOPATHY AND NEUROPATHY IN WELL CONTROLLED DIABETIC PATIENTS.

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The following tests were done in 61 well controlled diabetic patients: Platelet survival time (PST), platelet aggregation ratio acc. to Wu and Hoak (PAR), platelet aggregation test 1 acc. to Breddin (PAT 1), ADP-threshold concentration and filtragometer aggregation time acc. to Hornstra (Ta). The mean PST in 20 healthy volunteers was 99 hours ± 13,4 hrs.; in 12 pat. wihtout retinopathy 88,6 ± 12,8 hrs.; in 28 pat. with non-proliferative retinopathy 103,9 ± 18,2 hrs. and in 4 pat. with proliferative retinopathy 105,8 ± 7,5 hrs. In 16 pat. with a disturbed nerve conduction time the mean PST was 102,2 ± 21,5 hrs. and 96,0 ± 21,9 hrs. in 28 pat. with a normal time. The PST was shortened (mean-1SD) in 12 pat. out of 44 pat. The mean PAR was 0,87 ± 0,11 in 27 normal volunteers and 0,78 ± 0,12 in 61 diabetics. The PAR was < 0,71 in 16 out of 61 pat. The mean PAT 1 was 1,7 in 27 normal volunteers and 1,5 in 61 diabetics; the test was abnormal in 6 out of 61 pat. The ADP-threshold concentration was < 0,75 mumol ADP/L in only 4 out of 61 pat. The mean Ta was 242 ± 50,5 sec. in 16 normal volunteers and 167 ± 83 sec. in 44 pat.; the Ta was < 150 sec. in 23 out of 44 pat. There was no correlation between the severity of retino- and/or neuropathy with the PST, PAR, PAT 1, ADP-threshold or Ta or between the laboratory values. There was no abnormal laboratory test in 23 pat., one in 19, two in 16 and three in 3 pat. There was no relationship between the severity of retino- and/or neuropathy and number of abnormal laboratory tests.

15.30 **0797** PLATELET HYPERFUNCTION IN INFANTS OF MOTHERS WITH DIABETES MELLITUS (DM). M.J. Stuart,* H. Elrad, D.O. Hakanson, J.E. Graeber, S. Sunderji and M.K. Barvinchak. Dept. of Peds., SUNY, Upstate Med. Ctr., Syracuse, N.Y.

Platelet aggregation and prostaglandin formation was evaluated in 20 control mother-neonate pairs (Grp I), and in 13 pairs where maternal DM was present (Grps II and III). Grp I control mothers demonstrated normal platelet aggregation with their infants showing the physiological impairment in aggregation that occurs in the neonate. Using platelet malonyldialdehyde (MDA) as an indicator of platelet prostaglandin formation, Grp I mothers and infants demonstrated normal values of 3.20 ± 0.31 (1 SD) and 2.46 ± 0.61 n moles MDA per 10^9 plts respectively. In the 13 patient pairs, 8/13 diabetic mothers (Grp II) showed platelet hyperaggregability and platelet MDA was increased to 3.92 ± 0.22. All Grp II infants also manifested platelet hyperfunction and increased MDA formation ($p<.005$) to 3.37 ± 0.67. 2/8 Grp II neonates restudied on the fourth day of life no longer demonstrated the hyperfunction present at birth. 5/13 diabetic mothers (Grp III) showed normal platelet aggregation and MDA formation (3.18 ± 0.17) and their infants showed normal neonatal aggregation and MDA formation (2.27 ± 0.67). In the adult with DM, platelet hyperfunction and increased prostaglandin formation is present. These findings appear to be transmitted to the offsprings of such mothers as well. Platelet hyperfunction was not correlated with either neonatal blood sugar or blood viscosity measurements. Platelet hyperfunction may be the etiologic factor in the increased incidence of both arterial and venous thrombosis that occurs in the infant of the diabetic mother.

15.45 **0798** PROSTAGLANDINS AND PLATELET FUNCTION IN DIABETES

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We have studied platelet aggregation induced by 0,5 mM. Araquidonic acid (AA) addition to platelet-rich-plasma (PRP) from 21 insulin treated diabetic patients and in 21 non-diabetic controls. The velocity of aggregation was significantly higher in the diabetic group. There was no differences in the velocity of aggregation in patients with or witout retinopathy.

The incubation of PRP of normal subjects at 37º during 5 minutes with $5,8\ 10^{-4}$ M. Imidazole changed the pattern of aggregation: The velocity of aggregation was slower and appeared a wave of disaggregation. Imidazole had not effect on aggregation in the diabetic group.

This data add support to the findings published by COLWELL showing that platelets from diabetics have hyperactive AA metabolism.

Prostaglandin I_2 (PGI_2) obtained from rat aorta shows an inhibitory effect on ADP or AA induced aggregation. This effect is less marked in diabetic PRP than in PRP of normal controls. PGI_2 release in platelet-poor-plasma from diabetics is normal. This can represent a resistance of diabetic platelet to the antiaggregating effect of PGI_2.A similar finding was also appreciated with the PGE_1 in three out of six patients so far stu