ARACHIDONIC ACID METABOLISM OF PLATELET IN DIABETES. T.Kurosawa, T.Tojima, H.Funayama, Y.Takahashi and Y.Shiohara. Juntendo University, School of Medicine, Bunkyo, Tokyo, Japan.

Recent reports have indicated that platelet aggregation is enhanced in some diabetics who have proliferative retinopathy and that platelet function is altered by glucose loading. But the mechanism is not clarified yet. Arachidonic acid, the precursor of prostaglandin endoperoxide, plays a major role on platelet aggregation. Blood samples were collected with endoperoxide at 0, 30, 60, 120 and 300 minutes after 100 g glucose loading. Platelet-rich plasma was obtained by centrifugation and platelet aggregation was studied photometrically adding ADP. Platelet was obtained by further centrifugation and was kept freeze-dried. Platelet samples were extracted and transesterificated and separated by gas liquid chromatography. The quantitative regulation of arachidonic acid in platelets was measured by the composition ratio of arachidonic acid (C_{20-4})/linoleic acid (C_{18-2})=AL index. The results of platelet aggregation after glucose loading were as follows; platelet aggregation was not changed remarkably in normal subjects, but was enhanced at 30 and 60 and suppressed at 120 minutes in diabetics. AL index is as follows: prior to glucose loading, AL index of diabetics (4.6 ± 1.2) was higher than that of normal subjects (3.5 ± 0.5). After glucose loading, no significant change was observed in normal subjects, but AL index was increased at 30 (4.8 ± 1.4) and 60 (4.9 ± 1.4) and decreased at 120 minutes (4.1 ± 0.9) in diabetics. The results indicate that there is a certain relationship between quantitative regulation of arachidonic acid in platelet and platelet aggregation and that hyperaggregation may be induced by abnormal prostaglandin metabolism in diabetes.

STRUCTURAL FEATURES IN "SPONTANEOUS" FOCAL ENDOTHELIAL CELL DESQUAMATION OF RABBIT AORTA. E.Svendsen and L.Jørgensen. University of Tromsø, Institute of Medical Biology, Tromsø, Norway.

Rabbit aortas were examined by light, scanning and transmission electron microscopy to observe the morphological steps in focal endothelial cell desquamation. The aortas were not subjected to any instrumentation prior to fixation by either immersion or perfusion. In areas where boundary layer separation of flow with eddy formation is known to occur, many elongated protruding injured cells were observed, some obviously partly loosened. Some of the latter cells appeared to be suspended in two ends and were twisted longitudinally. Single cells, or even sheets of cells, were completely detached; the breaks seemed to have taken place close to, and parallel with the intercellular junctions, but not within these structures. Platelets had reacted both with injured cells and the denuded intimal surface. Thus, the sequence appears to be protrusion of cell body, partly loosening of cells through breaks near the periphery, twisting of cells, and complete detachment.

FIBRINOLYTIC ACTIVITY AND EFFECTS ON THE COAGULATION SYSTEM OF A MICRO-ENZYME OF BACILLUS CEREUS. B.T. af Ekenstam and P. Friberger, AB KABI, Peptide Research, Mölndal, Sweden.

A microenzyme of *Bacillus Cereus* isolated by J. Björkstén et al., Björkstén Research Foundation, Madison, Wisc., USA, showed a caseinolytic activity of 2600 CU/mg. This enzyme has a potent fibrinolytic activity tested on a plasminogen free fibrin plate. The effect on the coagulation system has been compared with that of plasmin and brinase. The microenzyme shows an inhibiting activity on the coagulation of plasma of the same magnitude as that of plasmin and brinase. This inhibition is also correlated to the results obtained from coagulation studies on whole blood using a modified Chandler loop technique. The chromogenic substrate S-2238 has been used for the indirect assay of the activity of the microenzyme over a standardized thrombin system.