

## Editorial

### PLATELET FUNCTION AND OCCLUSIVE VASCULAR DISEASE IN DIABETES MELLITUS

A disturbance in the platelet function may be a causative factor of occlusive vascular disease which is frequently encountered in diabetes mellitus. A change in platelet aggregation and disaggregation was observed by various research workers. Patients suffering from progressive retinopathy showed accelerated aggregation and deficient disaggregation (Colwell et al., 1973; Heath et al., 1971). The effect of insulin or oral antidiabetics on these two actions has not yet been completely investigated.

The release of platelet factors 3 and 4 has also been studied in diabetes. PF-3 is a complex phospholipid released after aggregation and it accelerates the coagulation process. PF-4 is a heparin neutralizing substance. PF-3 was found to be increased in juvenile onset diabetics (Kwaan et al., 1972; Nordoy and Rodset, 1970), whereas PF-4 was slightly increased in the maturity onset patients (Chmielenski and Farbiszewski, 1970).

An increased platelet adhesion in diabetics has not yet been fully established. Experiments in vitro show an increased platelet adhesion after adding glucose both to blood from normal and diabetic subjects (Bridges et al., 1965). In vivo this phenomena was observed in all diabetics and non-diabetics having coronary artery disease, after being given 50 Gm Glucose orally. The peak increment of adhesion occurred one hour post prandially (Bagchi et al., 1970).

A shortened platelet survival has been noted in diabetics suffering from retinopathy, nephropathy, coronary artery disease and peripheral vascular disease. Diabetics without vascular complications had normal platelet survival (Abrahamsen 1968). Von Willebrands' factor, a globulin normally present in plasma and necessary for platelet adhesion, is found in a higher concentration in patients with diabetes especially those with retinopathy (Pandolfi et al., 1974). Lipids and fatty acids show an increased concentration in platelets of diabetics. The increased activity of surface glucosyl-transferase which facilitates platelet collagen adhesion leads to increased aggregation (Waitzman et al., 1977). Prostaglandins and their precursors are now known to have an important influence on platelet aggregation (Samuelson 1977). If there is any alteration in this complex system is yet to be studied in the diabetic's platelets.

The experiments with coagulation dynamics report an abnormal finding of these factors in diabetic patients (Egeberg 1963; Valdorf-Hansen

1967). Thrombus size was found to be increased in the Chandler apparatus, in diabetics and was still larger in those with accompanying vascular disease (Rathbone et al., 1970).

The extensive studies of platelet function to establish a link between the deranged metabolism of diabetes mellitus and vascular disease have still not given any definite clue to the causative factor. It has however been established that there is a link between accelerated platelet function and accelerated vascular disease of diabetes mellitus thus offering suggestions that may aid the further determination of the etiologic relationship between the two conditions.

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