

## THROMBIN INHIBITING CAPACITY OF NORMAL AORTA.

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The present investigation was designed to investigate the capacity of normal vessels to inactivate activated coagulation enzymes.

Fresh and frozen porcine and canine aortae were exposed in vitro to thrombin, dissolved in an albumin solution. Thrombin activity appearing on the vascular wall was assayed with a chromogenic substrate (S-2238-Kabi). Aortic endothelium retained considerable amounts of thrombin. After exposure of the aorta with plasma, a rapid inactivation of thrombin was observed. Exposure of the thrombin-absorbed endothelium to a modified Ringer's solution resulted in a much slower inactivation. A similar slow inactivation was seen after exposure with plasma devoid of antithrombin III. No difference was observed between fresh aortae and those stored in a frozen state. No inactivation occurred during the observation time when the thrombin-albumin solution was mixed with the modified Ringer's solution without presence of aortic endothelium and when mixed with normal plasma only slow activation was seen.

It is concluded that the aortic wall can take up thrombin and inactivate it. This occurs in the absence of plasma but it is greatly accelerated by plasma containing antithrombin III.

## ERYTHROCYTE ADHESION TO ENDOTHELIAL CELLS AND GLYCOSYLATED

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We have observed increased erythrocyte adhesion to cultured endothelial cells in diabetes mellitus (see Wautier et al.). This study was designed to determine if glycosylated haemoglobin is involved in this abnormal RBC-endothelial interaction. Erythrocytes from 29 diabetic subjects were examined. Adhesion was measured using  $^{51}\text{Cr}$ -labelled erythrocytes and calculated by counting the radioactivity remaining after 5 washes. The percentage of haemoglobin  $\text{A}_{1\text{c}}$  ( $\text{HbA}_{1\text{c}}$ ) was measured by a standard microcolumn technique. Mean  $\text{HbA}_{1\text{c}}$  concentration was 11.4%, with a range of 6.8 to 17% (upper limit of normal 7.5%). Adhesion, expressed as an adherence ratio, did not correlate with the level of  $\text{HbA}_{1\text{c}}$  ( $r$  0.23, n.s.). To investigate if a sub-population of erythrocytes with an elevated  $\text{HbA}_{1\text{c}}$  adheres more strongly to endothelial cells, we have measured the concentration of  $\text{HbA}_{1\text{c}}$  in RBC from successive washes. The erythrocytes from 5 diabetics were studied, with a range of concentrations of  $\text{HbA}_{1\text{c}}$  between 9.8-12.6% (measured by a column technique). In each experiment, the RBC from a diabetic were incubated on 8 identical endothelial cultures. The cultures were then rinsed and the non-adhering erythrocytes from each of the 5 washes were pooled and the  $\text{HbA}_{1\text{c}}$  was measured.  $\text{HbA}_{1\text{c}}$  (mean  $\pm$  SEM) in the 5 washes varied between 10.94  $\pm$  0.49% and 11.26  $\pm$  0.49%.

These results indicate that increased adhesiveness of erythrocytes from diabetics is not related to the concentration of glycosylated haemoglobin.