FACTORS AFFECTING THE FACTOR X ACTIVATOR ACTIVITY OF HUMAN PLATELETS. J. Vermylen and N. Semere-
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Several recent studies have indicated that patients with a thrombotic tendency have enhanced platelet coagulant activity. It therefore seems important to attempt to identify factors which modify platelet coagulant activities. Recent work in our laboratory has provided evidence that human platelets possess the capacity to directly activate factor X. Adenosine 5'-diphosphate, collagen, acetylsalicylic acid and prostaglandin E1 do not modify this activity. All endotoxins studied so far clearly enhanced this activity. Furthermore, infusion of 'activated' prothrombin concentrates in haemophiliacs with or without factor VIII inhibitor enhanced the activity of this platelet activator of factor X. The hypothesis has been put forward that this may be the mechanism through which 'activated' prothrombin concentrates 'bypass' factor VIII of IX inhibitors. Platelet isolated from platelet-rich plasma to which the 'activated' prothrombin concentrate had been added at a concentration approaching the maximal concentration achieved following in vivo infusion, also showed an increase in platelet coagulant activity. Work is in progress to identify the component in 'activated' prothrombin concentrates which enhances platelet coagulant activity.

SPONTANEOUS PLATELET AGGREGATION IN VIVO. J.C. Hoak. University of Iowa College of Medicine Iowa City, Iowa, U.S.A.

In recent years several new methods have been developed to quantitate the presence of platelet aggregates in whole blood. Included are techniques using an electronic particle analyzer after lysis of the RBC's with saponin, fixation of platelet aggregates with dilaurel formalin, and measurement of changes in filtration pressure in systems where platelet aggregates obstruct the flow of blood across standardized filters.

Using the formalin-fixation method (Lancet 2:594, 1974) to detect platelet aggregates we have found abnormal values in patients with transient ischemic attacks, acute myocardial infarction, acute peripheral arterial insufficiency, and patients with recurrent deep venous thrombosis, migraine, and certain types of ischemic retinopathy. Questions remain, however, as to whether the detected aggregates are definitely circulating aggregates or whether the technique selects out a population of abnormal platelets which bear differently upon exposure to formalin than to the normal platelets. While the current method appears to be satisfactory to delineate patients in whom platelet aggregates may be playing a primary or secondary role, additional information is required to provide a satisfactory explanation of the mechanisms involved if we are to achieve an optimal application of the technique.

IN VITRO SPONTANEOUS PLATELET AGGREGATION IN CEREBROVASCULAR DISEASE. J.W. ten Cate.
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Fifty patients from a group of 130 patients with transient ischemic attacks or cerebral infarction were found to demonstrate in vitro spontaneous platelet aggregation (SPA). This phenomenon occurred within 15 minutes when platelet-rich plasma samples (PRP) of these patients were stirred at 37°C in an aggregometer.

In addition all patients showing SPA also demonstrated a lower threshold concentration for the onset of ADP-induced second wave aggregation (ADP < 0.25 μM; normal range 0.75 ± 0.2 μM). Of the remaining 80 patients 25 patients were found to be sensitive to low concentrations of ADP.

SPA remained present in samples of 8 patients studied at room temperature for two hours. SPA was found to be dependent upon the presence of divalent cations and could be prevented by adenosine, phenylamidine and aspirin. The following additional findings point towards a possible platelet defect.

1. Platelets from 10 patients with SPA when isolated and resuspended in normal plasma still demonstrated SPA while isolated normal platelets in patients did not.
2. Platelets demonstrating SPA showed an increased aggregation tendency upon incubation with ADP while normal platelets developed the expected refractory state for ADP.