H. Rieger and H. Schmidt-Schönbein (Department of Physiology, D 5: Aachen, Melatenstr. 211): Formation and Destruction of Platelet Aggregates in Viscometric Flow.

Even after pseudopodia formation platelets – unlike all other known formed blood elements – remain dispersed in stasis and creeping flow and become aggregated only in the presence of a minimum amount of shearing. The “rheoaggregometer” (Rieger et al., Pflüger’s Archiv, 343, R 33, 1973) allows to measure the minimum shear rate necessary for platelet aggregation (PA), as well as the initial rate and the maximum extent of PA in citrated PRP.

PA is quantified photometrically as a function of variable shear rates. The initial rate of PA steadily increases with increasing shear rates up to 460 sec⁻¹. However, the maximal extent of PA (indicating the mechanical integrity of formed aggregates) saturates at about 35 sec⁻¹ and then decreases because of a destruction of formed aggregates and of prevention of further PA. The aggregability of the platelets, as reflected by various degrees of shape changes, is enhanced by a drop of temperature and a rise in pH as well as by the so-called aggregating agents (e.g., epinephrine 10⁻⁴ up to 10⁻² M/l); consecutively lower shear rates (lower effects of collision) are necessary to induce PA. In citrated PRP stable platelet aggregates are produced only within a defined range of shear rates. Platelet aggregability and aggregate stability are independent variables influenced by different experimental conditions.

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Binding of 125I fibrinogen to platelets was studied after incubation of 125I fibrinogen with PRP, gel filtered and washed platelets. Thereafter 125I fibrinogen was completely removed from the platelets by four washes by the albumin cushion method. When the 125I fibrinogen preparation was contaminated by factor VIII antigen binding to platelets was observed. This suggested that factor VIII antigen present in some fibrinogen preparations could be responsible for the controversy in the literature concerning binding of fibrinogen to platelets or not. Therefore factor VIII antigen (V₈ fraction of sepharose 6 B chromatographed cryoprecipitate, von Mourik) was labeled to study its binding to platelets. Factor VIII (V₈) labeled with 125I as described by MacFarlane resulted in cocomposition of the factor VIII molecule as measured by its antigenic and procoagulant activity and Von Willebrand factor activity, whereas the labeling with the Chloramine-T method resulted in a different behaviour of the molecule on the polyacrylamide gel-electrophoresis. Using the lactoperoxidase method the factor VIII molecule remained completely intact. Incubation studies were performed with PRP and this 125I labeled factor VIII preparation. Subsequently the platelets were washed four times by the albumin cushion method and radioactivity was measured of the washing buffer and of the washed platelet suspensions. Binding was observed of radioactivity to the platelets. A dose-relationship was demonstrated.


Decreased quantitative RPA of PRP is used to distinguish patients with von Willebrand’s disease from normals and hemophiliacs. We observed that RPA (1.8 mg/ml final concentration) of PRP from 9 normals varied markedly with time after PRP preparation. In 6/9 the initial RPA was greatest and decreased progressively with time. In 3/9 RPA increased over the first half hour and then decreased progressively. The decrease from peak values was 37 ± 12% (mean ± 1 SD) at 1 hr, 69 ± 20% at 2 hr and 70 ± 10% at 2.5 hr.