On hydrophilic surfaces a loosely packed layer of up to 130 Å grows. The refraction index is 1.4 which is the value found in the literature for hydrated proteins. The fibrinogen readily reacts with a specific antibody. No other proteins adsorb onto this layer.

On hydrophobic surfaces a layer deposits that is much more closely packed (n = 1.5; thickness 70 Å). In the course of 0.5–1 hour this layer rearranges to a still denser structure (n = 1.8; d = 35 Å). These dense structures too react with specific antibodies but no other proteins will be adsorbed.

The structure of the γ-globulin layer is dependent on the structure of the underlying fibrinogen. Fibrinogen does replace adsorbed layers of other proteins. These observations can be used as the basis for the screening of surfaces for their antithrombotic properties.

A. L. Copley[1], *R. G. King*[1] *and B. Blombäck*[2] (Laboratory of Biorheology[1], Polytechnic Institute of New York, Brooklyn, N.Y. 11201, Plasma Protein Dept.[2], New York Blood Center, New York, N.Y. 10021, U.S.A. and Dept. of Blood Coagulation Research[2], Karolinska Institutet, Stockholm 60, Sweden): **Comparative Hemorheological and Biochemical Studies of a Bovine Fibrinogen. (182)**

Among numerous fibrinogen (Fg) preparations of different mammalian origins (human, bovine, sheep, rabbit, cat) in measurements of viscous resistance -VR- (torque values, τ, dyne. cm) of surface layers (SLs), we found as an exception a bovine Fg, FgSM (Schwarz-Mann, Orangeburg, N.Y. Lot # Y1013), which did not show measurable VR at shear rates (SRs) from 10^{-3} to 10^{-1} sec^{-1} without prior application of high shearing forces (hSF). This led to biochemical characterization, in which FgSM did not exhibit any significant difference in chain structure, as compared with highly purified Fg showing high τ, indicating that a dialysable component in the FgSM was responsible for the inhibition of VR. Our biochemical studies could not detect significant differences between the FgSM and Fg from other sources. A hSF at 1000 sec^{-1} for 3 min was then applied prior to the rheological tests at low SF from 10^{-3} to 10^{-1} sec^{-1}. This procedure always resulted in significant increases in VR of SLs of all Fg preparations, including the FgSM. High shear, which exists at the vessel wall in vivo, is particularly high in the microcirculation and is also augmented in vortex and disturbed flow at sites of branchings, bends and bifurcations in the vascular system. The hSF may contribute to the initiation of thrombus formation due to the proposed progressive adsorption of Fg, layer upon layer, at these sites (Copley, Biorheol. *8*, 79, 1971; Microvasc. Res. *8*, 192, 1974). A further hypothesis is introduced in which the polymerization sites of the Fg molecule are unfolded by the hSF at the vessel wall, resulting in intravascular polymerization of Fg.

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J. N. Lindon, D. Brier, E. W. Merrill and E. W. Salzman (Department of Surgery, Beth Israel Hospital, Harvard Medical School, Boston, Ma., U.S.A.): **Interaction of Platelets with Model Surfaces III: Platelet Reaction with Synthetic Polymers. (183)**

Blood/material surface interactions have been studied by passing citrated whole blood over beads in a column and examining the resultant activation of platelets and coagulation factors. Examination of many polymer and some crystalline surfaces indicates that platelet adhesion occurs with all surfaces except after pretreatment with albumin in some instances. Induction of the platelet release reaction and platelet adhesion vary from one surface to another and are not well correlated. The release reaction in response to some but not all surfaces can be blocked by pretreatment of blood with aspirin *in vivo* and indomethacin *in vitro*. PRP exhibits less activation than whole blood, but varying the hematocrit of whole blood from 23 to 53% does not change surface reactivity.

Of the materials studied, polyethylacrylate (PEA) and polymethylacrylate (PMA) appear least reactive. Certain materials, e.g. polystyrene and polyvinylacetate, exhibit variable surface reactivity with different blood samples, while others, e.g. pyrolytic carbon and PMA, produce a uniform response. A general trend appears to be less surface

reactivity with decreasing glass transition temperature. Chemical modification of polymers and production of copolymers has been undertaken to define the nature of reactive sites. For example, acrylonitrile and aminomethylacrylate copolymers of PMA exhibit more reactivity than PMA.

Sepharose 4B (4% agarose gel beads) appears essentially non-reactive. However, Sepharose with covalently bound heparin promotes extensive platelet adhesion. Pretreatment of this surface with increasing amounts of plasma, but not albumin, produces decreasing surface reactivity.

J. L. Brash and I. A. Feuerstein (McMaster Univ., Hamilton, Ont., Canada): **Kinetics of Platelet Adhesion to Artificial Surfaces in Vitro.** **(184)**

Adhesion of platelets to glass, collagen-coated glass, albumin-coated glass, polystyrene, sulfonated polystyrene and a segmented polyurethane, has been studied in vitro. The apparatus is of the Couette flow type and allows close control of fluid shear and diffusional factors. Suspensions of washed pig platelets constitute the basic platelet medium. This can be modified by adding back red cells and specific plasma proteins in varying concentration and the platelet concentration can be varied without compromising viability. Adhesion is measured by radiolabelling methods.

In the absence of red cells, low levels of adhesion were seen on all surfaces with saturation occurring at 4 to 6 platelets/1000 μ^2 in 2 to 4 minutes. In the presence of red cells adhesion was much greater. Collagen was the most reactive surface and adhesion data was consistent with a platelet diffusivity 10 to 100 times that predicted by Brownian motion. The diffusivity was dependent on shear rate and hematocrit. All other surfaces showed a 2-fold increase in adhesion compared to the values without red cells. However adhesion was independent of hematocrit above 10% and reached a constant value of about 12 (less for albumin monolayer) in 2 to 10 minutes.

H. Lagergren, R. Larsson, P. Olsson, K. Rädegran and J. Swedenborg (Surgical Research Laboratory, Thoracic Clinics, Karolinska Sjukhuset, and Aminkemi AB, Stockholm, Sweden): **Decreased Platelet Adhesion as a Characteristic of Non-Thrombogenic Heparinized Polymer Surfaces.** **(185)**

Non-thrombogenic surfaces with a stable heparin layer were made of ionically bound heparin cross-linked with glutardialdehyde.

In vivo the retention of platelets on heparin coated and untreated arterio-venous plastic shunts were compared. The retention of ^{51}Cr labelled platelets on treated surfaces was less than 5% of that on untreated surfaces. The results were not influenced by systemic heparinisation or fibrinogen depletion.

In vitro heparinized blood was rotated in coated and nontreated tubes. The retention of platelets on treated tubings was less than 0.5% of that on non-treated tubings. The heparin concentration of blood did not influence this platelet retention.

It is concluded that the non-thrombogenic capacity of the heparinized surface is due to inhibited platelet adhesion.

A. Carpentier, J. Relland, S. Carpentier, A. Lessana, J. N. Fabiani, A. Deloche, S. Chauvaud, L. Schahmaneche and G. Gory (Laboratoire d'Etudes de Greffes et Prothèses Cardiaques. Institut Biomédical des Cordeliers. 15, rue de l'Ecole de Médecine, 75006 – Paris): **Tissue Antithrombogenic Inducing Factor: Experimental evidence and practical applications in the construction of cardiac valves and the artificial heart.** **(186)**

Thrombo-embolic complications remain the major problem in human organ support and replacement devices: artificial kidney, artificial heart, cardiac valves. Based on our 7 year experience with heterograft valves which did not present thrombo-embolic problems, we postulated the existence of an anticoagulant factor inherent in tissue valves and in the biopolymers extracted from these valves.