LYMPHOCYTES


A specially constructed apparatus has made it possible to reinfuse lymph and lymphocytes continuously from the thoracic duct into the femoral vein of the male albino rat (140-200 Gm.). Quantitative studies indicate that lymphocytic output is independent of the rate of lymph flow and body weight. The hourly output of cells was fairly constant during the first day, but a sharp fall occurred during the third and fourth days. Neither cell-free lymph nor killed lymphocytes prevented this fall, but the continuous intravenous re-infusion of lymph and living lymphocytes did. Apparently the entrance of living lymphocytes into the blood is essential for maintaining the output of lymphocytes from the thoracic duct. Lymphocytes spend about 2 hours or less in the peripheral blood.—O. P. J.


Splenectomized rats were injected intravenously with a large amount of suspension of lymphocytes which were obtained from the thymus and mesenteric lymphnodes of adult rats, and which were labelled with $^{32}$P. When the lymphocytes were injected into the femoral vein, the majority of lymphocytes was blocked in the lung capillaries at first, and later they were transported to the liver and accumulated there to a large extent. When they were injected into the mesenteric vein, they were arrested by the liver directly. Although a relatively large amount of injected lymphocytes entered the bone marrow and the small intestine, few seemed to accumulate there. The injected lymphocytes were deposited only in very small amounts in the lymphoid organs and in the kidney.—K. M.
PLATELETS


The importance of a dialysable plasmatic co-factor and of thrombin for clot retraction was confirmed on the basis of further experiments, which were carried out according to the first investigations of Lüscher. Glucose seems to play an important role in this connection, and clot retraction itself should depend, up to a certain extent, on glucose catabolism.—P. d. N.


Clot retraction was evaluated in human plasma clotted with citrate-activated thrombin in the presence of calcium ion, to which various test reagents were added. Under these conditions, plasma collected in glass gave retracting clots, whereas silicone-processed plasma did not, despite a similar lack of intact platelets in either. Dialysis of glass-collected plasma removed its ability to undergo clot retraction. In dialyzed plasma, clot-retracting ability was restored by the addition of serotonin, plasma dialysate and platelets ruptured by freezing and thawing. Dialyzed ruptured platelets were ineffective. The ability of glass-collected plasma to yield retracting clots was also abolished by adsorption with barium carbonate. Retraction was restored by an eluate from the adsorbing agent, whereas serotonin and plasma dialysate were ineffective. When plasma was subjected to both adsorption and dialysis, there was no clot retraction and the clots were transparent instead of being typically opaque. Opacity was restored by any of the agents previously noted to be retraction-active, but retraction was restored only by a combination of eluate with serotonin or dialysate. Glycerol added to the system abolished clot retraction and produced transparent clots.

It is concluded that clot retraction follows the interaction of serotonin (or related compounds) and a protein found in the barium carbonate eluate. The latter is not antithrombin I or II, since purified preparations of these agents proved to be inert.—T. H. S.


In the thromboplastin generation test, excessive concentrations of fresh or lyophilized platelets were found to be inhibitory. The optimal concentration of platelets approximated their physiologic level. Similar inhibitory effects were obtained with certain platelet extracts; and all these preparations had a parallel effect in the recalcified clotting time of plasma.

Since no concentration of platelets produced excessive activation of thromboplastin, it was suggested that platelet concentrates would not represent a thrombotic hazard when administered in vivo.—T. H. S.


The authors consider platelet co-factor I to be synonymous with antihemophilic factor. The basis of purification is similar to that described by Lorand and Laki, and depends upon adsorption with subsequent elution from kaolin. An additional step is selective pre-
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precipitation with ammonium sulfate. The product obtained has two peaks in the ultracentrifuge, but only one by electrophoresis. Activity was labile at the extreme pH ranges and at temperatures over 30°C. It was not contaminated by fibrinogen.—T. H. S.


Purified clotting factors were prepared by methods previously described at the authors’ laboratories. Silicone whole blood and recalcified plasma clotting times of hemophilic specimens were improved by platelet factor 3 more efficiently than by platelet co-factor I. A combination of these two reagents had intermediate activity.—T. H. S.


A turbidimetric method for the evaluation of the osmotic resistance in platelets is described. Hypotonic solutions of sodium chloride are employed as for the determination of the osmotic resistance of red cells. The evaluation of the thromboplastic activity released from the platelets and thrombelastographic determinations were carried out. In blood diseases, without involvement of platelets, no significant variations were observed. In thrombocytopenias with hypoplasemia, a decrease of the minimal resistance and an increase of the maximal resistance were detected. In thrombocytopenias an increase of the maximal resistance might be observed.—P. d. N.


The previously described technic (mixed thrombocyto-erythrocytic agglutination) was applied to the detection of M and N agglutinogens in the platelets.—P. d. N.


Electron microscope preparations were made on formvar-coated wire loops by dipping them in freshly drawn venous whole blood for 2 to 5 seconds. They were examined in a Philips EM 75 after washing in water, fixing in 2% buffered osmic acid, washing again and then drying in air. The platelets in serial preparations showed a progressive change from relatively small compact forms with few processes to larger more spread-out forms with more processes, and the number of platelet clumps increased. Platelets disintegrate before clot formation begins. The fibrin fibrils seem to form at some distance from the platelets with no evidence that platelet processes form them. Platelets in preparations from the blood of a patient with pancytopenia were morphologically different from all others. —O. P. J.


Normal serum contains a platelet-like property stimulating the formation of thrombin in a platelet-free or thrombopenic plasma (O’Brien). The authors have measured the kine-
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Tics of prothrombin consumption in a mixture of normal, platelet-free plasma with diverse normal and pathologic sera. This has suggested a new test to measure the platelet activity of serum (S.P.A.); details and findings in 204 patients with hemorrhagic disorders are presented. The test is sensitive and narrowly specific for platelet abnormalities, whether thrombocytopenic or not. The nature of the platelet factor and its relationship to platelet prothromboplastin are discussed. The S.P.A. test measures the residual prothromboplastin activity which is always grossly abnormal when this factor is absent from the platelets.—J. D.


The influence of platelets on the maximal-amplitude values of thrombelastogram (maximal amplitude) can be evaluated under pathologic conditions by comparing the thrombolastograms in platelet-rich plasmas and platelet-poor plasmas. The index thus obtained might represent a useful criterion for characterizing the platelet functional activity, particularly in the so-called thrombocytopenias.—P. d. N.

CONCENTRATION OF PLASMA COAGULATION FACTORS ADSORBED ONTO HUMAN PLATELETS. Y. Bounameaux. From the Laboratoire pour l’étude de la coagulation sanguine and the Clinique médicale de l’Université, Zurich, Switzerland. Rev. franc. études clin. & biol. 2:52–63, 1957.

Several plasma coagulation factors are adsorbed on platelets. Their presence can be demonstrated even after repeated washings, up to 8 times. Prothrombin, factor V, factor VII (S.P.C.A.), antihemophilic globulin (AHG), P.T.C. and Koller’s factor X were detected in various amounts. Their concentration is inversely proportional to the number of the washings, within certain limits. The theoretical and practical implications of these findings are discussed. It is particularly pointed out that the usual platelet suspensions, which are employed in the coagulation tests, cannot be considered as completely free of plasma constituents.—P. d. N.


The hypothesis is advanced that “immune” thrombocytopenic purpuras may develop as a result of antigenic changes in platelets induced by certain drugs and infections. An attempt was therefore made to reproduce this pathogenesis in rabbits. Animals were immunized with human platelets, platelets of other rabbits, and their own platelets variously treated. To achieve antigenic modification, platelets were incubated with the drugs quinidine sulfate, quinidine hydrochloride and aminopyrine; with filtrates of beta-hemolytic streptococci, staphylococcus aureus and albus; and with the B-1 strain of Newcastle virus.

Rabbits immunized against human platelets developed antibodies which failed to distinguish between untreated and modified platelets. In these animals iso- and autoimmunization did not occur. In isoimmunized rabbits no antibodies were demonstrable by in vivo technics. However, some animals immunized with modified platelets developed thrombocytopenia of mild degree when injected with the respective platelet-modifying agent. The sera of such animals given with the modifying agent to donor animals likewise resulted in some recipient thrombocytopenia. Autoimmunized rabbits developed platelet auto- and isoagglutinins. Although none of these animals became thrombocopenic, platelet reduction sometimes followed injection of the agent used in antigenic modification.

The authors suggest that antibodies against antigenically altered platelets may also act
against normal platelets, and thus explain the development of idiopathic thrombocytopenic purpura.—T. H. S.


The authors suggest a new procedure for the evaluation of antiplatelet antibodies in vitro. If antiplatelet sera, which are obtained by means of injections of human platelet suspension into rabbits, are added to normal, platelet-rich human plasmas, there is an inhibition of the maximal amplitude of the thrombelastogram, as a result of an inhibition of that platelet activity which is responsible for the maximal amplitude. It is also possible to obtain similar results by adding the antiplatelet sera to platelet suspensions in saline and to evaluate the functional activity of the platelets before and after the addition of the sera by adding the suspensions to platelet-poor normal plasma and performing a thrombelastographic determination. The results thus obtained are often more definite than those of the platelet agglutination tests.—P. d. N.


Immunohematologic data upon leukocytes and platelets are reviewed, especially the technics used to detect leukocyte and platelet agglutinins. Leukoagglutinin was studied by the technic of Dausset; the platelet agglutinin test was developed by the authors. In 13 patients with disseminated erythematous lupus, 6 exhibited a positive leukocyte agglutinin test; 27 out of 76 different hemopoetic disorders gave a positive leukoagglutinin test. Three peculiar cases with active leukocytic agglutinating sera were observed: the first serum was able to agglutinate every sample of several different leukocyte suspensions, but it was unable to agglutinate the leukocytes of its own blood; the other two were cases of sera acting specifically at low temperatures (4° C.), "cryoleukoagglutinins."

In reference to the immunologic thrombocytopenic purpura, the "drug purpuras" are considered as well as the technics to be used. With technic employed while 100 blood donors gave negative agglutination tests, 7 out of 22 cases of thrombocytopenic idiopathic purpura (31%), 1 out of 10 cases of nonthrombocytopenic purpura, 1 out of 12 cases of acute leukemia, 1 out of 9 cases of chronic myeloid leukemia, 3 out of 9 cases of acute disseminated lupus, 2 out of 5 cases of idiopathic aplastic anemia and 2 out of 9 cases of acquired hemolytic anemias gave positive platelet agglutination tests. In 7 cases of Hodgkin's disease, 3 cases of reticulosarcoma and 32 other cases of different kinds of hemopathies none was positive.—M. A. J.
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