ERYTHROCYTES


Using $^{59}$Fe in rats made polycythemic by intermittent hypoxic hypoxia, the authors studied the erythropoietic activity after treatment with cyclic AMP (cAMP) or its dibutyl cyclic AMP (db-cAMP). Both compounds gave a significant erythropoietic effect, which was proportional to the injected dose (1-5 mg per animal). The administration of rabbit immune serum neutralizing erythropoietin (Ep) completely inhibited the erythropoietic action of db-cAMP. More elevated quantities of immune serum were capable of reducing erythropoiesis to levels lower than those found in untreated control animals. From this finding it was inferred that the effect of cAMP and db-cAMP is mediated by an endogenous erythropoietic hyperactivity. The latter is most likely determined by an increase in Ep production rather than by an effect on kinetic or erythropoietic activity of Ep. In fact, db-cAMP potentiates the erythropoietic action of a hypoxic stimulus, but not that of exogenous Ep. These investigations suggest that cAMP most likely plays a role in the mechanism of Ep production. — G.L.


Using the uptake of $^{59}$Fe in the peripheral blood of rats with polycythemia induced by intermittent hypobaric hypoxia, the authors studied the level of erythropoietin after treatment with E$_1$ prostaglandin (PGE$_1$). This compound had a significant erythro-stimulating
effect, which was proportional to the injected dose (10-100 micrograms per animal). The administration of rabbit immune serum neutralizing erythropoietin (Ep), completely inhibited the erythropoietic action of PGE₁. More elevated quantities of immune serum were capable of decreasing erythropoiesis to values which were lower than those in untreated control animals. From this it could be inferred that the PGE₁ effect is mediated by an endogenous erythropoietic hyperactivity. The latter is most likely determined by an increased production of Ep rather than by an effect modifying the erythropoietic action of Ep. In fact, PGE₁ potentiates the erythropoietic action of a hypoxic stimulus, but not that of exogenous Ep.—G.L.


Hepatic heme oxygenase levels of newborn Sprague-Dawley rats were 2.7 times normal adult levels, while splenic heme oxygenase levels were half normal. During the first wk of life, hepatic heme oxygenase rose 50%, while splenic levels fell by 50%. From day 7 to day 30 enzyme activity progressively moved toward normal adult values. Food deprivation of fetus or newborn led to significant increases in hepatic enzyme activity, but did not affect levels of splenic heme oxygenase.—J.B.S.

A New Type of Hereditary Nonspherocytic Hemolytic Anemia Due to Phosphofructokinase Deficiency. S. Miwa, T. Sato, H. Murao, M. Kosuru, and H. Ibayashi. Yama-guchi University School of Medicine, Ube and Faculty of Medicine, Kyushu University, Fukuoka, Japan. Acta Haematol Jap 35:113–118, 1972.

Phosphofructokinase (PFK) deficiency was first described by Tarui et al. in 1965, followed by Layzer et al in 1967. Although the main clinical features are indistinguishable from muscle phosphorylase deficiency, all of their patients had, in addition, a mild nonspherocytic hemolytic disease. Whereas the skeletal muscle has a profound PFK deficiency, the erythrocytes are found to have about half the normal enzyme activity. In 1969, Waterbury and Frenkel described a case with hereditary nonspherocytic anemia which also had about half the normal red cell PFK activity. However, Waterbury’s case did not show muscle dysfunction. This communication briefly reports a case with hereditary nonspherocytic anemia due to an apparently new type of PFK deficiency. The erythrocytes of the patient showed marked PFK deficiency, and the patient had no muscle dysfunction. Both of her two children had about half normal red cell PFK activity.—K.F.

Properties of the Unstable Hemoglobin Vienna. E. Kleihauer and K. Betke. Pediatric Department of the University and Pediatric Clinic of the University, Munich, Germany. Klin Wochenschr 50:907, 1972.

Hb–Vienna, a fast moving, unstable hemoglobin was first described in 1966 by Kleihauer, Betke, and Pietschmann. The authors now report on some properties of this hemoglobin. The methemoglobin spectrum was only slightly different from HbA₁ and methemoglobin formation resembled that of normal hemoglobin. On heat denaturation, Hb–Vienna was as stable as HbA₁. HbA₂ was within normal limits and HbF was increased.—K.P.


The pattern of fatty acids in the red cell phospholipids of newborns differs from that of adults, but is not different in erythroblastotic infants contrasted with their healthy peers. The difference appears to result from deficiency of essential fatty acids in the fetus, perhaps induced by a placental permeability barrier, or by poor protein binding.—J.B.S.


Acute hemolytic anemia induced by ingestion of fava beans, was seen in five girls heterozygous for the Mediterranean type of G-6-PD deficiency. The severity of the anemia was proportional to the degree of enzyme defect.—J.B.S.

In three siblings with both hereditary spherocytosis and SS disease, the peripheral blood showed a predominance of spherocytosis and only a few target cells and moderate numbers of sickled red cells. Significant anemia appeared early in two, associated with neonatal icterus in one. Fetal hemoglobin concentrations were higher than usually seen in children with sickle cell anemia, ranging between 13% and 30%. Two of the three developed massive splenomegaly, and evidence of "hypersplenism," the signs of which were reversed by splenectomy in one, and by transfusion therapy in the other. —J.B.S.


The authors evaluated techniques capable of detecting sickle cell anemia (SCA) in cord blood samples. They concluded that the classical sickling test, the modified Itano solubility test, and paper electrophoresis, were valueless, while cellulose acetate, agar gel, and starch gel electrophoresis were capable of identifying SCA in neonates. The agar gel appeared to be the most suitable medium for delineating HB SS Disease, while paper electrophoresis was a good screening technique for demonstrating the presence of Hb S.—J.B.S.

LEUKOCYTES


The levels of NANA and of proteins were determined in the serum of patients with myeloid and lymphocytic leukemias and in a control group of healthy subjects. It was demonstrated that in 18 untreated cases of myeloid leukemia the serum level of NANA was significantly increased (mean 140 mg/100 ml) if compared with controls (40 healthy people, mean 80 mg/100 ml) but returned to normal values after treatment with corticosteroids and cytostatic agents. On the contrary, in lymphatic leukemias the level of NANA did not differ from that of controls (82 mg/100 ml). The authors conclude that changes in NANA level may be considered as a biochemical test for differentiation of micromyeloblastic leukemias from lymphocytic and lymphoblastic leukemias as well as an index of the dynamics of the disease in cases of myeloid leukemia. —M.K.


The authors report studies on two cases of idiopathic myelofibrosis in children. From their observations and from review of 24 cases in the literature, it is concluded that idiopathic myelofibrosis in childhood shows a more rapid and acute course than in the adult age with remarkably malignant aspects of bone marrow cell morphology. —G.L.


Vincristine, vinblastine and cyclophosphamide were used singly, or sequentially, in the treatment of infants and children with generalized histiocytosis X. The complete remission rates were 50%, 20%, and 36%, respectively; the complete plus partial remission rates were essentially the same for each drug ranging between 50% and 63%. Patients unresponsive to one drug were not infrequently responsive to another. —J.B.S.

Remissions, with a median duration of nine wk, were obtained in half the children receiving L-asparaginase, either at a dosage of 200 I.U./kg/d x 14 or 5000 I.U./kg b.i.w. x 2 wk. Aside from changes in liver function tests, which occurred in all patients, significant toxicity was limited to the high intermittent dosage group. These results differ from those reported by Pratt et al. (J Pediat 77:474, 1970) and may reflect the fact that the total cumulative dose in that series was greatest for the daily treatment regimen, while in this study, the total cumulative dose was much greater in the children treated twice weekly.—J.B.S.


An electroencephalographic investigation was carried out in 20 children presenting acute lymphoblastic leukemia and submitted to prophylaxis with intrathecal methotrexate. Clear alterations of the electroencephalographic tracing were observed in 18 of the 20 children, when the examination was performed 24 hr after methotrexate introduction. In nine children, with a normal initial electroencephalographic picture, widespread alterations were noted in four and focal ones in five. In the remaining patients with electroencephalographic abnormalities existing before methotrexate introduction, a worsening of the initial picture was observed. The electroencephalographic examination carried out every 24 hr for 10 days revealed a slow, progressive reversion to the initial picture.—G.L.

L-Asparaginase in Acute Leukemia Refractory to Treatment. J. Japa, E. Kardaszewicz, and S. Kowalski. First Department of Internal Medicine, Silesian School of Medicine, Katowice, Poland. Pol Arch Med Wewn 48:627-631, 1972.

In 12 patients with various types of acute leukemia refractory to cytostatic drugs L-asparaginase treatment was applied. Complete remission was obtained in two cases and partial in seven. No improvement was observed in 2 patients. The longest duration of remission after L-asparaginase was 5 mo. Hypoalbuminemia, hypo frightening damage were the most frequent complications (seven cases). These symptoms disappeared quickly after withdrawal of L-asparaginase.—M.K.


Seventy-five patients, adults and children, were treated with cytosine arabinoside intravenously (2 mg/kg, slow infusion), orally (4 to 5 mg/kg, for 8-10 days, followed by other drugs: 6-MP and or MTX) and intrathecally. More than 100 courses were given. In 55 patients, remissions were observed in 70% of cases (41% complete and 21% partial in myeloid leukemia; 29% and 52%, respectively in lymphatic leukemia). The survival was prolonged also in patients who had become refractory to other treatments. The oral route was used above all for maintenance treatment, after a first remission was obtained.—P.dN.

HEMOSTASIS


Factor VIII purified by gel chromatography, using normal cryoprecipitate as starting material, corrected the abnormal platelet retention in Von Willebrand's disease. Specific precipitating anti-factor VIII sera reduced the platelet retention of normal blood. This, and the formation of only one precipitin line in immunodiffusion and immunoelectrophoresis, suggest that the antigenic determinants of factor VIII and correcting substance are similar. This implies that normal plasma contains a bifunctional molecule with both factor VIII and correcting activity or that both activities are located on different molecules which were not separated under the experimental conditions.
Purified factor VIII related antigen from hemophilic plasma corrected also the abnormal platelet retention in Von Willebrand's disease. The detection of an antigen related to factor VIII in classic hemophilia gives further evidence that the lack of factor VIII activity is due to a structural defect. It is suggested that the synthesis of both factor VIII and correcting factor is reduced in Von Willebrand’s disease. — K. P.


Patients with F VIII or F IX deficiency who had oral bleeding (dental or nondental) were treated with a primary dose of appropriate coagulation factor-rich material, then, with EACA 100 mg/kg q.i.d. for 5 to 10 days. With one exception, hemostasis was excellent, without repetition of plasma therapy. — J. B. S.


This is an accurate experimental study (method of Harada and Zucker) of the release of the antiheparin platelet factor after ADP-induced platelet aggregation. The release took place during the irreversible phase of aggregation and was independent of incubation temperature. — P.dN.


During platelet aggregation induced by serotonin there was no release of platelet antiheparin factor, whatever serotonin concentration, incubation time and temperature was used. Antihemiparin factor was studied according to Harada and Zucker. The results were interesting for the evaluation of the interrelationships between serotonin and antihemparin factor: the latter was released during the irreversible phase of platelet aggregation ("release reaction"), and various agents, like ADP, thrombin, catecholamines, collagen should be concerned with this reaction. Serotonin was able to produce a limited, reversible aggregation, and this may explain why no release of platelet antiheparin factor takes place following the action of serotonin on platelets. — P.dN.


The incidence of deep vein thrombosis was studied using the $^{125}$I fibrinogen test in 42 patients admitted to an intensive care unit with acute myocardial infarction. Deep venous thrombosis developed in 3 of 27 patients treated with anticoagulants and in 8 of 15 not so treated. Evidence of thrombosis at the site of an indwelling intravenous cannula was found in 75% of the patients. — A. A. M.


Human plasminogen obtained by affinity chromatography was activated by insolubilized streptokinase. $^{125}$I labeled material and DFP inactivated plasmin were also prepared. All these preparations were mixed with purified human alpha$_2$-macroglobulin and the reaction mixtures studied by radioautography and immunoelectrophoresis. The results were in favor of the concept that the initial phase of interaction between the protease and its inhibitors is proteolytic in nature. — J. C.


Unbound unconjugated bilirubin, in contrast to albumin-bound bilirubin, or light-treated bilirubin, causes yellow staining and aggregation of washed platelets at low (0.5 mg/100 ml) levels. In platelet-rich plasma these changes were much less evident, even at bilirubin concentrations above 10 mg%. Bilirubin-induced platelet aggregation was calcium dependent and enhanced by presence of potassium ions, and was accompanied (preceded) by ADP and ATP
release. These effects of bilirubin on platelets may, in part, explain the hemorrhagic tendency seen in neonates with severe hyperbilirubinemia. The effect of bilirubin on platelet aggregation probably depends upon free bilirubin, since large amounts of bilirubin bound to albumin appear to inhibit platelet aggregation.—J.B.S.


The shortened platelet survival noted in patients with hydrocephalus and indwelling silastic catheters appears to be reversible by the administration of aspirin plus dipyridamole.—J.B.S.


One of two brothers (2 ½ years of age) with eczema, early onset of thrombopenia and immune responses which were not entirely suggestive of Wiskott-Aldrich syndrome, underwent splenectomy, and subsequently had two episodes of severe pneumococcal disease, succumbing to the second. The authors, after a review of the literature, suggest that when sex-linked congenital thrombopenia is seen, splenectomy should be approached with great caution.—J.B.S.


Using platelets from a case of Bernard-Soulier syndrome (large, heavy, and decreased in number) isolated on albumin gradient after Nicholson and Hampton, the authors have shown that these platelets do not respond to rifocetin, an antibiotic which is known to interfere with Von Willebrand factor (Howard, Weiss, Meyer, Hutton). The interpretation they give is that "Bernard-Soulier platelets" (probably due to their decrease in sialic acid) are unable to adsorb Von Willebrand factor. As shown by Bishell, these platelets do not respond to bovine fibrinogen or bovine Factor VIII but they do undergo shape changes in the presence of ADP. A new concept of the physiology of primary hemostasis is given in which platelet membrane (abnormal in Bernard-Soulier syndrome) and plasmatic Von Willebrand factor (decreased in Von Willebrand disease) are the main components reacting with a vascular component (? microfibrils). The prothrombin consumption test, abnormal in Bernard-Soulier syndrome, is not related with a decrease in platelet F-3 (which is increased), but probably to an abnormal activation of clotting factors around the abnormal platelets. In regard to these new ideas, further work on the composition of the platelet membrane in Bernard-Soulier syndrome is warranted.—J.C.


The authors studied the effect of anticoagulants and some products that affect platelet aggregation upon the conservation of platelet populations obtained by analytical ultracentrifugation with discontinuous sucrose gradients. Collections of blood with ACD A or with EDTA seemed to be equivalent since the platelet populations were similar. An hypercitrated medium gave a more important proportion of heavy platelets: but, if in excess, this medium made the platelets lighter. The authors studied two inducers of aggregation: without agitation, ADP had no effect upon platelet populations, while adrenaline decreased the populations of heavy platelets. The authors found many differences in the effects of inhibitors of aggregation: adenosine had no effect, but NEM decreased the heavy platelets and increased light platelets. Adenosine can protect this drastic effect of NEM, but this protection is not long lasting.—J.C.

Influence of Lithium on Aggregation, Release Reaction and other Functions of Human Platelets. P. Geerdink, S. Levy-Toledano, H. Wessels, J. Caen, and C. Haanen. Division of Hematology, Department of Internal Medi-
Preincubation of platelet suspensions with lithium chloride increased the intensity of aggregation and prolonged the disaggregation phase. These phenomena were observed with the aggregation inducers ADP, thrombin, and serotonin. The aggregation induced by collagen showed after preincubation of platelets with LiCl, an increase in velocity as well as in intensity. Preincubation with LiCl was an essential prerequisite of the potentiation of aggregation; in effect, without incubation, LiCl inhibited the aggregation process induced by thrombin. Sodium and potassium chloride in the same concentrations had no effect on the aggregation process. LiCl exerted no influence on ADPase and adenosine deaminase activities in plasma. The effect of LiCl was due to an action on the platelet itself as was shown on gel-filtered platelets. The inhibitory effect of various aggregation inhibitors was counteracted by preincubation of platelets with LiCl. LiCl augmented the PF3 availability, and apparently, did not change the ADP-release. The 4C-adenosine incorporation of platelets was not influenced by LiCl. Platelet populations were not changed by LiCl. Lithium restored to normal the disturbed aggregation in cases of abnormal release thrombopathia, uremia, after aspirin ingestion, and von Willebrand's disease, but not in thrombasthenia. In vivo lithium normalized the disturbed aggregation in cases of thrombopathies with impaired release reaction. The observed effects of lithium could be due to an inhibitory action on the cAMP forming enzyme adenylylclase.—J.C.

**IMMUNOHEMATOLOGY**


The results of serological investigations carried out in six patients with immunohemolytic anemia are reported. In all cases direct Coombs' test was negative while i 5 autoantibodies belonging to IgG class were detected. The authors present the evidence that the negative Coombs' test results not always from too low number of immunoglobulin molecules adsorbed on erythrocytes and describe a method for obtaining a positive Coombs' test in such cases in which this test examined by the routine technique is negative due to inhibition by the presence of anti-Gm\(^4\) antibodies and anti-antibodies in the serum. Selected human serum containing anti-antibodies from a patient with plasmacytoma was used in the method described instead of animal antiglobulin sera. The authors suggest that the negative results of the direct Coombs' test examined by the routine technique may at times depend on coating of erythrocytes by denatured immunoglobulins.—M.K.

The Immunochemical Studies of M-N Blood Group Glycoproteins from Human Erythrocytes. E. Lisowska. Department of Immunchemistry, Institute of Immunology and Ex-

The author reports the results of a large series of investigations on isolation, purification, and immunologic properties of sialoglycoproteins from human erythrocytes. It was demonstrated that sialoglyco-proteins which can be isolated from erythrocytes without cleavage of covalent bonds possess high tendency to aggregate and to complex with other components of the cell membrane. Along the peptide chains of these sialoglycoproteins, uneven distribution of two types of carbohydrate side chains could be recognized: those that are alkali-labile and contain sialic acid, galactose and galactosamine; the other that are alkali-stable composed of galactose, glucosamine, mannose, fucose, and a rather small amount of sialic acid. The structure of tetrasaccharide identified with the majority of alkali-labile chains was established. It was found that sialoglycoproteins of erythrocytes membrane contain immunodeterminants of blood groups M and N, receptors for phytoagglutinins and for influenza viruses. After selective cleavage of sialic acid residue, two additional immunologic activities appeared, namely the reactivity with anti-I-antibodies and with anti-AHP agglutinins from H. pomatia. MN and Nv activity seemed to be connected with alkali-labile, while I activity was connected with alkali-stable carbohydrate chains. Blockage of amino groups in sialoglycoproteins modified the MN activity but remained without influence on their other examined serologic properties. M.K.


Three hemophiliacs with a history of multiple plasma product transfusions suffered severe pulmonary reactions after transfusion of fresh frozen plasma. In one patient the reaction was fatal and the chest radiographs of all three patients showed widespread patchy rounded opacities resembling bronchopneumonia. Treatment, including intravenous frusemide and hydrocortisone, lead to complete clinical and radiographic resolution in two of the three patients. There was evidence for the transfer of white cell and Gm antibodies from donor to recipient in two of the three patients. The authors recommend that hemophiliacs with a history of transfusion reaction should only be treated with concentrated preparations of factor VIII and not with fresh frozen plasma although only one of their patients had such a history.—J.A.W.


Lymphocyte-poor, stem cell-enriched bone marrow suspensions were obtained in animals and man by discontinuous albumin-gradient centrifugation. In mouse marrow, a 10- or 20-fold enrichment in stem cells could be obtained as compared to the normal tissue. In several animal species, it was possible, in addition, to decrease by a factor of 10 or more the proportion of immunocompetent cells, indicating that on the basis of a fixed number of stem cells, the risk of producing acute graft-versus-host (G-v-H) disease was decreased by a factor of 100 or more. This method may be used (1) in severe combined immune deficiency disease, normally accompanied by high susceptibility to acute G-v-H reactions. Ten infants received stem cell enriched fractions. Among the six who survived the 2-wk period after grafting, two were cured, three died from infection on day 45, 51, and 70, respectively but with definite signs of graft take, and one is still living 2 yr after grafting although the graft was eventually rejected; (2) in aplastic anemia, the procedure should be considered in children and will benefit from the recently described method of storing bone marrow stem cells (see Schaefer et al. Rev Europ Etud Clin Biol 17:481, 1972).—J.M.P.


Another demonstration of the “functional asplenia” of children with sickle cell anemia is their inability to respond to intravenous
antigenic stimulation with sheep erythrocytes. Response to intramuscular injection was not deficient, nor was splenic clearance of radio-colloid (99mTc-sulfur).—J.B.S.


In children with SLE, positive immunofluorescence reaction of kidney biopsy specimens with fluorescein isothiocyanate-conjugated goat antihuman IgG, and anti B, C, may be demonstrated well before onset of other laboratory or clinical evidence of lupus nephritis.—J.B.S.


In 22 patients with multiple myeloma (diagnosis based on clinical, radiological and bone marrow findings) γG and γA dysproteinemias were evidenced by means of immunological methods. Radioimmunoelectrophoresis of the fluid from bone marrow cultures was carried out in 19 cases, and of fluid from peripheral blood lymphocytes in 14 cases. Combined treatments with Prednisone, endoxan (cyclophosphamide) and alkeran (melphalan) were applied. After treatment, a reduction of the in vitro production of the concerned gamma globulin (identical to that present in excess in the serum) was observed. The production of other gamma globulins, not concerned with the myelomatous process, was reduced in the lymphocyte cultures, and this probably accounted for the reduced antibody level in these patients and for their increased sensitivity towards infections.—P.dN.

During an electron-microscopic study of the myeloma cell, a possible mode of immunoglobulin secretion via vesicles was observed. The probable sequence of secretion was as follows: First, secretory vesicles of varied sizes appeared at the periphery of the cytoplasm, then their inner material became gradually more electron dense. Next, these vesicles moved to the outside of the cell, then, their content became lower in electron density than when located in the cytoplasm, suggesting its moving-out into the blood. Cytoplasmic dense bodies, which were usually seen in large numbers in myeloma cells and often accompanied with the above-mentioned vesicles, might play some role in the completion of the immunoglobulin. Cell lysis, as seen in a holocrine cell, was also considered to be another probable mode of secretion of IgG, especially when both the myeloma cells with a highly dilated, cyst-like endoplasmic reticulum in the bone marrow, and a marked increase in IgG in the serum were seen.—K.F.


Serum and urine CSF levels were estimated from the mean number of granulocyte and/or macrophage colonies from a standard femoral marrow suspension cultured in agar. Tests were also used to assay inhibitors of CSF. It was shown that in approximately half the patients with renal disease, serum levels of CSF were elevated, yet urine levels were normal or subnormal. The failure to excrete CSF, a glycoprotein appeared to be selective, as the urinary CSF activity per unit of protein was much lower in the patients than in normals. These results suggest a possible mechanism for the increased myeloid activity which may be found in chronic uremia, although associated infections may be contributive in some cases.—J.M.P.

ABSTRACTS

Fine structures of human spleens in normal subjects, patients with Banti's syndrome (idio-pathic portal hypertension) and patients with cirrhosis of the liver were studied by scanning electron-microscopy. Three-dimensional fine structures of normal human spleens observed by this method were comparable to those obtained by transmission electron microscopy of comparable human spleens and also to the fine structures of animal spleens described by Miyoshi et al, although there were minor differences in the structures of venous sinuses between human and animal spleens. The architecture of the red pulp of spleens in Banti's syndrome and cirrhosis of liver was very different from normal. The abnormal findings observed in both types of pathologic spleens appeared to be essentially identical, although minute quantitative differences were noted.—K.F.


Using a finely dispersed emulsion derived from a lipiodol derivative, it was possible to obtain good quality tomograms of the spleen after injection in the splenic artery. A total dose of 1.5–2.5 ml/kg (0.13 to 0.23 g iodine/kg) was employed. Fever and chills were noted in four cases out of 22, 3 to 6 hr after the injection. A spleen infarct could be recognized as a triangular radiolucent area, whereas Hodgkin lesions, later confirmed histologically, could be detected in two cases.—J. M. P.


A review of current information and speculation regarding HUS, and a discussion of the diagnosis and management thereof. Nicely done.—J. B. S.

NEWS AND VIEWS

RESEARCH FELLOWSHIPS IN HEMOPHILIA

The National Hemophilia Foundation announces its 1974 competitive fellowships in clinical and basic research for studies relating to problems of hemophilia. Postdoctoral applicants engaged in or planning research in areas and disciplines related to hemophilia are eligible. The fellowships are offered through medical schools on an annual basis starting with the academic year July 1, 1974.

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A more complete description of the contents may be found in “The Availability of Translations and Reproductions of Scientific Treatises in Blood Group Immunology,” (Transfusion 7:201, 1967).

HEMOGLOBINS SYMPOSIUM

ABSTRACTS