ABSTRACTS

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ERYTHROCYTES


It is known that with hemoglobin Lepore (Hb Lepore) trait there is mild anemia, hypochromia, and microcytosis, similar to that seen in heterozygous β-thalassemia, while with homozygous Hb Lepore there is a severe anemic disorder similar to that with homozygous β-thalassemia. Studies are here reported of three patients with Hb Lepore trait studied. These findings were similar to those seen in patients with heterozygous β-thalassemia. There was also a relative instability of the synthetic mechanism for normal beta chain in these patients. In fact, the results indicated that the ability to synthesize a normal β-chain decreased more rapidly than that for α-chain during red cell maturation in the patients with Hb Lepore trait. Decreased synthesis of Lepore globin chains may be related to the fact that the initial chain sequence is derived from the β-chain gene.—M.G.B.


Two relatives with Hb-Malmö had erythrocytes.
ABSTRACTS


Ten subjects absorbed 31% of 0.56 mg 59Fe-dose after a week with normal food without meat, liver, and blood sausages, and 20% after a week with these iron-rich foods.—P.G.R.


To a homogenized, average Swedish diet, 59Fe-labeled hemoglobin and 55FeCl3 were added, and the paste was served to four controls and four blood donors. Absorption, measured in blood and whole body, was on average 3% (0.04 mg) of heme iron in controls and more than twice that in the blood donors. It is suggested that the added iron represents food iron, because in a separate experiment with labeled maize, wheat, eggs, and others, the extrinsic/intrinsic labeling ratios varied only between 0.93 and 1.08, with a variation coefficient of around 10%.—P.G.R.


The results of ferrokinetic investigations in 20 anemic patients with rheumatoid arthritis (RA) and in 15 healthy subjects are presented. It was found that disturbances of iron metabolism in anemia of rheumatoid patients share many features in common with those of the anemia of true iron deficiency. The 59Fe t1/2 in plasma was shortened; incorporation of 59Fe into erythrocytes was rapid and efficient; the plasma iron pool was markedly diminished and its turnover rate increased; red cell renewal was abnormally high; and the erythrocyte lifespan was shortened. However, the very early accumulation of transferrin-bound radioiron in spleen and liver, as demonstrated by external measurements, was the sign that differentiated anemia of RA from true iron deficiency anemia and may support the assumption of an abnormally high avidity of the RES for iron in chronic inflammatory states.—M.K.

Investigations on Glycolysis and on the Character of Hemoglobin in Red Cells of Hereditary Spherocytic Anemia. L. Hiriolowa, J. Kwiatkowska, J. Rybusinska-Lebuda, W. Dobrzeszycka, and W. Wlodarczyk. Department of Internal Medicine of Specialized Hospital, Department of Physiological Chemistry of Medical Faculty and Department of Physiological Chemistry of Pharmaceutical Faculty, School of Medicine, Wroclaw, Poland. Acta Haematol Pol 2:121–1281, 1971.

Hereditary spherocytic anemia was studied in six patients, all of whom were close relatives. In the erythrocytes of five nonsplenectomized patients, increased lactate production and increased phosphofructokinase and aldolase activities were observed. Erythrocytes were separated by gel filtration on Sephadex G-100, and it was found that fractions containing spherocytes showed three to five times higher values than those free of spherocytes. In all patients, the hemoglobin A2 content in erythrocytes was markedly increased. In four cases, an additional hemoglobin with different electrophoretic mobility was observed, and in one case, a changed absorption spectrum for oxyhemoglobin in the Soret band was present.—M.K.

The Effect of Dextran 3000 on the Rate of Sedimentation and Aggregation of Erythrocytes. W. Tkaczewski and Z. Zielonka. Department of Infectious Diseases, Military School of Medicine, Lodz, Poland. Pol Tyg Lek 27:560–562, 1972.

Erythrocyte sedimentation rate (ESR) was determined after addition of dextran 3000 and normal saline in amounts corresponding to 10% and 30% of the volume of...
blood samples. The degree of aggregation of erythrocytes in these samples was also evaluated. Blood for investigations was collected from 15 subjects with high ESR. It was found that: (1) addition of dextran 3000 to a blood sample decreased the rate of sedimentation and reduced the degree of aggregation of erythrocytes, and (2) the effect on the ESR and on aggregation increased in proportion with the increase of dextran concentration in the blood samples.


Physiologic chronic erythrocytic polycythemia is found generally in homeotherms residing at high altitude as a result of the hypoxic environment. The effect of this phenomenon, however, on the blood volume and its constituents has not been clearly established. It is generally agreed that an increase in the body erythrocyte volume produces an increased blood volume; however, the effect on the body plasma volume is not clear. To differentiate the specific effect of erythrocytic polycythemia from the general effects of high altitude on the plasma volume, two kinds of physiologic chronic erythrocytic polycythemias were compared. These were produced in female domestic chickens: (1) hormonally (by androgen injection) at sea level, or (2) by protracted high-altitude exposure (12,000 ft). Plasma volumes were determined directly by injected $^{131}$I human serum albumin dilution methods. Total body erythrocyte and blood volumes were calculated from plasma volumes and adjusted peripheral hemocrits. Androgen treatment or high altitude exposure similarly increased the hemocrits approximately 45% and the body hemocrit volumes approximately 65%. These two procedures, however, affected the plasma volumes differently. No change in plasma volume was found at high altitude; however, there was a significant (17%) reduction in plasma volume in the androgen-treated birds as compared with either the sea level controls or the high altitude birds. It appears, therefore, that the vascular system of the body accounts for an increase in erythrocytic mass by either a reduction in plasma volume or no change in plasma volume, in which case differential changes occur in total blood volumes.—M.C.B.


Three cyanocobalamin binders have been isolated from normal serum by DEAE-cellulose column chromatography: transcobalamin II (TC II), main protein peak vitamin B12 binder (MPPB) and transcobalamin I (TC I). There is some evidence that TC II has the tendency to aggregate. In this study, preparative polyacrylamide gel electrophoresis was used for further purification of TC II prepared by DEAE-cellulose column chromatography. This preparation resulted in aggregation of this vitamin B12 binder. Exposure of transcobalamin II to 8 M urea resulted in liberation of the vitamin and aggregation of the binder. After removal of urea by dialysis, the binder retained the capacity to bind $^{57}$Co B12 and exhibited a molecular weight slightly above 120,000, clearly higher than the accepted molecular weight (56,000 of TC II. It appeared that TC II had aggregated during the polyacrylamide gel electrophoresis run.—M.G.B.

LEUKOCYTES


Molecular hybridization with radioactively labeled DNA complementary to the RNA of the Rauscher leukemia virus was used to probe for homologous RNA in
human lymphomas. Twenty-two of 32 specimens contained RNA possessing homology to the RNA of the mouse leukemia virus but not to that of the unrelated viruses causing mammary tumors in mice or myeloblastosis in chickens. Normal adult and fetal tissues failed to show significant levels of the leukemia-specific RNA. It appears that human lymphomas contain RNA sequences homologous to those found in a viral agent known to cause leukemia and lymphomas in an experimental animal. The fact that human leukemias and sarcomas also contain this type of RNA further emphasizes a remarkable similarity between the corresponding neoplasias of murine and human origin.—J.E.U.


The records of 29 patients with Hodgkin's disease involving the lung were reviewed to determine the pathologic features of Hodgkin's disease with lung involvement, the diagnostic and prognostic significance of the radiographic manifestations of lung involvement, and the clinical course of the disease after lung involvement was recognized. Twenty-six of the 29 patients had nodular sclerosing Hodgkin's disease, a frequency far in excess of the relative frequency of this particular cell type in the population from which the patients were selected. In all patients who had not previously received anterior mediastinal radiation or systemic chemotherapy, hilar adenopathy was present when lung involvement was recognized. The development of diffuse involvement of both lungs during the course of follow-up was a grave prognostic sign. Mean survival after recognition of lung involvement was 23 mo.—J.E.U.


Thirty patients have had laparotomy for staging of Hodgkin's disease from 1967 to 1972 at Lexington, Ky. hospitals. Sixteen of 30 patients (53%) had alterations in staging as a result of laparotomy. Fourteen patients were reclassified to a more advanced stage and two patients to a less advanced stage. Clinical assessment of abdominal involvement was in error in 54% of 26 evaluable patients. The most frequent histologic type in these patients was mixedcellularity, noted in 46% of all slides examined. Thirteen of 30 patients (43%) had combined histologic types noted at different anatomic sites at the same time. The value of laparotomy and splenectomy in staging of Hodgkin's disease is confirmed. Significant morbidity and mortality occurred (27% and 6.6%, respectively). We recommend that selective criteria be defined for laparotomy and splenectomy in staging patients with Hodgkin's disease.—J.E.U.


Liver involvement, proven by laparoscopy and biopsy, was found in 24 of 161 patients with Hodgkin's disease in various clinical stages and different histologic forms. In the remaining 116 patients (84.6%) a mesenchymal reaction of differing severity was demonstrated, ranging from diffuse activation of the reticuloendothelial system (grade I) to histiocytic granulomas (II) with demonstration of eosinophilic granulocytes and atypical reticulum cells (III). There was no positive correlation between the incidence or severity of the mesenchymal reaction and certain histologic forms of Hodgkin's disease. With increasing involvement of lymph nodes, the incidence and severity of mesenchymal reactions increased. In all 24 patients with liver involvement, the mesenchymal reaction was of the grade III. It is not yet clear whether grade III mesenchymal reaction is a forerunner of Hodgkin's infiltration or a nonspecific reaction.—J.E.U.

Anemic Stress as a Trigger of Myelogenous Leukemia in the Unirradiated RF Mouse. J. Gong, P. Braunschweiger, and C. Glomski. Schools of Dentistry and Medi-

Ninety-six per cent of mice that were bled of 50% of their blood volume when they were 9 wk old succumbed to myelogenous leukemia by 15 mo after phlebotomy, the majority of them dying between 7 and 10 mo after this treatment. These results suggest that (1) anemia is an effective stress for triggering myelogenous leukemia in animals that are predisposed to the disease, (2) the RF mouse is "naturally" prone to the development of myelogenous leukemia, and (3) the concept of two-step de novo induction of myelogenous leukemia appears to be applicable in this animal.

J.E.U.


Clinical and pathologic evidence suggests that a wide spectrum of lymphoproliferation exists in Sjögren's syndrome (SS), from benign disease with lymphoid infiltrates confined to glandular tissue on the one end, to widespread lymphoreticular malignancy on the other. In the middle of the spectrum are patients threatened by extraglandular extension of lymphoproliferation that is not clinically or histologically malignant and that apparently has the potential to regress with appropriate therapy or to progress to frank neoplasia. Illustrative patients are described. Over 30 other case reports associating SS with pseudolymphoma, Waldenström's macroglobulinemia, reticulum cell sarcoma, or other lymphomas appear in the literature. Similar lymphoproliferative processes have been observed in other autoimmune diseases, in certain immune deficiency states, with hydantoin and other anticonvulsant drugs, and in experimental animal models. In SS, as in these other conditions, it seems likely that a combination of genetic, immunologic, and viral or other unknown environmental factors plays a role in pathogenesis. An excellent review.—J.E.U.


Lactoferrin has a molecular weight of 76,000 and binds iron. Normal sera have up to 3.5 mg lactoferrin/liter, and normal granulocytes 3 g/10^8 cells. Many patients with chronic myelocytic leukemia and polycythemia have increased serum values.

—P.G.R.

Lysozyme in serum was over 5 mg/ml in all patients with monocytic leukemia and polycythemia vera. Urinary lysozyme was over 10 mg/liter in all patients with monocytic leukemias and in two of six patients with chronic myelocytic leukemias.

—P.G.R.


Thymidine-labeling indices in promyelocytes and myelocytes were 10% and 13% in two untreated patients with myeloblastic leukemia, as compared to 9% and 42% in two others in remission.—P.G.R.


Thirty patients with myelosclerosis (Ms) and myeloosteosclerosis (Mos) were examined. In 26 cases, the clinical diagnosis was confirmed by histologic examination of bone marrow trephine biopsy specimens. In 20
cases (66%), sclerotic areas were detected by x-ray examination. Radiobiologic bone changes were most frequently localized in the vertebral column, ribs, metaphyses, and epiphyses of humerus and femur. These x-ray changes were encountered more frequently in patients with the histologic diagnosis of Ms and Mos than in patients with myelofibrosis.—M.K.

The Leukocytic Reaction In Experimental Candidiasis in Mice. E. Kowal, A. Brylinska, and A. Perzanowski. Faculty of Microbiology and First Department of Internal Medicine, School of Medicine, Bialystok, Poland. Patol Pol 21:159–167, 1972.

Changes in white blood cell counts were examined in mice after intravenous injection of living Candida albicans, as well as preparations of cell walls and somatic antigen isolated from disintegrated Candida albicans. A common property of all injected antigens was the induction of marked granulocytosis and lymphopenia. A fairly pronounced reaction of reticulum cells was observed particularly after injections of somatic antigen preparations. The highest mortality was induced by injections of living Candida albicans, followed by the group of animals receiving the somatic antigen. —M.K.


Proerythroblasts had a mean in vitro thymidine-labeling index of 76%, and marrow lymphocytes (0.51%–1.14% of marrow cells) had an index of 0.6% in 11 untreated pernicious anemia bone marrows, suggesting either that marrow lymphocytes have a much shorter S-phase than proerythroblasts or that they are not their immediate precursors.—P.G.R.


The effect of 50 mg of prednisone on peripheral blood granulocyte counts was examined in 24 patients with bone marrow hypoplasia and in ten healthy subjects. In the latter group, the rise of the granulocyte count attained 2000 or higher values in all cases. Similar results were obtained only in ten of the patients with marked depression of granulopoiesis. Determinations of alkaline phosphatase activity of granulocytes performed simultaneously gave less clear-cut results and could not be considered as a valuable method for examination of the granulocyte bone marrow reserve.—M.K.


Six hours after 3H-thymidine labeling, guinea pig spleens began to release labeled lymphocytes.—P.G.R.

HEMOSTASIS


The authors used two denaturing media in order to normalize the chromophores before determining tyrosine and tryptophane in bovine fibrinogen. These were KCl–KOH (0.2 µg) and 6 M guanidine (0.2 µg). The latter more fully denatures the fibrinogen molecule than KCl–KOH. Abstracter’s comment: This technique can be useful in studies for the understanding of the action of bovine fibrinogen on abnormal human platelets.—J.C.
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A patient with pancreas cancer and thromboses in spite of dicoumarol had intravascular coagulation and 11% soluble fibrin in plasma, possibly because of protease (trypsin?) action.—P.G.R.

Breathing with Air Enriched in O2 and Fibrinolysis in Euglobulins. O. Bukowska. Department of Anesthesiology and Reanimation and Department of Clinical Biochemistry, School of Medicine, Gdask, Poland. Pol. Tyg. Lek. 27:511-513, 1972.

The effect of breathing with air enriched in O2 on fibrinolysis in euglobulins was examined in 15 surgical patients. Significant shortening of the lysis time from 170 ± 11 min to 130 ± 10 min was demonstrated after 20 min of breathing with O2 enriched air. Since no changes in blood PCO2 were observed, the activation of fibrinolysis could be ascribed to hyperoxygenation and not to respiratory alkalosis.—M.K.


Analysis of kinetics of plasma clotting in patients treated with Sintrom confirmed the report by Hemker et al. on the existence of a clotting inhibitory protein induced by the absence of vitamin K. An effect of this inhibitor on levels of factors II, VII, and X was observed. It suggested the existence of inactive precursors of these factors in the plasma of patients treated with oral anticoagulants. The inhibitor was adsorbed on barium sulfate and was eluted from it with sodium citrate.—M.K.

Inhibition of Human Platelet Aggregation by Dimethylsulfoxide, Dimethylacetamide, and Sodium Glycerophosphate. G. C. Holtz, and R. B. Davis. Division of Hematology and Department of Internal Medicine, University of Nebraska College of Medicine, Omaha, Neb. Proc. Soc. Exp. Biol. Med. 141:244, 1972.

Dimethylsulfoxide (DMSO), dimethylacetamide (DMAC), and sodium glycerophosphate are known to reduce the platelet damage associated with freezing. DMSO and DMAC, with dextrose, are known to improve the ability of frozen platelets to support clot retraction and to improve morphologic preservation of platelets observed by phase contrast microscopy. Also, recovery of frozen rat platelets after transfusion into thrombocytopenic rats is known to be enhanced by exposure of platelets to DMSO or DMAC and dextrose at the time of freezing. Sodium glycerophosphate alone is known to provide partial protection of human platelets against loss of platelet aminopeptidase activity by freezing. In the present experiments, aggregation of platelets after exposure to the above cryoprotective agents (without freezing) was studied. DMSO, DMAC, and sodium glycerophosphate reduced aggregation of human platelets by ADP, epinephrine, and thrombin. Initial aggregation by ADP and the secondary wave of aggregation associated with the release of platelet ADP were inhibited. DMSO and DMAC with 5% dextrose blocked aggregation as effectively as DMSO or DMAC alone. Platelets exposed to DMSO, DMAC, or sodium glycerophosphate and resuspended in platelet-poor plasma, free of cryoprotective agents, did not aggregate as did platelets exposed to Tris-buffered saline, suggesting that platelets had been irreversibly altered in vitro. It is concluded that the above cryoprotective agents are somewhat deleterious for platelet function.—M.G.B.


The results of routine tests of blood coagulation and fibrinolysis carried out in 50 patients with acute leukemia, 50 patients with chronic granulocytic leukemia, and 50 patients with chronic lymphatic leukemia are presented. In 22.6% of all patients, an
increase in plasma fibrinogen was found. An increase in platelet count was observed in 22% of patients with chronic granulocytic leukemia. A prolonged bleeding time and a prolonged clotting time, as well as diminished numbers of platelets and increased plasma fibrinolytic activity were observed more frequently in patients with acute leukemia than in patients with other types of leukemia.—M.K.


Platelet function was determined in 13 patients with acute granulocytic, two with acute monocytic, four with acute lymphocytic, two with chronic granulocytic leukemia in blast crisis and one with "pre-leukemia." In 11 of 15 patients in relapse and three of five patients in partial remission, there was impaired platelet aggregation and/or defective platelet factor III release, and these were associated with reduced platelet survival. In complete remission, both platelet function and survival were normal. It is possible that, in acute leukemia, a shortened survival of platelets due to abnormalities in their structure or function may contribute to the production of thrombocytopenia.—F.W.G.


Collagen from two children with hyperelasticity and friability of the skin, hyperextensibility of the joints and eye changes had decreased platelet aggregating effect possibly explaining bleeding in Ehlers–Danlos syndrome.—P.G.R.


$^{51}$Cr-labeled platelets and $^{99m}$Tc scintigraphy of spleen were studied in 28 subjects. With increasing splenomegaly a larger portion of the body’s platelets were found in the spleen.—P.G.R.


Platelet survival studies with a $^{51}$Cr-labeling technique were carried out in patients with untreated Hodgkin’s disease before and after splenectomy. The results have shown that: (1) platelet recovery increased from 30% to 90% after splenectomy, indicating a considerable pooling of platelets in the spleen; (2) the splenic platelet pool increased with increasing spleen weight; (3) platelet survival was not affected by splenectomy except for a transitory shortening in the postoperative phase; (4) the thrombocytosis after splenectomy increased with the weight of the removed spleen and may, to a large part, be due to removal of a splenic platelet pool.—J.E.II.

IMMUNOHEMATOLOGY


Using various reagents the authors were able to show that allotypes of immunoglobulins supported by $\gamma G_1$ and $\gamma G_3$ in humans and nonhominal primates are heterogenous. They are the consequences of paleosequences identical for all groups and of neosequences found only in highly differentiated strains.—J.C.

Eighty-two patients with Hodgkin's disease and 40 patients with multiple myeloma were HL-A typed. The results were compared with those of 255 healthy individuals of the same geographical area. In both diseases W18 (4c'), belonging to the 4c-complex, was significantly increased. - T.E.U.


The phenomenon of lymphocyte transformation, triggered by surface interaction with antigens and certain "mitogens" is readily observed in tissue culture and correlates well with cellular immunity in vivo; however, the control mechanisms remain unclear. Studies were done to consider the role of cyclic adenosine monophosphate (C-AMP) in lymphocyte transformation. The studies were performed in vitro utilizing the addition of C-AMP, 5'-GMP and related compounds to normal human lymphocytes in tissue culture. It was shown that C-AMP, in high concentrations, suppressed tritiated thymidine uptake of normal lymphocytes in tissue culture. GMP increased but AMP and CMP did not significantly affect the tritiated thymidine uptake of normal human lymphocytes in tissue culture. Furthermore, C-AMP in low concentrations increased the tritiated thymidine uptake of PPD-stimulated human lymphocytes in tissue culture. - M.G.B.


Among 54 patients with chronic lymphocytic leukemia (CLL) there was not one with entirely normal serum immunoglobulin patterns. Most had severe reductions in IgA and IgM. IgG was less drastically reduced. Six patients had paraproteins. With increasing lymphocyte counts there was a progressive tendency to lower IgG and IgA levels, but IgM was low even with low lymphocyte counts. Similarly IgG levels dropped with increasing duration of the disease, with or without treatment with chlorambucil and/or prednisone. There was a strong correlation between low IgG levels and recurrent infections. - F.W.G.

**Impairment of Renal Function in Multiple Myeloma.** Z. Hanicki, W. Hirszel, M. Magdon, W. Pajdak, W. Szczepkowska, and T. Zebro. Department of Nephrology, Department of Clinical Chemistry and Department of Anatomopathology, School of Medicine, Krakow, Poland. Przegl. Lek. 29:541–548, 1972.

Multiple myeloma was diagnosed in ten of 622 patients with renal disease admitted to the Nephrological Unit. Proteinuria and/or renal insufficiency were the leading or the only manifestations of the disease at the time of admission. Additional tests
were carried out in order to make the diagnosis more secure. These included a radiological survey, histological studies of kidney biopsy specimens, hematological tests, and analysis of serum proteins. From the results of these tests it was possible to demonstrate that the impairment of renal function was a secondary phenomenon. Myeloma nephropathy was described and the importance of immunological studies of plasma immunoglobulins and urinary proteins for a correct diagnosis of myeloma was underlined. The necessity of taking into consideration multiple myeloma in the differential diagnosis of proteinuria was stressed since manifestations of this disease may frequently be atypical, particularly in the initial stages.—M.K.


The effect of several high-molecular substances upon the hemolytic activity of crystalline SiO$_2$ was investigated. This activity was lowered by coating of dust with No-polyvinylpyridine, pentamerithrit monoooleate, Tween-40 and oleinol-7. It could not be concluded that each substance inhibited the development of silicosis by lowering the hemolytic activity of SiO$_2$. However, these results could be of use in the study of prevention of pneumoconiosis.


Bul sulfan (4 mg) induced improvement in five out of nine patients with psoriasis.

BOOK REVIEWS

Synthesis, Structure and Function of Hemoglobin. Edited by Helmut Martin and Lothar Nowicki, Munich, Lehmanns Verlag, 402 pages, no price indicated on the review copy.

This book is the product of the international symposium “Synthese, Struktur und Funktion des Hämaglobins” held at Bad Nauheim, April 22-24, 1971. As a result it is a mixed bag, stylistically and linguistically. About two-thirds is in English; the remainder in German. Clinical hematologists, clinicians in training, and investigators in other areas of hematology should find little of value here. Those investigators working in one of the areas defined by the title will be much more interested. Multiple authorship leads, as usual, to a lack of continuity, with several omissions and duplications in the presentations.

The book begins with several brief but good discussions of hemopoiesis, then continues with papers on hemoglobin synthesis of very mixed quality. In the latter group Anderson’s review and two papers from Cambridge, England stand out. There is little continuity in the next group of reports which concern structure, genetics and evolution, synthesis and function; but several are noteworthy including Ludloff’s brief review of sequencing, Fitch’s discussion of evolutionary variations, Rachmilewitz’s and Harari’s studies on hemichromes, Bartel’s