ABSTRACTS
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ERYTHROCYTES

REGULATION OF IRON ABSORPTION. M. S. Wheby.

This clear and concise review emphasizes the importance of mucosal cell iron content in regulating absorption. Less iron is absorbed when mucosal storage is ample; more iron is absorbed and relatively little is stored in newly formed mucosal cells with increased erythropoiesis. Iron stored in the mucosal cell, probably as ferritin, is not transferred to plasma but is lost when the cell is sloughed at the villous tip.—R. O. W.


Although iron deficiency is rare in Bantu subjects, hypochromic anemia is not uncommon in hospitalized patients. The possibility that infection might be a causative factor was investigated by studying the hematologic status in 34 patients selected on the grounds only that they suffered from chronic infection. The mean results in the infected subjects were as follows: Hb 9.6 Gm./100 ml. MCHC 29.4 per cent. plasma iron 44 μg./100 ml. and TIBC 162 μg./100 ml. The anemia was regarded as hypochromic in 70 per cent. Hemosiderin deposits in the marrow were graded as 1–2+ in 6 patients and 3–6+ in the remainder.—T. H. B.


The effect of allopurinol on iron absorption was studied in 28 normal subjects. With three dosage schedules, there was no effect. The study was thought to provide indirect evidence that the ferritin-xanthine oxidase system is not of major importance in the mucosal control of iron absorption.—P. B.


The effect of desferrioxamine on iron deposits was studied in a case of primary hemochromatosis, one of secondary hemochromatosis, 2 of Cooley’s anemia and one of Silvestroni-Bianco anemia. A decrease in sideroblasts and hemosiderin in the bone marrow was noted in all cases. Sometimes,
ABSTRACTS

however, the values returned to those obtained before therapy. The PAS reaction was positive in the erythroblasts of the Cooley’s anemia patients and was not modified by treatment. In secondary hemochromatosis, 23 per cent of the erythroblasts had PAS positive granules. This percentage fell after the use of desferrioxamine. The orthochromatic erythroblasts decreased in the case of secondary hemochromatosis, in one case of Cooley’s anemia and in the Silvestrini-Bianco anemia. During treatment, a decrease in the number of erythroblasts in the blood of the cases of hemosiderosis already submitted to splenectomy was noted.—M. J.


Desferrioxamine was used in three patients with primary hemochromatosis, one with secondary hemochromatosis and 4 with hemosiderosis. Excretion of iron in the urine reached, in some cases, levels of about 80 mg./day. The drug, in doses up to 1000 mg. daily IM, did not cause any toxic effects. The patients, however, were not able to tolerate the treatment for a long time because of the local effects of the injections. In spite of the large amounts of iron excreted, no significant improvement was seen. Only a minor degree of subjective amelioration was noticed, associated with a reduction in the number of bone marrow siderocytes and sideroblasts. Plasma iron was not reduced and erythrocyte, leukocyte and differential cell counts were not influenced. The patients with primary hemochromatosis were not anemic and were submitted to phlebotomy with more gratifying results. Iron excretion was greater and a marked reduction in deposited iron with improvement in clinical symptoms was seen with diminution of the plasma iron level.—M. J.


The concentration of intrinsic factor in the gastric juice of patients who had had partial gastrectomy and who were deficient in vitamin B₁₂ was similar to that found in patients with Addisonian pernicious anemia. Estimation of the serum vitamin B₁₂ concentration, especially where this was greater than 80 μg/ml., was inadequate evidence upon which to base a diagnosis of vitamin B₁₂ deficiency because iron and/or folie acid deficiency which was not uncommon in the gastrectomized patients can lower the serum B₁₂ levels. Corroborative evidence, such as impaired absorption of vitamin B₁₂, should be required and the data presented in this paper suggested that a low concentration of intrinsic factor in the gastric juice may also provide useful information.—P. B.


The excretion of Figlu and urocanic acid has been measured in 40 post-gastrectomy patients without folate or vitamin B₁₂ deficiency or hypoproteinemina and in 50 post-gastrectomy patients with folate or vitamin B₁₂ deficiency or hypoproteinemina. The excretion of urocanic acid without Figlu was raised in 8 patients. 7 with hypoproteinemina. In 4 whose hypoproteinemia was corrected, urocanic acid excretion fell to normal. In contrast, folie acid or vitamin B₁₂ therapy did not affect urocanic acid excretion by these patients, even though these deficiencies were present. Urocanic acid was excreted by folate-deficient patients only when hypoproteinemia or liver damage also was present. In such circumstances, neither the estimation of Figlu alone nor the estimation of the combined excretion of Figlu and urocanic acid was a reliable test for folate deficiency.—H. H. F.


The incidence of Addisonian pernicious anemia in this study (3 of 90 in hyperthyroidism and 4 of 47 in myxedema) was similar to that recorded in other studies. None of these 7 patients was anemic and in 2 the serum vitamin B₁₂ level was in the “equivocal” range. Four patients were found who had antibodies against intrinsic factor, but without pernicious anemia (3 of 90 with thyrotoxicosis and 1 of 47 with spontaneous myxedema). They also had antibodies to gastric parietal cells and histologic evidence of gastritis which varied from
superficial gastritis with no apparent loss of secreting tissue to atrophic gastritis with only scanty residual parietal cells. All of these patients had adequate vitamin B12 absorption and all had intrinsic factor in the gastric juice. The findings in these 4 patients cast some doubt on the importance of intrinsic factor antibody in the pathogenesis of pernicious anemia. The authors speculated that, if the antibody does influence intrinsic factor-mediated vitamin B12 absorption, it may do so only via antibody produced locally in the gastric mucosa and in the ileal wall. Free antibody in the serum, even in higher titer, may be unimportant.—F. B.

**ABSTRACTS**


Serum mucoprotein values were studied by a polarographic method in 15 cases of pernicious anemia. In 12, values were markedly reduced (0.6 units and less). In one, the value approached the lower limit of normal (0.75 U.) and in two the values corresponded to the lower limit of normal (0.8 U.).—L. D.


Patients (123) with pernicious anemia were followed regularly for 8 years. Cancer of the stomach was found in 9 cases, i.e., 21 times more than in the population of Czechoslovakia. In 87 per cent, gastric resection was performed and 5 year survival was recorded in 81 per cent of all resected cases. Long term, systematic gastroenterologic and roentgenologic follow-up was fully justified as a means for early diagnosis of gastric cancer.—L. D.


Sickled erythrocytes fixed in glutaraldehyde and examined by electron microscopy possess an ultrastructure consistent with the presence of crystals of reduced sickle hemoglobin within the cells. The crystals are extremely anisometric, taking the shape of needles or sheaves of needles. The results clearly support the conclusion that sickling is due to intracellular formation of long slender crystals of reduced sickle hemoglobin.—H. S. J.


An investigation was undertaken to determine the frequency of the carrier state for abnormal hemoglobin forms, excluding thalassemia, among 3325 pregnant women in Cape Town. The calculated incidences of the various heterozygous states in per cent were as follows: Cape Malay colored—A + C 0.3, A + E 0.9, A + S 0.5; Non-Malay colored—A + C 0.3, A + E 1.0, A + S 0.3; White—A + C 0.4, A + E 0.2, A + S 0.2. No abnormal hemoglobins were found in Bantu subjects.—T. H. B.


Experiments were described which led to the choice of sucrose as a standard marker for the measurement of trapped plasma sodium. Human red cell sodium concentration could be measured with accuracy (1) if trapped plasma sodium was estimated with radioactive sodium and a correction was made for entry of sodium into the cells, providing cells and plasma were separated rapidly, (2) by the use of sucrose to derive the amount of trapped plasma sodium, and (3) by washing the cells with sodium-free solutions.—O. P. J.


When blood is centrifuged, some plasma remains trapped in the layer of packed cells. Since the concentration of sodium in plasma is approximately 20 times greater than that in red cells, an error of 1 per cent in the estimate of trapped plasma will lead to an error of 20 per cent in the calculated intracellular sodium concentration. When radioactive sodium is injected intravenously, the initial ratio of plasma to red cell specific activity is much greater than 20 and accurate esti
mation of trapped plasma sodium is extremely important. Those aspects of the problem which have arisen from or which have been clarified by in vivo experiments are considered.—O. P. J.


The influence of ionic strength, ionic composition, temperature and pH of the medium on salt efflux from red cells was reappraised with a modified technic which allowed precise and continuous measurement under steady-state conditions even at very low (3 × 10^{-5} M NaCl) ionic strength.—O. P. J.


Glycolysis of red cells from healthy children and from patients with congenital microspherocytosis was studied. Production of lactic acid and aldolase and phosphofructokinase activities were estimated under aerobic and anaerobic conditions. In all cases of congenital microspherocytosis, increased production of lactic acid and increased aldolase activity were found. Phosphofructokinase activity was either increased or normal. Aldolase activity was increased under aerobic. as compared with anaerobic, conditions.—E. K.


Periodate consumption by horse erythrocyte mucoid (HEM), untreated, and devoid of sialic acid (HEM1) was determined during oxidation. The sugar composition of HEM and HEM1 before and after periodate oxidation was compared. Periodate oxidized N-glycolylneuraminic acid and about 30 per cent of hexoses in HEM and 50 per cent or more of hexoses in HEM1. Hexosamines were not oxidized. The oxidation of galactose destroyed the activity of mucoid against anti-N phytoagglutinins from Vicia graminea. The structure of the heterosaccharide side chains of HEM was discussed.—E. K.


The activity was studied by determining oxygen uptake in the Warburg apparatus and by the incorporation of ^131I into tyrosine. In the peroxidase reaction, the Hp-Hb complex utilized H2O2 generated from glucose by glucose oxidase. The complex exhibited peroxidase activity in the iodination of tyrosine and it behaved as a single enzyme with two different activities, corresponding to two steps in the synthesis of iodotyrosines, i.e., peroxidation of iodide to "active iodine" and incorporation into tyrosine.—E. K.


This paper should be of interest to comparative hematologists, parasitologists and virologists. The genus Pirhemocyton, was created to accomodate an intraerythrocytic parasite of the lizard, Tarentola mauritanica. This electron microscopic investigation has shown that Pirhemocyton is not a protozoon and has disclosed cytoplasmic particles which in all probability are viruses.—O. P. J.


Problems of diagnosis and classification pertaining to primary refractory anemias were discussed, based on an analysis of 50 cases. The main subgroups comprised hypoplastic pancytopenia, panmyelopathy, primary refractory anemia in the strict sense and sideroblastic refractory anemia. Transition types between these occurred occasionally, but change into early leukemia was rare. Splenectomy performed in 20 cases resulted in excellent improvement in 7 cases followed up for a long time.—L. D.

LEUKOCYTES

Serum and Urinary Lysosome (Mucamidase) in Mycosytic and Monosomyocytic Leukemia. E. F. Osserman and D. P. Lawlor. From Columbia University College of Physicians and Sur-
Markedly increased quantities of lysozyme were found in the serum and urine of ten consecutive cases of monocytic and monomyelocytic leukemia. Normal levels were found in myelocytic leukemia without associated monocytosis and reduced levels were noted in cases of lymphatic leukemia. Moderately elevated levels were found in patients with chronic infections. Lysozyme isolated from the urine of several monocytic leukemia patients was carefully studied and was found apparently to be identical to the lysozyme of normal tears, saliva, leukocytes and serum. An agar plate method developed for quantitating lysozyme activity in small samples of serum, urine or other biological fluids was found to be superior to previously available assay procedures. The present evidence indicated that lysozyme was the principal product of the proliferating monocytes in monocytic and monomyelocytic leukemia.—T. E. B.


Blood filtered through glass wool is virtually free of monocytes. In vitro leukocyte cultures showed that macrophages do not arise from leukocyte cultures of monocyte-free blood or from lymph. This finding supports the view that macrophages arise from monocytes in the peripheral blood and not from lymphocytes.—H. H. F.


Human monocytes, isolated from peripheral blood, were incubated with neuraminidase which cleaves the terminal charged sialic acid units. After incubation, the monocytes showed a decrease in their usual negative surface charge density and a suggestive increase in deformability. Increased cell deformability may be a consequence of the reduction in surface charge density. After incubation, more monocytes made contact with negatively charged particles and, among those cells contacting particles, more phagocytosis occurred than in control cells. Decreasing the negative surface charge on monocytes, therefore, seemed to have increased their capacity for phagocytosis.—T. E. B.


TPNH-DPN transhydrogenase activity was assayed in the particulate fractions of the leukocytes from normal and leukemic subjects. The highest levels of enzyme activity were found in the cells from patients with acute lymphocytic leukemia, while the lowest levels were found in the cells from normal subjects. Cells from patients with chronic lymphocytic leukemia, acute myelocytic leukemia and chronic myelocytic leukemia showed intermediate levels of activity.—T. E. B.


The authors’ hypothesis was that human leukemia might be the result of viral infection from animals. Bovine blood was studied from 37 farms where cases of leukemia were observed. Controls were cattle from farms owned by 37 healthy subjects and by 37 patients with peptic ulcers. A statistically significant increase in lymphocytosis was found in the blood of cattle from farms owned by patients with chronic granulocytic leukemia or lymphosarcoma. The lymphocytosis found in these cattle suggested a subclinical or clinical form of lymphatic leukemia. These cows probably were a potential source of infection for people.—E. K.


Twenty-one patients with acute leukemia (lymphoblastic and myeloblastic) were given transfusions of leukocytes from donors with chronic myeloid leukemia. With one exception, all patients
were resistant to the available forms of chemotherapy or had contraindications, mainly neutropenia. There were 9 remissions: 6 complete (normal blood and bone marrow) and 3 incomplete. The remissions were of short duration. The mechanism of the antileukemic effect of these leukocyte transfusions seemed to depend on immunologically competent cells in the transfusion.—P. B.


A horse was immunized with saline suspensions of canine lymphocytes prepared from mesenteric lymph nodes of exsanguinated dogs. The antiserum obtained agglutinated lymphocytes from numerous dogs at a titer of more than 1:1000. Daily subcutaneous injections of dogs with 10–20 ml. of antiserum resulted in severe lymphopenia, mean peripheral lymphocyte counts falling from about 4000/mm.² to 0–300/mm.² and remaining low for as long as daily injections were continued (110 days). Renal transplants in dogs treated with the horse antiserum survived for long periods, two surviving beyond 350 days with normal function and histologic appearance. Long-term administration of the antiserum was well tolerated. Heterologous antiserum to dog lymphocytes was administered for more than 350 days without apparent loss of lymphopenic and immunosuppressive effects. Perhaps immunologically competent dog cells, which might have made antibody against the horse serum, were destroyed by contact with the serum. Heterologous antiserum to lymphocytes can be as potent a lymphopenic and immunosuppressive agent in dogs as it is in smaller animals.—T. E. B.


The influence of these agents on the activity of some hydrolases and oxidases was investigated in KB cell cultures. The activities were measured by histochemical methods 6, 24, 48, 72 and 96 hours after addition of the compounds. After addition of Endoxan, a higher proportion of cells with high acid phosphatase activity was noted, but a decrease in the percentage of these cells after addition of hydrocortisone and Antistine was observed. Endoxan and hydrocortisone caused a decrease in the percentage of cells with cytochrome oxidase and succinic acid dehydrogenase activity.—E. K.


The activity of the enzyme was investigated in 101 patients. Slightly increased activity was found in 2 chronic myelocytic leukemias, 1 acute leukemia, 2 polycythemias, 2 myelofibroses, 3 pannylphthiophes, 1 myeloma, 1 malignant reticulosis, 1 lymphosarcoma, 3 hemolytic anemias and 2 pernicious anemias.—L. D.


Of 3500 peripheral blood smears examined, 122 showed vacuolated neutrophils. Bacterial infection was the principal diagnosis in 119 of these 122 patients. Sixty-five percent of the patients whose blood was cultured had sepsisemia. A review of the reports of blood smears of 21 consecutive patients with septicemia revealed that 19 had been recorded as showing vacuolated neutrophils. The digestion of engulfed bacteria by neutrophils was thought to depend upon lytic enzymes released from lysosomes within the white blood cell. The products of digestion apparently were enclosed thereafter within vacuoles in the neutrophils.—

T. E. B.


Many investigators have shown that the lysosomes of polymorphonuclear leukocytes contain a number of potential phlogistons with widely differing modes of action. These substances probably contribute to the development of tissue injury.
reactions in which PMN leukocytes are a causal factor. The partial purification and characterization of one of these phlogistons, a mast cell-rupturing polypeptide, is described. The component present in the lysosomes of rabbit exudate PMN leukocytes is a low molecular weight (1200 to 2400) polypeptide containing a relatively large proportion of arginine, is thermostable and dialyzable and does not cause contraction of the isolated guinea pig ileum. The mast cell-rupturing activity of the agent is destroyed by trypsin.—T. E. B.

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Ascidian blood is interesting because of the rich variety of cell types and their ability to concentrate vanadium from sea water $3 \times 10^6$ fold. The present study, undertaken to determine the developmental relationship of the blood cells and to study the fine structure of each well defined cell type, revealed that the lymphocyte was the source of all 8 types. After glutaraldehyde fixation alone, the only extremely dense components were particles in the compartment cells and signet ring cells, but there was some indication that vanadium compounds were leached out in the preparative procedure.—O. P. J.


Neutrophils of 74 patients with infectious hepatitis were examined by a cytochemical method. Enzyme activity was either within the normal range or lower. There was no marked difference between values in adults and children nor any relation to clinical severity. After administration of prednisone, enzyme activity rose markedly, with a peak after 3 days. After the fourth day, activity declined even when treatment with the same amount of prednisone was continued. No connection was found between alkaline phosphatase in leukocytes and serum alkaline phosphatase, bilirubinemia, thymol test and transaminase levels.—L. D.

**Inclusions in Leukocytes from Urine of Patients with Rheumatoid Arthritis. O. Vojtíšek, S. Havelka and P. Vilímek. From the Institute for Rheumatology, Prague, Czechoslovakia. Čas. Lék. Česk. 105:801–802, 1966.**

Granular inclusions in leukocytes from synovial fluid from knee joints were found. Similar inclusions were identified in the leukocytes from the urine of patients with rheumatoid arthritis.—L. D.

**HEMOSTASIS**


Anti-AHG antibodies obtained by immunization of rabbits with Factor VIII preparations freed of fibrinogen were tested for their inhibitory activity in the thromboplastin generation test and in thromboelastography. These antibodies could be neutralized by normal plasma or serum. Plasma from the majority of patients with moderate hemophilia A had the ability to fix anti-AHG antibodies equal to or slightly less than that of normal human plasma. There was no antigenic form of Factor VIII in severely affected patients with hemophilia A and Von Willebrand's disease. The results suggested a molecular defect of Factor VIII in patients with hemophilia A.—E. K.


In 15 cases of Wilson's disease, fibrinogen, euglobulin lysis time, Factors II, V, VII, IX, X,
antithrombin III and platelet count were determined. In 5 patients, the disease had a hepatic form, in 10, neurologic symptoms prevailed. In the hepatic form, there was a marked decrease in fibrinogen, prothrombin. Factors II, V, VII, X, antithrombin III and thrombocytes. After long treatment with D-penicillamine, there was a marked improvement in the coagulation factors.—L. D.


Heparin levels were investigated. Temperature influenced the heparin levels: hypothermia slowed the escape of heparin from blood. Normothermia enhanced it. When whole blood and blood diluted with dextran were used, the level gradually fell and reached the critical value of 20 µg/ml in 60 min. At the end of one hour, a repeat dose of heparin was recommended. Heparin levels exhibited individual variation due to different sensitivity of the subjects and varying effectiveness of the preparations.—L. D.


Infusion of 500 ml. 6 per cent dextran (molecular weight 70,000) in isotonic saline in six healthy men after withdrawal of 500 ml. blood greatly diminished platelet adhesiveness and reduced their ability to be aggregated by ADP. These changes were maximal 4 hours after infusion and were opposite in direction to those observed in five controls who were given isotonic saline solution alone. It was thought that these changes may be related to the antithrombotic action of dextran.—P. B.


Platelet adhesiveness was measured by the Helm method (citrated platelet-rich plasma with added ADP passed through a glass bead column) in control subjects with no obvious arterial disease and in patients who received femoropopliteal bypass grafts. The results, both in a retrospective and prospective study, showed increased adhesiveness related to long-term patency, but not to initial patency in the immediate postoperative period. Blood glucose concentration 2 hours after an oral dose of glucose and serum cholesterol esters appeared unrelated to high platelet adhesiveness. Oral administration of linseed and maize oil did not reduce platelet adhesiveness and long-term anticoagulant therapy did not increase the long-term patency of the grafts.—P. B.


The electrophoretic technic was used to investigate the response of platelets to ADP and noradrenaline in patients with ischemic heart disease. Arterial disease in the limbs and cerebrovascular disease. The majority of these patients showed increased platelet sensitivity to ADP, but normal sensitivity to noradrenaline. A similar differential response pattern was observed in patients with hypercholesterolemia, homocystinuria and in some patients with diabetes. The abnormal sensitivity to ADP could be conferred on platelets from controls by incubation with platelet-poor plasma from abnormal subjects, provided the plasma was less than an hour old and had not been in contact with glass. The transfer was prevented by potassium cyanide. It was thought that this factor was released from other blood cells during the preparation of platelet-rich plasma.—P. B.


This congenital hemorrhagic syndrome with thrombocytopenia was observed in two brothers. The older boy died at 13½ and the younger at 2½. Immunologic examination showed marked changes with a decrease in IgM, a marked increase in IgA and the presence of a paraprotein of IgG-L type. The disease was considered to be a model of a hereditary immunologic deficiency. Subtle
morphologic and functional alterations in the ultrastructure of the platelets were noted.—L. D.

**ABSTRACT**


An unusual coagulation disorder was observed with the serum of three patients with thrombocythemia and manifested in pathologic thromboplastin generation. The coagulation defect was partially or completely corrected by leaving dilute serum to stand, by normal and hemophilia B serum and by small amounts of thrombin. The activation of serum coagulation factors was impaired in the patients reported.—L. D.


Thrombocytosis was found in 50 per cent of rheumatic patients. Adhesiveness of platelets in vivo, examined by the method of Borhgevink, was significantly diminished in 36 per cent of cases.—E. K.


Synovial fluids and plasmas of patients with rheumatoid arthritis were compared. In synovial fluids, high activity of contact factor, Factors VIII, IX and XIII and immediate and progressive antithrombin were found. Factor V, plasminogen and antiplasmin showed low activity as compared with plasma. Fibrinogen usually was absent in synovial fluids. The possible significance of the high activity of contact factor for the inflammatory process in joints was discussed.—E. K.


During the plasmin proteolysis of fibrin and fibrinogen, an active product is formed which evokes slow contraction of rat ileum. The factor differs from fibrinopeptide B. Due to its action on smooth muscle, this fibrinogen-derivative may also influence the muscles of blood vessels. The authors have suggested that plasmin plays an important role not only in hemostasis, but also in the control of the total circulatory system in some physiologic or pathologic conditions.—E. K.


A definite enhancement of euglobulin fibrinolysis was found in the placental artery and vein. Antiplasmin activity was near the upper limit of normal in both vessels. In delivering women, it was lowered to 53 per cent. The increase in thrombin time was definitely higher in arterial than in venous blood.—L. D.