ABSTRACTS

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ERYTHROCYTES


Respiration of red cells was studied in blood samples from 21 normal children less than 12 yr-old and 12 normal adults with ages varying between 24 and 30 yr. Oxygen consumption of red cells was demonstrated to be lower in children than in adults (p < 0.0001). Blood samples from seven children with different diagnoses were also studied. Red cells from patients with acute leukemia, aplastic anemia, osteosarcoma, and Letterer-Siwe's disease showed decreased oxygen consumption. Sickle cells and red cells from patients with Fallot's tetralogy showed increased values. Normal red cells suspended in plasma from patients with acute leukemia, osteosarcoma, or Letterer-Siwe's disease showed decreased oxygen consumption. The above findings suggest a plasma-red cell relationship as far as oxygen consumption is concerned.—M.J.


The authors describe a case of erythroleukemia in which a normal erythroid population coexisted for 2 yr with an abnormal clone characterized by hypochromia, decreased osmotic fragility, loading of mitochondria with ferritin aggregates, presence of hemoglobin H, and abnormal amounts of erythrocyte antigens A1, I, and i. Using...
staining of hemoglobin H by cresyl blue and a modified peroxidase reaction, the aberrant clone could be recognized in the electron microscope at the polychromatophilic, orthochromatic, and reticulocytic stages. Microspectrophotometric studies showed increased proportion of 4 and 8N basophilic erythroblasts and proerythroblasts, indicating a block at the G2 stage of mitosis. This finding suggested intramedullary destruction, which was confirmed at the ultrastructural level by increased phagocytosis of the abnormal cells. Deficiency in secondary neutrophilic granules, as well as in megakaryocyte granules and demarcation membranes, was also found, showing that all hematopoietic lines were involved. *Abstractor’s comment:* These abnormalities are part of a growing list of defects that are detected not only in leukemias but also in refractory anemias, with or without sideroblastosis or myeloblastosis. See Dreyfus et al. Press Med. 78:359, 1970.—J.M.P.


Carbonic anhydrase B (CA B) content was measured, by radial immunodiffusion, in red cell hemolysates in 20 nondialyzed and 10 dialyzed patients with chronic renal failure and was found to be significantly increased. No correlation was found between the erythrocyte CA B levels and the severity or duration of the uremic state or the change in acid-base metabolism. This indicates that the rise in erythrocyte CA B levels plays no role as a compensatory mechanism in uremic acidosis. The factors that do influence the rise in the CA B content of the erythrocytes remain to be explored.—B.R.


Fetal erythrocytes were detected using the Kleihauer technique in 45.3% of 461 Rh-negative patients following delivery. In one-fifth of the patients in whom fetal cells were detected, the cells were again detected on the fourth day. Clearance of fetal cells seemed to be a function both of the dose of anti-Rh globulin and the volume of fetal blood transferred.—A.A.M.


It appears well established that the cholesterol and phospholipid content of young red cells, primarily reticulocytes, is increased. The maturation of macrotelocytocyte membranes was studied over a period of 11 days in two animal models in vivo. (1) Reticulocytosis was induced by acute blood loss in rats and terminated by irradiation, and (2) reticulocytosis was produced in polychromic rats by exchange transfusion. Reticulocytes fell from 46.1% in irradiated rats and 30.7% in exchanged rats to normal by day 5. The mean cell volume of reticulocytes decreased 29% during this time (from 108 to 77 cu μ; normal = 63 cu μ). The mean cell hemoglobin content decreased 26%. The 51Cr survival of reticulocyte-rich blood was decreased 1.5–1.9 days, but cell survival was normal when corrected for the loss of hemoglobin per cell during maturation. Young reticulocytes had a cholesterol content 2½-fold and a phospholipid content fourfold that of mature red cells. Forty four per cent of the cholesterol and 67% of the phospholipid were lost during the maturation process. Isolated plasma membranes of reticulocytes had a cholesterol:phospholipid molar ratio equal to that of mature cells. The plasma membranes accounted for 50% of the phospholipid and more than 80% of the cholesterol of young reticulocytes. Approximately one-third of this lipid was lost during maturation. The mitochondrial fraction that was lost largely during the first few days of reticulocyte maturation and completely by day 5 was found cholesterol-poor. Splenectomy decreased the rate and extent of lipid loss during the reticulocyte maturation, resulting in mature red cells rich in lipid. The

Lipids comprise approximately 40% of the red cell membrane by weight. While there is little quantitative variation in membrane lipids among normal members of mammalian species and only minor qualitative variation between mammalian species, striking changes in red cell membrane lipid composition occur in the presence of liver disease. In obstructive jaundice, and in hepatitis or cirrhosis with an obstructive component, excess cholesterol phospholipid are acquired by the mature red cell. This addition of membrane lipid leads to the formation of target cells; at times spur-shaped red cells are seen. Because bile acids can influence red cell membrane structure and in view of the alteration of bile acid metabolism in liver disease, the authors investigated the role of bile acids in the genesis of abnormal cells in vivo. Rhesus monkeys were fed cholic acid and lithocholic acid (LCA) alone and in combination. Whereas the animals receiving cholic acid alone remained normal, those receiving LCA developed spur-shaped red cells within 3 days after beginning the administration of the bile acid. There was also evidence for hepatic injury, as evidenced by increased serum bilirubin and glutamic oxaloacetic transaminase activity. Normal alpha lipoprotein diminished on day 8. Beginning on day 11, the cholesterol content of red cells increased, reaching 28.5% greater than normal at 4 wk. There was no change in the red cell content of phospholipid; also, there was a small increase in the per cent of lecithin found. A mild hemolysis was noted, as shown by reticulocytosis and lowered hematocrits. On cessation of LCA administration, the serum levels rapidly fell and red cell morphology swiftly returned to normal. The cholesterol content of red cells, however, became normal over a slower time course. Sera obtained at various times during the LCA feeding were also incubated with normal red cells. All sera were first heated to 56°C for 30 min to inactivate lecithin: cholesterol acyltransferase. No change in the morphology and lipid content of red cells occurred in sera from animals receiving a normal diet. However, sera of animals receiving LCA induced spicules in normal red cells. The induction of changes in red cell cholesterol by various sera in vitro appeared to correlate with the degree of cholesterol accumulation that had occurred in vivo. Heated sera obtained on day 8 induced spicules but no change in lipid content of normal red cells in vitro, whereas sera obtained on day 31 caused both spicules and cholesterol accumulation. Spicules appear to be related directly to lithocholic acid. Accumulation of cholesterol by red cells appears to follow liver injury and alterations in serum lipoproteins.

—M.S.


Protein transfusions were used to treat 116 patients with various kinds of protein deficiency, including infections, cirrhosis, and nephrosis. Treatment was followed by weight gain and disappearance of ascites or edema. Abnormalities of blood amino acids, enzymes, and sodium and potassium levels were corrected, and hepatic function tests were improved. Also noted was a favorable effect on blood coagulation and adrenal function. While in patients with postgastrectomy anemia, erythropoiesis was improved.—J.V.

Iron in Enriched Wheat Flour, Farina, Bread, Buns, and Rolls. Council on Foods and

The Food and Drug Administration has proposed an upward revision of current standards for iron enrichment of wheat flour (white bread only), farina, buns, and rolls; the American Medical Association council on food and nutrition has concurred. Iron content of these foods would be raised from approximately 15 mg/lb to 40 mg/lb. On the average in the U.S., grain products now contribute one-quarter of total calories consumed; they contribute a significantly higher proportion in low income households, where iron deficiency is more prevalent. Average daily intake is a third of a pound of grain food; since only 5% of iron in bread is absorbed, the increase from 15 to 40 mg/lb would raise the daily iron absorption by approximately 0.5 mg. This is entirely a preventive measure. Theoretically, this should prevent iron deficiency and iron deficiency anemia in a substantial portion of the population now at risk. The concern has been expressed that this amount may also precipitate clinical disease in patients with latent hemochromatosis. Since current debate has exhausted existing data and on-going evaluations are planned, a trial of this program seems warranted to learn (1) if enrichment really helps reduce iron deficiency in the population and (2) if the incidence of hemochromatosis will increase.—R.O.W.


The functional adaption of the organism to hypoxia consists of increased cardiac output, increased pulmonary ventilation, shunting of blood, changes of hemoglobin affinity of oxygen dependent on the concentration of 2,3-diphosphoglycerate in red blood cells, and increased production of red blood cells. Increase in pulmonary ventilation is mediated by the stimulation of chemoreceptors in the carotid sinus and aortic arch. These chemoreceptors are stimulated by intracellular metabolic changes induced by lack of oxygen. The administration of cyanide, which blocks electron transport to oxygen by inhibiting cytochrome a_{3}, is followed by the same functional changes of pulmonary ventilation that are encountered in other types of hypoxia. The aim of this work was to study the effect of various metabolic inhibitors that are known to affect the chemoreceptors in the glomus caroticum and, thus, have an effect on the erythropoietin production. The experiments were performed using normal and hypoxic animals, either rats or rabbits. The metabolic inhibitors tested included potassium cyanide, sodium fluoracetate, and monooiodoacetic acid. Simultaneous administration of cyanide and monooiodoacetate to hypoxic rabbits was also tested. Although intoxication by these drugs reached the extreme levels compatible with survival of the experimental animals, no effect on erythropoietin production could be demonstrated. In normal animals, there was no sign of an elevation of erythropoietin titer in the plasma, which suggested that histotoxic hypoxia contrary to other types of hypoxia does not cause erythropoietin production. Exposure of animals to low pO_{2} atmosphere resulted in increased erythropoietin production, and administration of metabolic inhibitors did not interfere with erythropoietin production during experiments lasting up to 6 or 7 hr. Thus, the receptors controlling erythropoietin production appear to be stimulated by different metabolic reactions during hypoxia than the chemoreceptors in carotid and aortic bodies.—M.S.


The increased oxygen affinity of Hb F is apparently due to the lesser degree of interaction between γ-chains and 2,3-DPG. The authors demonstrate that the in vivo effect of DPG on oxygen affinity of Hb F is approximately 40% of the effect of DPG on Hb A. Thus, the P_{50} of fetal blood can be estimated best by what the authors call the "Effective DPG Fractions," which is calculated from the equation DPG × (Hb A% + 0.4 × Hb F%). The P_{50} of fetal blood in-
creases with gestational age, as might be expected inasmuch as both DPG and Hb A% rise progressively during fetal life. —J.B.S.

LEUKOCYTES


The authors tested the hypothesis that the total length of the G-group chromosomes is shorter in patients with chronic lymphocytic leukemia than in controls. They studied five male and five female leukemic patients and a similar number of controls. Ten metaphases were examined from each subject, and the areas of the G and A1 chromosomes were measured by planimetry. No significant difference between leukemic and normal subjects was found.—A.A.M.


Rats were fed a protein-deficient diet for 8 wk, were sacrificed, and the number of neutrophils in the intestine was assessed both by direct counting of peroxidase-positive cells and by chemical assay of peroxidase. The number of neutrophils was greatly decreased when compared to controls. This finding may partly explain the high incidence of infection in protein deficiency. —A.A.M.


The cytotoxic effects of a number of rifamycin derivatives on normal and leukemic blood leukocytes were compared. Normal “blast” cells were obtained by stimulation with phytohemagglutinin; leukemic “blast” cells were obtained from the peripheral blood of untreated patients with acute lymphoblastic or acute myeloblastic leukemia. Toxicity was evaluated by the number of cells surviving in short-term culture. Rifamycin derivatives, which were potent inhibitors of viral reverse transcriptase and human DNA polymerase, were more toxic to leukemic than to normal leukocytes. However, the authors advance several reasons indicating that an effect on a DNA polymerase may not necessarily be the critical effect responsible for selective toxicity.—A.A.M.


This report describes the occurrence of myeloblastic leukemia, 8 mo to 6 yr after recovery from thrombocytopenia, associated with normal or increased number of megakaryocytes in the bone marrow and a reduced platelet life span.—J.M.P.


Three cases of reticulum cell sarcoma occurred in 151 renal allotransplant recipients during the course of 438 patient years of survival, an incidence of 0.7%/yr. This incidence was more than 100 times greater than in the general population and was far greater than that for other types of malignant tumors in transplant recipients. It is suggested that the presence of the foreign organ transplant together with the immunosuppressive therapy was responsible for this remarkable increase in the incidence of lymphoma.—J.E.U.

Delayed Hypersensitivity in Hodgkin’s Disease: A Study of 103 Untreated Patients. R. Young, M. Corder, H. Haynes, and V.

Delayed hypersensitivity in Hodgkin’s disease was examined in 103 untreated patients in all stages of disease and was related to prognosis. No relationship was found between skin test reactivity at the time of diagnosis and survival, frequency of relapses, or duration of remission. Skin test reactivity at the onset of Hodgkin’s disease is not a useful prognostic sign. Cutaneous anergy was uncommon in these patients; only 11.7% had no reaction to any of the six skin tests applied. No patient with stage I disease was anergic. The incidence of anergy increased with stage, but only 26.6% of the patients with stage IV disease were anergic. Mumps antigen and dinitrochlorobenzene (DNCB) were the most likely skin tests to rule out anergy. Skin test reactivity correlates with the absence of systemic symptoms, as well as with histologic type. In patients with nodular sclerosis and lymphocyte predominant patterns, the incidence of skin test reactivity is higher than in patients with mixed cellularity and lymphocyte-depleted Hodgkin’s disease. Absolute peripheral lymphocyte counts were noted to reflect staging and skin test reactivity. The mean absolute lymphocyte count increased. Peripheral lymphocytopenia at the onset of the patient’s course, however, did seem to be a bad prognostic sign.—J.E.U.


Two patients with advanced Hodgkin’s disease had persistent hypouricemia. Renal clearance studies showed elevated uric acid clearances of 35 and 23 ml/min, respectively. Only minimal abnormalities of other renal tubular functions were present. In both cases, the abnormal renal handling of uric acid was corrected with therapy of the Hodgkin’s disease, strongly suggesting that the disease was the causal factor. In one patient, Hodgkin’s disease and hypouricemia recurred, associated with a renal uric acid clearance of 20–25 ml/min, and studies with pyrazinamide were done. Assuming that pyrazinamide nearly completely blocks renal tubular secretion of uric acid in man, the study suggests that excessive secretion of uric acid was the cause of the serum and urine abnormalities.—J.E.U.


This review brings up-to-date current research and practices in the management of patients with lymphoma. The authors summarize current studies in correlation of histopathology with prognosis, emphasize the modifications of the approach to staging of the Ann Arbor Conference, and discuss results achieved in recent trials with radiotherapy or chemotherapy.—J.E.U.


Giant cell metaplasia of the alveolar epithelium of the lungs was noted in autopsies on 23 patients with acute or subacute leukemia. Such changes are characteristic of viral disease. On the basis of observations of his own and in the literature, the author argues for a viral etiology of acute leukemia.—J.V.


In 11 children with acute leukemia, the average therapeutic remission was 38.4 mo, and the average duration of the disease was 44.8 mo. Treatment was by various combinations of 6-MP, methotrexate, vinblastine, and prednisolone with supportive measures. Prolonged remissions were related to several factors: a relatively slow progress of the leukemia with no marked leukocytosis initially, aggressive therapy in the acute period, complete return to normal leukopoietic function, stability of indicators of
nonspecific immunity during treatment, and continued active therapy to control the course of the remission.—J.V.


In 18 children, 34 courses of treatment with L-asparaginase were carried out. The drug was administered every day in a single intravenous injection, with a mean dose of 347 μg/kg. The total dose of the drug used in one course was 2780 μg/kg, on the average. L-asparaginase was used jointly with prednisone and low-protein diet. The treated children were refractory to other methods of treatment, especially to cytostatic drugs, and were often in a late relapse of the disease. After one course of treatment with L-asparaginase, complete remission was obtained in 67% of cases. The most frequent toxic reactions were hypofibrinogenemia and hypoalbuminemia.—M.K.


An adolescent boy with acute lymphocytic leukemia, on prednisone and 6-MP, developed vomiting, weakness, generalized myalgia, and hypertension. Serum calcium was 19.1 mg/100 ml, and his ECG showed depressed ST segments but a normal QT interval. On parenteral fluid therapy plus oral phosphate solution, the serum calcium fell to 10.3 mg/100 ml, and he rapidly became asymptomatic. The patient succumbed to Pseudomonas sepsis several weeks later. At autopsy, in addition to widespread leukemia, calcific deposits were found in the lungs, kidneys, and coronary arteries.—J.B.S.


The authors report three cases of mixed lymphopenia due to sulfonylurea. These adverse effects on the blood may occur with any sulfonylurea compound and seem to be favored by the use of high doses, by sulfonamide association, and by certain hereditary or pathologic conditions, e.g., cirrhosis of the liver. In the three cases presented, the blood damage was neither peripheral nor central, but mixed. It is important to recognize this point, since maturation disorders of blood cells precede medullary aplasia. Prognosis depends on immediate discontinuation of the drug. During treatment with sulfonylurea, regular controls by blood cell counts and, at times, by bone marrow study are required.—J.C.

HEMOSTASIS

The Effects of Volatile General Anesthetics on Adenosine Diphosphate-induced Platelet Aggregation. I. Ueda. University of Utah College of Medicine, Salt Lake City, Ut. Anesthesiology 34:405, 1971.

The authors studied the effects of various volatile anesthetics on ADP-induced platelet aggregation. All anesthetics inhibited aggregation, and the partial pressures that inhibited aggregation by 50% showed a good correlation with those used clinically. By analogy with their effects on nervous tissue, the authors suggest that anesthetics may change the surface characteristics of platelet cell membranes, thereby interfering with their cohesion.—H.J.W.


The authors describe a radioimmunoassay for fibrinopeptide A. The antibody used in the assay was prepared by injecting rabbits with native fibrinopeptide coupled to albumin. N-Tyrosyl fibrinopeptide A was synthesized by the solid-phase method and was iodinated with 125I by the chloramine-T method. The specificity of the assay for fibrinopeptide A was high in comparison with other proteins, and the sensitivity of

Human blood platelets are completely and irreversibly aggregated by phospholipase C from Clostridium welchii. The duration of the lag phase and the maximal velocity of aggregation depend on the amount of enzyme added. The inhibition of aggregation by lecithin confirms the effect of phospholipase C, which moreover hydrolyzes platelet phospholipids. The mechanism of this phenomenon is not elucidated, but is related to the adsorption properties of clotting factors.—J.C.


The method is based on the phenomenon of impedance presented by any biological suspension when crossed by a low frequency current and was applied to the study of the changes (volume and shape) that occur in platelets in the presence of aggregating agents. Platelet survival was also measured. It was found that value of impedance was directly related to platelet volume. Swelling in the presence of ADP, collagen, adrenaline, hypo-, or hypertonic NaCl could be rapidly measured by this method. The study of impedance during platelet preservation showed an increase in cell volume after 24 hr and a total lysis after 80 hr.—J.C.


The test of platelet aggregation according to Born was used in a comparative study of a population of 103 controls and 93 patients with transient or permanent cerebral ischemia. The results were fed into a computer. The curves were classified according to the intensity of platelet aggregation at the fifth minute. This shows the presence or absence of platelet secretion (release reaction). "Platelet hyperaggregability" was defined as the property of platelets from a given subject to release a sufficient quantity of ADP to produce a second wave of aggregation when placed in the presence of a small amount of exogenous ADP. The patients presented this phenomenon much more often than the controls. Treatment with Aspirin acted effectively against hyperaggregability, but it did not change the aggregation curves. The authors discuss the significance of hyperaggregability and the value of Aspirin treatment for the prevention of the thrombotic complications of atheroma.—J.C.


Trypsin and chymotrypsinlike activities, as well as elastase activity, have been identified in human blood platelets lysed by Triton X 100, using synthetic substrates specific for these three types of enzymes. Particularly, the hydrolysis of trialanine methyl ester (specific for pancreatic elastase) by platelet extracts confirms the presence of an elastolytic enzyme in blood platelets. This enzyme has been partially purified, and a high elastase activity has been recovered at the end of the purification procedure.—J.C.

Sialic acid is responsible for negative electric charge of blood platelets. In thrombasthenia an abnormality of the membrane is suggested; the authors report a moderate decrease of sialic acid with an increase of L-fucose. In some acquired thrombocytopenias occurring during malignant hematologic disorders, an important diminution of sialic acid associated with a megakaryocyte qualitative defect has been observed.

—J.C.


It is well known that high-speed centrifugation of platelet-rich plasma can cause platelets to aggregate. This is due to a release reaction induced by the close proximity of platelets during the centrifugation. Prostaglandin E₁ (PGE₁) is one of the most potent inhibitors of platelet aggregation known. The study was designed to investigate the effect of PGE₁ on the aggregation and shape of platelets during their collection and storage. PGE₁ was added in varying concentrations ranging from 0 to 60 ng/ml to platelet-rich plasma. After centrifugation at room temperature, the shape of the resultant platelet pellet was examined. While platelets that were originally centrifuged without or with low concentration of PGE₁ (equal to or less than 2 ng/ml) spread in a thin film over the bottom of the test tube, platelets mixed with concentrations of PGE₁ (equal to or greater than 5 ng/ml) lay in a smooth, clearly defined round pellet. Furthermore, control samples without PGE₁ showed macroscopic aggregates, whereas PGE₁-treated platelet concentrates had only a few microscopic aggregates and produced a smooth suspension of platelets. The shape of the platelets was flat when PGE₁ was added (equal to or greater than 10 ng/ml), while control platelets appeared spherical. The effects of long-term storage at 4°C were also studied. Aggregation induced by cold storage and warming was measured quantitatively at 1 hr and during 1–4 days of storage. While in control samples aggregation increased rapidly during the first day of storage, platelet concentrates containing PGE₁ (20 ng/ml added before cooling) showed markedly less aggregation than did the controls. Although aggregation increased with time of storage, it was always less than that of the control. When platelet concentrates were stored at room temperature, PGE₁ added during the preparation of those concentrates prevented almost completely the formation of platelet aggregates during the first two days of storage. In the controls, marked aggregation was observed after only 1 day of storage. These results demonstrate that PGE₁ in concentrations of 10⁻⁶–10⁻⁷ M is effective in improving the preparation of human platelet concentrates from platelet-rich plasma.—M.S.


Studies of 38 children with Werlhof’s Disease (ITP) revealed that adrenal function is impaired in this disease, the disturbance being related to its severity. In mild cases only adrenaline synthesis is inhibited, while in more severe cases noradrenaline is also inhibited. These disturbances have a statistically valid relationship to abnormalities of the coagulation system; the more depressed the adrenal function the less thromboplastin is produced, with impaired utilization of prothrombin in the coagulation process.—J.V.


Hypoplastic thrombocytopenia of short duration (generally less than 4 mo) with platelet levels varying between 9000 and 60,000 was found in five out of 21 newborns having trisomy 21. A preliminary report on two cases appeared in Pédiatrie 26:857, 1971.—J.M.P.
IMMUNOHEMATOLOGY


The fluorescent treponemal antibody test (FTA), while generally considered specific for luetic infection, has occasionally reported to be positive in patients with systemic lupus erythematosus (SLE). An adolescent girl with SLE had a positive VDRL and an atypical positive reaction to FTA. This reaction, which from previous reports is probably suggestive of lupus, is characterized by the presence of multiple fluorescent globules along the length of the treponema, with no fluorescence between the organisms.


Using the Farr ammonium sulfate technique, anti-DNA was found in sera of children with systemic lupus erythematosus (SLE) but not in youngsters with juvenile rheumatoid arthritis. All children with untreated active SLE demonstrated more than 40% DNA-bound precipitate.

MISCELLANEOUS


Hematopoietic depression caused by cytostatic therapy was treated in 31 patients by transplantation of allogeneic marrow, from 0.6 to 8.3 \times 10^9 nucleated marrow cells being given. In 28 patients, hematologic indices were rapidly restored, and after 2 wk cytostatic therapy could be resumed if required.


Determination of the red cell life span by labeling with radioactive chromium and study of iron kinetics by radioactive iron are essential before deciding on splenectomy to correct the anemia of myelofibrosis with myeloid metaplasia. Two cases where splenectomy brought about disappearance of the anemia for 5 and 16 mo, respectively, are reported. In each case, the anemia was hemolytic in type with a rapid disappearance of red cell radioactivity from the circulation (7.8% and 7% per day, respectively). The splenic uptake study, using ^59Fe, showed that the spleen was not the only erythropoietic organ and that there was no major erythropoietic insufficiency. The anemic forms of myelofibrosis with myeloid metaplasia of the spleen justify splenectomy when the following conditions are fulfilled: anemia of hemolytic origin, not due to insufficient erythropoiesis; splenic uptake of red cells; and absence of erythropoietic predominance of the spleen.