ABSTRACTS

ERNST R. JAFFE, M.D., Editor

ABSTRACTERS

P. Barkhan, M.D., London, England
T. H. Bothwell, M.D., Johannesburg, South Africa
T. E. Brittingham, M.D., Nashville, Tenn.
Jacque Caen, M.D., Paris, France
J. B. Chatterjea, M.D., Calcutta, India
Pietro deNicola, M.D., Pavia, Italy
Ludvik Donner, M.D., Prague, Czechoslovakia
A. J. Erslev, M.D., Philadelphia
H. Hugh Fudenberg, M.D., San Francisco
Katsuhiro Fukutake, M.D., Tokyo, Japan
Robert Goldstein, M.D., New York City
Ira Green, M.D., New York City
F. W. Gunz, M.D., Christchurch, New Zealand
Susanna R. Hollan, M.D., Budapest, Hungary
Harry S. Jacob, M.D., Boston, Mass.
Michel Jamra, M.D., Sao Paulo, Brazil
Oliver P. Jones, M.D., Buffalo
Joseph Kassirsky, M.D., Moscow, U.S.S.R.
Sven-Age Killmann, M.D., Copenhagen, Denmark
Frederick A. Klipstein, M.D., New York City
E. Kowalski, M.D., Warsaw, Poland
E. A. Loeliger, M.D., Leiden, The Netherlands
Michel Jamra, M.D., Sao Paulo, Brazil
Edgardo S. Sack, M.D., Buenos Aires, Argentina
Julian B. Schorr, M.D., New York City
Ralph O. Wallerstein, M.D., San Francisco


Adult New Zealand rabbits given 500 or 550 r of x-radiation, followed 2 and 7 days later by intravenous injections totaling 300 mg. of bovine serum albumin (BSA), were made tolerant to BSA but not to HCG. They were unable to make antibodies against BSA antigen when appropriately challenged 2 months later. The amount of BSA per Kg. required to induce tolerance appeared to be approximately the same for irradiated adult rabbits and for neonates.—H. H. F.


The capacity of adult male mice to respond to injection of sheep erythrocytes by production of hemolytic plaque-forming cells was decreased by irradiation (Co60 r-rays). The spleens of normal mice contained a minimum of 10^6 cells which did not produce hemolysin, but which responded to injection of antigen by proliferation and differentiation into hemolysin-producing cells. When these cells, which may be termed antigensensitive cells, were rendered incapable of proliferation by radiation, they were not replaced to a significant degree for at least 10 days.—H. H. F.


Genetic factors evidently influence the electrophoretic mobility of antibody in mice. Some of the factors determining these differences in the electrophoretic mobility of immunoglobulin molecules are independent of antigen-binding (antibody) sites and the skin-sensitizing site.—H. H. F.

HEMOSTASIS


Twenty-four women with profuse menstruation were treated with e-ACA. Six had a generalized hemorrhagic diathesis, while the remaining 18 were otherwise healthy. Pelvic examination was normal and curettage had been without effect on
uterine bleeding. Oral doses of ε-ACA reduced menstrual blood flow in all patients. In some cases, the effect was dramatic. Uterine blood flow was not measured directly, but the effectiveness of treatment was substantiated by a definite rise in the hemoglobin concentration of these anemic patients. The dose of ε-ACA was 1 Gm. per 10 Kg. body weight. In order to avoid possible teratogenic effects, the drug was not started until menstrual blood flow had become profuse. The dose was repeated as needed; usually 2 to 4 doses were necessary during the first 2 days and 1 or 2 doses on the third day. Fifteen patients complained of dizziness and/or nausea and, for this reason, treatment had to be discontinued in seven. Two patients became pregnant after ε-ACA had been stopped and both gave birth to normal children.—S. A. K.


The biological properties of EACA, prepared in the Department of Physiological Chemistry of Białystok Medical School and in Plant "Ziolołek" Poznan, were investigated. The LD50 of EACA for mice was 80 times greater than the therapeutic dose for man. EACA was nontoxic during prolonged administration to dogs and rats. A new method for determining EACA in blood serum was elaborated and it was found that EACA is rapidly excreted and does not accumulate, even when administration is prolonged. Thirty-two patients with gynecological and obstetrical bleeding, when administration is prolonged. Thirty-two patients became pregnant after ε-ACA had been stopped and both gave birth to normal children.—S. A. K.


A child with the Kasabach-Meritt syndrome was described. There were no chromosomal anomalies and no immune bodies. The presence of a hemorrhagic syndrome with thrombocytopenia in a number of other congenital, chiefly benign, vascular tumors was emphasized and individual clinical forms were characterized. The term “Kasabach-Meritt syndrome” should be used only for a solitary gigantic hemangioma with thrombocytopenic purpura.—J. K.


Twelve children with an acute form of Werlhof’s disease were treated with prednisolone, 1.5-3 mg. per Kg. On days 9 to 22 of therapy, an active participation of young and polychromatophilic megakaryocytes in thrombocyte formation was demonstrated. The percentage of functioning megakaryocytes became normal on the 32nd to 50th day.—J. K.


Eight cases of chronic benzene poisoning with variable degrees of thrombocytopenia were studied. Prolongation of the reaction and clot formation time were observed in the majority of cases, as well as a rather marked reduction in maximal amplitude. Alterations were most marked with severe thrombocytopenia. Patients’ platelets were not as effective as normal platelets in normalizing the maximal amplitude of normal, platelet-free plasma. A thrombopathic component in benzene poisoning was postulated.—P. d. N.


Intact erythrocytes considerably accelerated the recalcification time and the rate of dense clot formation in the thrombotest. Blood always coagulated more rapidly than did unstabilized plasma. This effect depended on the presence of thromboplastic factors in erythrocytes and the capacity of erythrocytes to bind heparin and to influence the adhesiveness of thrombocytes. The author also considered that, in the presence of erythrocytes serving as a base for thrombus formation, less fibrin was required for appearance of a dense clot. In the author’s opinion, intact erythrocytes were capable of discharging compounds possessing thromboplastic activity into the plasma, leading to a rise in thrombotest values and a reduction in recalcification times. The data obtained help to explain why, in diseases accompanied by marked thrombocytopenia, the blood coagulation time often remains normal.—J. K.

ON PLASMIN ACTIVATION IN CLOTTING BLOOD. M. Kotschy. From the Medical School, Wroc-
Activation of plasminogen is linked with the generation of thrombin. The basic phenomenon responsible for activation of fibrinolysis during blood coagulation is the so-called "thrombino-genetic disinhibition of plaminogenesis," i.e., the inactivation of a postulated inhibitor of plasminogenesis, probably by different esterases formed in the course of prothrombin activation. The conformational structure of the fibrin network is particularly suited as a matrix for plasmin formation.—E. K.


The clotting and arginine esterase activities of the "active contact factor" fractions (alkaline kaolin eluates) were investigated. A close correlation between both activities, except in fish and frog plasma, was observed. Formation of the active contact factor was ascertained in all mammalian plasmas, but not in bird, human Hageman-deficient or PTA-deficient plasma. The active contact factor was formed in a mixture of chicken and normal human or PTA-deficient plasma and in a mixture of human Hageman factor and PTA-deficient plasma, but not in a mixture of chicken and human Hageman-deficient plasma. The lack of Hageman factor in bird plasma was confirmed. Arginine esterase activity may be connected with "activation product" or "active contact factor."—E. K.


A decrease in Factor X was observed in normal blood, beginning 30 minutes after the onset of coagulation and continuing for 120 minutes. Consumption of Factor X in blood of patients with hemophilia A, hemophilia B and thrombocytopaenia was decreased. A certain degree of parallelism between Factor X and prothrombin consumption was noted. The activity of Factor VII increased during coagulation of normal blood and blood with a coagulation defect.—L. D.


The author studied the effect of various concentrations of heparin, thrombin, fibrinogen, antihemophilic globulin, albumin, globulin and tissue and blood thromboplastin on spontaneous fibrinolysis in whole blood, fibrinolysis in whole blood activated by streptokinase and pure plasmin and fibrinolysis by the euglobulin fraction. Substances were added to blood before coagulation. Heparin accelerated fibrinolysis in whole blood (82.6 per cent of cases) and in whole blood activated by streptokinase or plasmin, but inhibited fibrinolysis by the euglobulin fraction. Fibrinolysis by streptokinase was retarded by increased amounts of thrombin, various concentrations of antihemophilic globulin, pure globulin, fibrinogen, and tissue and blood thromboplastin prepared from the same blood. Tissue thromboplastin enhanced euglobulin fibrinolysis.—L. D.


The authors examined 37 patients who were treated with intravenous streptokinase injections. In all instances, a marked increase in the rate of euglobulin fibrinolysis was observed. Plasminogen and proactivator and, to a lesser extent, fibrinogen decreased. In the thrombelastogram, the "ma" value disappeared and only a straight line was seen. The thrombin time was prolonged and the prothrombin complex was reduced. Very satisfactory therapeutic results were obtained, especially in those instances where the thrombin time was prolonged 4 to 5 times and where there was a considerable drop in plasminogen. Hemorrhage was very rare, usually manifested only by subcutaneous hematomas at the site of injection. With hemorrhagic manifestations, the bleeding time was prolonged and the adhesiveness and agglomeration of platelets were reduced—L. D.


A new procedure, based on tannin precipitation and subsequent caffeine elution, was described.—E. K.
LEUKOCYTES


The author reports on 2 girls with 8 and 6½ year remissions of acute leukemia. One was treated initially with corticosteroids and the other with 6-mercaptopurine. Both children have been on continuous maintenance therapy with 6-MP (50 mg. daily), which does not appear to have interfered with growth, secondary sexual development or resistance to infection.—P. B.

ALEUIU.RSII: FORMSIS WITH 6-MERCAPTOPURINE. Both children have been given 6-mercaptopurine. Both children have been on continuous maintenance therapy with 6-MP (50 mg. daily), which does not appear to have interfered with growth, secondary sexual development or resistance to infection.—P. B.

CONCLUSION THAT A HEREDITARY TENDENCY WAS PRESENT WITH THE SAME CLINICAL MANIFESTATIONS LED TO THE PHYLLOGIC CHANGES IN THE HEMATOPOIETIC SYSTEM AND WITH SIMULTANEOUS ONSET OF LEUKEMIA AND BLIND REPEATED CULTURE THROUGH INOCULATION CAUSED DEATH OF THE MICE IN 24 TO 48 HOURS.

ACHIEVING A REMISSION IN ACUTE LEUKAEMIA. P. B. I. K.

ALEUIU.RSII: CONCLUSION THAT A HEREDITARY TENDENCY WAS PRESENT WITH THE SAME CLINICAL MANIFESTATIONS LED TO THE PHYLLOGIC CHANGES IN THE HEMATOPOIETIC SYSTEM AND WITH SIMULTANEOUS ONSET OF LEUKEMIA AND BLIND REPEATED CULTURE THROUGH INOCULATION CAUSED DEATH OF THE MICE IN 24 TO 48 HOURS.


Chronic lympholeukemia appeared in two identical twins at age 56. The disease pursued a typical course and lasted about three years. The simultaneous onset of leukemia with similar morphologic changes in the hematopoietic system and with the same clinical manifestations led to the conclusion that a hereditary tendency was present in these cases.—J. K.


Acute hemocytoblastosis was described in identical twin boys, aged 19 months. The course and morphologic peculiarities were the same in both.—J. K.


The authors were unable to isolate viral agents from 19 bone-marrow cell suspensions obtained from 17 children with acute leukemia after 4-6 blind passages in human embryonic kidney cells.—P. B.


The original observation of high-energy production in leukemic patients is confirmed in only a minority of patients with malignant disease. With similar dietary intakes, pregnant mice gain weight and tumor bearing mice lose weight. Hyperalimentation temporarily reduces loss of host tissue, but may increase tumor growth. Patients with leukemias and lymphomas often have folic acid depletion. (Abstracter's note: Or, rather, a disturbed folic acid metabolism.) Why the tumor bearing animals "starve in the midst of plenty" is not known.—P. G. R.


Nine cases with the clinical and pathologic characteristics of Burkitt tumor were described: 7 in children living in the São Paulo area and 2 in Paraná. The geography of these regions (latitude, altitude and temperature) resembled that of the areas studied by Burkitt. The illness was always acute, ages ranged from 2 to 13 years, and every case had at the initial clinical examination a lesion located in the maxillo-facial region without systemic symptoms. Seven were without regional adenopathy, but 2 had adenopathy on the same side as the tumor. The histopathologic features were round cell neoplasia with cells of the mature lymphoid type. In some lymph nodes the "starry sky" picture was present. One case showed a pattern of reticulum cell sarcoma.—M. J.

STUDIES ON AN EPIDEMIC OF INFECTIOUS LYMPHOCYTOSIS IN A CHILDREN'S INSTITUTION. M. J. Grabowska. From the Medical Department Hospital, Gubin, Poland. Pediat. Pol. 39:1153, 1964.

In the winter of 1960-1961, an epidemic of infectious lymphocytosis was observed at a kindergarten where 37 of 82 children fell ill. Lymphocytosis with a predominance of large lymphocytes and the presence of Gumprecht's shadows were observed. Further observations for 12 months revealed abortive cases in the winter of 1961-1962. Intranasal inoculation of white mice with throat washings from children in the first days of illness caused death of the mice in 24 to 48 hours. Changes were present in the lungs. Intraperitoneal inoculation and blind repeated culture through chick embryos gave negative results. Inoculation of a sick child's blood into an adult intravenously gave no result. These observations confirm an infective etiology and are suggestive of a droplet method of spread.—E. K.

NEUROLOGIC SYNDROMES IN HODGKIN'S DISEASE. C. Bertin and G. Zeni. From the University,
ABSTRACTS


Forty-two of 203 cases (20.6 per cent) exhibited neurologic involvement. Four cases had cerebral syndromes, 4 radiculo-medullary syndromes and 34 lesions of peripheral nerves.
—P. d. N.


The nucleolar coefficient (NC) of lymphocytes in the peripheral blood and the absolute and relative counts of uninucleolar and multinucleolar lymphocytes were studied in 36 patients with Hodgkin's disease and in 100 normals. No age or sex differences were found in the control group.

In patients with advanced disease, a rise in the NC was noted. A drop in absolute uninucleolar lymphocytes was seen in patients in remission. The NC decreased in all patients following X-ray treatment and chemotherapy. —L. D.


Follow-up of 5 patients with anticomplementary sera, detected during routine serology studies in 1945-1952, revealed that in each serum an M-component was demonstrated when studied electrophoretically in 1951-1953. In the meantime, a diagnosis of multiple myeloma had been established in all. One patient died from coronary occlusion 6 years after demonstration of anticomplementary activity, while the remaining 4 were alive 10-17 years after detection of anticomplementary activity and approximately 10-12 years after demonstration of the M-component. —S. A. K.


Fifty patients with thyrotoxicosis and 50 healthy subjects were investigated. All thyrotoxic patients had increased adenosinetriphosphatase activity in their leukocytes. Increased enzymatic activity was found only 6 times in 40 patients with other disorders. —L. D.


Observations on the microspectrophotometric analysis of the nucleoprotein of mature thymocytes, before and after administration of antigen, were presented. These methods may be capable of revealing important cytochemical features involved in the earliest responses of lymphoid cells to antigens. —O. P. J.

AN ELECTRON MICROSCOPIC STUDY OF LYMPHATIC TISSUE IN RUNT DISEASE. L. Weiss and A. C. Aasen. From The Johns Hopkins University School of Medicine, Baltimore, Md. J. Cell Biol. 25:149-177, 1965.

Runt disease is induced by injection of immunologically competent homologous cells into an animal, typically a newborn, unable to destroy such cells promptly. An electron microscopic study of spleen, thymus, and lymph nodes in runt disease of rats is described. The participation of lymphocytes, plasma cells and histiocytes indicates a complex process involving both delayed hypersensitivity, presumably mediated by lymphocytes, and immediate hypersensitivity, associated with the plasma cells and the production of antibody. —O. P. J.


The modifications consisted of: (1) the separation of white and red blood cells by low speed centrifugation, after 1-4 hours of spontaneous sedimentation, and (2) leukocytes grown in plasma without addition of phytohemagglutinins. —E. K.


Exposure of human lymphocyte cultures to pharmacologic concentrations of thalidomide resulted in chromosome aberrations (mainly chromatid and isochromatid breaks) in one-fourth to one-
third of metaphases. This procedure may have potential value as a simple method for screening drugs with respect to possible teratogenic and carcinogenic effects.—S.-A. K.


Kariological analysis (determination of mitotic indices, chromosome count and morphology) was carried out in cells of marrow and spleen of mice of the CBA and C57BL strains suffering from leukemoid reactions induced with endotoxin of the salmonella group. Even with increased proliferative activity of myeloid tissue, no alterations were revealed in chromosomes of these cells.—J. K.

ERYTHROCYTES


Haptoglobin values were 216 ± 83 mg. per cent in a group of 1143 professional blood donors; males 223 ± 88 mg., and females 188 ± 79 mg. The professional character of the donors might have been the reason for the high values. No statistically significant difference was found among the genetic types of haptoglobin. — E. S.


In 11 Iranian males with long-standing dietary iron deficiency, D-xylose absorption, iron absorption, serum proteins and intestinal biopsy were all essentially within normal limits; several patients had achlorhydria. Patients responded normally to oral iron therapy.—R. O. W.


Twenty-four patients with liver disease and 10 without received 15–30 mC. Fe59 in 50 mg. iron orally. Body retention of iron was measured with a simple shielded crystal, calibrated by giving 10-15 mC. iron i.v. to each subject 2–3 weeks after the oral dose. Red cell iron uptake, bone marrow iron concentrations and histologic iron concentrations in parenchymal cells, portal areas and Kupffer cells of liver slices were also determined. Two patients with hemochromatosis and one with transfusion hemosiderosis absorbed iron normally. Most non-iron-deficient cirrhotic patients absorbed iron normally, but a few had high absorptions. The authors suggested that enhanced absorption of iron occurs with liver cirrhosis. (Abstracter's note: Perhaps because of simultaneous pancreatic fibrosis?) — P. G. R.


Twenty-one normals and 21 iron-deficient subjects were given radioactive wheat to compare its absorption with that of ferrous ascorbate, hemooglobin iron and ferritin iron. Normal subjects absorbed similar amounts of iron (4.5 to 11.7 per cent) of the various forms tested. Iron deficient subjects absorbed 6 times as much ferrous ascorbate, but were unable to increase wheat iron absorption significantly.—R. O. W.


An attempt was made to select a model for "internal" (excretion excluded) iron turnover. Unlike Pollycove and like Stohlman, the author used short-term plasma iron disappearance curves plus erythrocyte appearance curves, rather than long-term plasma iron disappearance curves. Curves were fitted with a digital computer. An initial model, where all erythrocyte maturation stages were assumed to take up iron at the same rate and where there was a large exchangeable pool between erythroblasts and plasma, could not be made to fit. In a second model, this assumption was not made, but this model made numerical integration necessary and mathematical treatment difficult. This model could be made to fit, provided a certain amount of inefficient erythropoiesis was assumed. Radio-iron studies in infants (1) were also performed, showing fecal excretion of about 0.03 mg. iron/Kg. body weight.—P. G. R.

The degree of anemia and the amount of blood lost by latent gastric hemorrhage in 25 patients with cancer of the stomach were compared. The blood loss during 6 days was determined by the Cr¹⁹ method. A distinct relationship between the degree of anemia and the intensity of hemorrhage was noted. It was concluded that latent bleeding is a decisive factor in the development of anemia accompanying cancer of the stomach.—J. K.


Serum iron, transferrin and conalbumin were studied. In hens, the possible intervention of conalbumin in the transport of serum iron as an auxiliary element of transferrin during the laying period was confirmed. Conalbumin represented 73 per cent of the total transport capacity. In turkey and pigeons, conalbumin represented 27 and 11 per cent, respectively, of the total transport capacity and played a small role during the laying period. In ducks, conalbumin constituted an active transport mechanism and represented 53 per cent of the total capacity.—E. S.


The average amount of each mineral studied was increased above normal in vitamin B₁₂ deficiency states and was reduced in iron deficiency states, except for calcium which was usually increased.—F. A. K.


C₁₃-2-methyl-2 amino-propanol (1)-B₁₂ had the highest anti-B₁₂ activity in bacteria and chicks and competed with human B₁₂ absorption 3 times more than did C₁₃-DL-1 phenyl-2-amino-ethanol B₁₂ which had less anti-B₁₂ activity. The incomplete B₁₂ analog, cobinamid, competed still less. The first analog (100 μg.) prevented the erythropoietic effect of 1 μg. B₁₂ in patients with pernicious anemia in relapse and competed with B₁₂ tissue uptake.—F. G. R.


Hematologic studies were reported in 34 patients with kwashiorkor. Iron deficiency was common and various degrees of megaloblastic changes in the bone marrow were found in 12 of 17 patients who had marrow aspirations. Serum vitamin B₁₂ levels varied widely and correlated poorly with megaloblastic changes. Serum vitamin E levels were subnormal in all 14 patients assayed, and 9 of these patients had megaloblastic changes. The authors suggested that deficiency of this vitamin may be related to the development of macrocytic anemia in kwashiorkor. Serum folate determinations were not performed.—F. A. K.


Investigations into the nature of the active principle present in the proximal area and mid-ileum of the rat small intestine responsible for the release of vitamin B₁₂ from its bound form to rat stomach extract or normal human gastric juice gave results which suggest that this factor is principally endogenous vitamin B₁₂ which equilibrates with the bound form.—F. A. K.


Urinary excretion of FIGLU may be excessive during early pregnancy, probably as a result of abnormal histidine metabolism, serum folate con-
This observation suggests that the urinary excretion of FIGLU, and vitamin B19 supplements administered vitamin concentrations show little change, and serum in subnormal serum vitamin B12 concentrations. As a result, decreased urinary excretion of FIGLU, this vitamin-F.

be a reflection of depletion of maternal stores of that subnormal serum vitamin B12 levels may not be a reflection of depletion of maternal stores of this vitamin.—F. A. K.


Nine patients with vitamin B12 deficiency excreted methylmalonic acid in the urine. There appeared to be no correlation between the amount excreted and the degree of hematologic and neurologic deficiency. Methylmalonic acid disappeared from the urine at different rates in patients receiving similar modes of vitamin B12 replacement therapy and, in 4 patients, excessive urinary excretion persisted after return of the serum vitamin B12 concentration to normal and correction of the hematologic abnormalities.—F. A. K.


A family with 8 cases of methemoglobinemia in 2 generations was reported. Hb $M_{\text{Arhus}}$ was shown to be transmitted as a dominant trait. Spectroscopy of the acid methemoglobin showed a maximum at 600 $\mu m$. This methemoglobin reacted more slowly with cyanide than did methemoglobin $A_A$, whereas the reactions with dithionite were identical. The abnormal hemoglobin was calculated to make up 50 per cent of the total hemoglobin. Subsequent fingerprinting and amino acid analysis have proved Hb $M_{\text{Arhus}}$ to be identical with Hb $M_{\text{Katowice}}$—S. A. K.

**Studies on Sulphaemoglobin Formation.** A. Koj and J. Frendo. From the Medical School, Kraków, Poland. Acta Biochim. Pol. 11:211, 1964.

The reaction of oxyhemoglobin with hydrogen sulphide was studied with intact erythrocytes and hemolysates. Phenacetin, phenylhydrazine and hydroxylamine accelerated, while glutathione, cysteine and ergothioneine inhibited the reaction. The -SH compounds in erythrocytes appeared to participate in a mechanism protecting hemoglobin from conversion to sulphhemoglobin. The findings supported the suggestion that peroxide groups are involved in sulphhemoglobin formation.—E. K.


One group comprised 10 cases not splenectomized; the second, 5 cases after splenectomy. In both groups, glutathione values before and after incubation with phenylhydrazine and acetylcholinesterase were normal. Higher values for acid phosphatases, glycolysis, oxygen consumption in the presence of methylene blue and erythrocyte pentoses were found in the first group. All values were normal in the splenectomized group. The differences may be explained by the increased number of reticulocytes in nonsplenectomized patients.—L. D.


Inorganic and organic phosphates of red cell hemolysates were shown to be bound to hemoglobin in the absence of salts. No evidence was available to indicate the type of interaction. The organic phosphate concentration of hemolysates decreased at different rates after dialysis against cacodylate buffers, sodium chloride solutions and diethylbarbiturate buffers.—E. K.


The ATP content was higher in males than in females.—E. K.

The Wachstein and Meisel (1957) technic was used with electron microscopy. Blood cells from two amphibian species—the newt, Triturus cristatus, and the edible frog, Rana esculenta—and from two mammalian species—the rat and the rabbit—were used. Washed and unwashed rat and rabbit erythrocytes showed no ATPase activity and were always unstained, whereas both frog and newt erythrocytes had ATPase activity on the outer surface of their plasma membranes which hydrolyzed ATP and ADP, but not AMP or Na β-glycerophosphate. The plasma membranes of amphibian basophilic granulocytes were stained after incubation in media containing either ATP or ADP and the staining was most intense on parts of the cell surface thrown into processes and projections. The plasma membranes of neutrophilic granulocytes and lymphocytes were unstained, even when they were thrown into processes and projections.—O. P. J.


The technic described was based on the conversion of heme to a porphyrin which fluoresces and which can be detected readily with the ultraviolet light microscope. Hemoglobin was demonstrated in nuclei of avian erythrocytes and in nuclei of human normoblasts at an earlier stage than previously described. Heme proteins, presumably cytochromes, have been detected in the cytoplasm and nuclei of myelocytes, in thymus lymphocyte nuclei, in chick embryo liver cytoplasm and in chick embryo somites.—O. P. J.


The chick blastoderm simulates a phased culture of erythroid cells in which hemoglobin synthesis has been studied from the precursor to the fully hemoglobinized cell. In these erythroid precursor cells, the limiting enzyme in heme synthetis is ALA synthetase. Bypassing ALA synthetase by addition of ALA to the medium stimulates globin synthesis and hemoglobin formation. Studies with actinomycin D and puromycin suggest that heme stimulates synthesis of globin at the ribosome-mRNA level; globin synthesis is limited by the availability of heme.—H. H. F.


These two studies are interesting and important because they were based on fetal livers obtained from 9 human embryos 7–20 weeks of age. Two new observations will require additional study and confirmation. The first was the appearance of the hemocytoblast nucleus with its deeply indented profile. The second was that extruded erythroblastic nuclei did not have a narrow rim of cytoplasm. Ferritin-containing invaginations of the erythroblastic membrane were encountered with extreme rarity and certainly not often enough to support the hypothesis of direct iron transfer advanced by Bessis and his group. Other authors have observed neutrophilic myelocytes and tissue mast cells developing extravascularly between hepatic cords, but these were not observed here. The lack of leukopoiesis may have been a matter of sampling.—O. P. J.

MISCELLANEOUS


Chromium-labeled strain A red cells which survived normally in H-2 incompatible recipients induced the formation of humoral antibodies directed against themselves. Transfusion of erythrocytes into actively immunized recipients resulted in the rapid elimination of one fraction, whereas the surviving fraction was eliminated at the same rate as in isologous hosts. Analogous results were obtained after transfusion into H-2 incompatible recipients passively immunized with humoral anti-H-2 antibodies. The failure of antibodies to eliminate all transfused cells was not caused by the existence of a fraction of antibody-resistant cells,
since the antibodies caused complete lysis of the red cells in vitro in the presence of complement. It was suggested that a combination of antibodies and some unknown host factors induced a change in the red cells (the cells no longer contained free antigenic determinants?) that made them resistant to isomimmune destruction.—H. H. F.


Human red cells (1 ml.) were injected into rabbits 3 times a week for 441 days. The antibody titer reached the highest peak (1:1226) on the 55th day and was stable until the 96th. A brisk reduction was observed on the 118th day, and thereafter a gradual diminution in titer was observed until the end of the experiment, interrupted only by a low peak on the 281st day. The final titer was 1:310.—E. S.


Rabbits were immunized with human serum, ovalbumin and sheep erythrocytes. Examined with various antigens, the rabbits fed with chloroquine showed the same antibody production as control rabbits which received no chloroquine.—S. R. H.


The embryonic tissue mast cell arises, proliferates and matures in a manner analogous to the process of granulopoiesis. This sequence has been followed in rat embryos ranging in crown-rump length from 16.5 to 45.0 mm. The mast cell arises from a relatively undifferentiated cell of mesodermal origin which cannot be distinguished from other primitive mesenchymal cells and is first recognizable by the appearance of specific cytoplasmic granules. In both mast cells and granulocytes, mitotic proliferation continues until many cytoplasmic granules are present, but essentially ceases at a certain point after which maturation is completed by a process of granule formation and morphologic alteration. Unlike the granulocytes, mast cells in embryos do not arise in hematopoietic tissue, but rather in loose connective tissue. The process of formation of mast cell granules has been interpreted as reflecting the synthesis and accumulation of highly N-sulfated heparin, along with mast cell chymose and, finally, histamine in safranin-positive granules.—O. P. J.


No adequate description of ultrastructures of germinal centers exists, possibly owing to difficulties of orientation and to the cyclic activities and changes which these structures undergo. The characteristic large cell of the germinal center has a basophilic cytoplasm. This paper describes the fine structure of these rapidly proliferating cells labeled with tritiated thymidine in antigen-stimulated mouse spleen. Reticular cells, both phagocytic and nonphagocytic, are not observed to be labeled in these centers.—O. P. J.


An editorial annotation to point out the possibility of controlling certain genetic blood disorders through elimination of incompatible environmental stresses.—J. B. C.


Preservation of blood with glucose-heparin solution for 4 to 8 days at 2 to 4 C. has been studied. Morphologic changes during the first 24-48 hours are relatively small. In the course of storage, changes increase. The hemolysis of red cells increases and their osmotic fragility decreases. Heparinized blood is suitable for transfusion only within the first 48 hours when 90 per cent of transfused cells can be identified 24 hours later. The withdrawal of blood into heparin influences unfavorably the prothrombin complex factors, except for Factor VII. After the third day of storage, clot retraction is diminished and fibrinolysis is enhanced.—L. D.

PRESERVATION OF RED CELL: OBSERVATIONS ON CERTAIN METABOLIC CHANGES IN STORED...
ABSTRACTS


Changes in the patterns of autohemolysis, plasma hemoglobin and erythrocyte reduced glutathione are reported.—J. B. C.


The relationship between peptic ulcer and blood groups A1, A2, B, and O was studied in 1452 patients. A highly significant increased incidence of blood group O, a highly significant decreased incidence of blood groups A1 and A2, and a significantly increased incidence of blood group AB was found in patients with duodenal peptic ulcer. In patients with gastric and combined peptic ulcers, no significant differences were found. The incidence of Rh0 (D) in patients with peptic ulcer was not different from that in controls. The number of nonexcretors of ABH substance among 684 patients with duodenal ulcer was significantly increased, but not in patients with gastric and combined ulcers.—L. D.
ABSTRACTS

Updated information and services can be found at:
http://www.bloodjournal.org/content/27/2/283.citation.full.html
Articles on similar topics can be found in the following Blood collections

Information about reproducing this article in parts or in its entirety may be found online at:
http://www.bloodjournal.org/site/misc/rights.xhtml#repub_requests

Information about ordering reprints may be found online at:
http://www.bloodjournal.org/site/misc/rights.xhtml#reprints

Information about subscriptions and ASH membership may be found online at:
http://www.bloodjournal.org/site/subscriptions/index.xhtml