ABSTRACTS

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ERYTHROCYTES


The authors present data of two different conditions of high red cell ATP level. A 37-year-old female patient with significant nonspherocytic hemolytic anemia (Hb 9 Gm. %, anisocytosis and poikilocytosis with irregular, contracted cells, reticulocytes 12-20 %) had 2160 nmoles ATP/ml red cells (normal controls 1280 ± 125 nmoles) and 697 nmoles ADP/ml red cells (normal controls 279 ± 59 nmoles). The activities of the enzymes of glycolysis, of the pentose-phosphate shunt and of glutathione reduction were normal. In the red cells of the patient incubated without glucose, ATP decreased with only 15 per cent the rate of normal controls. In a different family, the two parents and their three children had elevated red cell ATP while ADP was normal. There was no sign of clinical impairment in these persons; in fact the high ATP level was found by chance. While the latter condition appears similar to the cases described by Zürcher et al., the former case seems to represent a new disorder.—K.B.


Although organisms capable of synthesizing folate have been isolated from the intestinal tract of persons with intestinal blind-loop syndrome (Lancet 2:1339, 1966) and with tropical malabsorption (Amer. J. Clin. Nutr. 19:237, 1966), it remains uncertain as to whether folate synthesized by bacteria in this location is available for host nutrition. In the present study, mice fed a folate free diet developed deficiency of this vitamin despite the presence of an abundant folate-
synthesizing coliform population present within the proximal small intestine—F.A.K.


An infant is described who presented gastrointestinal bleeding, normocytic anemia, failure to gain weight, and who died at 7 weeks of age. He was found to accumulate abnormally large amounts of cystathione, homocystine, and homocysteine-cysteine mixed disulfide and to have low concentrations of methionine in the blood and urine. In addition, he had methylmalonic aciduria which was unresponsive to $B_{12}$ administration. The hepatic concentration of total $B_{12}$ was normal but that of coenzyme $B_{12}$ was abnormally low. The liver was also found to be deficient in the $B_{12}$-dependent enzyme $N_5$-methyltetrahydrofolate-homocysteine methyltransferase. The authors propose that deficiency of this enzyme resulted in inadequate metabolism of vitamin $B_{12}$ with resultant failure to accumulate enzymatically-active derivatives of this vitamin—F.A.K.


In over one half of cases, megaloblastic anemia of pregnancy manifests itself after delivery, in some instances after a period of several months or longer. This article reviews the evidence which indicates that the supply of folate to breast milk takes precedence over maternal needs. This situation not infrequently results in folate deficiency during the puerperium in populations where dietary folate is suboptimal and prolonged lactation the rule—F.A.K.


Megaloblastic anemia occurred in a 10-month-old entirely breast fed infant whose mother was a vegetarian. Serum $B_{12}$ level was low and serum folate was elevated. A partial hematologic response occurred with $B_{12}$ therapy and was complete after addition of iron therapy.—J.B.S.


Sixty-four pernicious anemia patients received 0.5 mg. $B_{12}$ orally daily for 2 months to 5 years. About 7 per cent of serum-$B_{12}$ values were under 150 pg./ml., but were normalized after increased dose of oral $B_{12}$. No neurological complications.—P.G.R.


A brief clinical description of two rare and apparently unrelated congenital disorders appearing in a young girl.—J.B.S.


Among infants whose birth weight is significantly below the expected for their gestational age, erythrocytosis is a common finding. Among 23 such infants the mean capillary hematocrit at 2 days was 67 per cent. The reticulocyte count in this group was below the usual level, and the fetal hemoglobin concentration was significantly higher than in “normal birth weight” infants of similar gestational age. Two male infants had symptomatic polycythemia, with respiratory distress, hypocalcemia tetany, hyperirritability, hypoglycemia and priapism.—J.B.S.

ABSTRACTS


Structural chromosomal aberrations (breaks, endomitoses) characteristic of Fanconi’s anemia were demonstrable in two cases. The red cell ATP level in both cases and hexokinase activity in the first one were normal. Thus, no correlation between chromosomal aberrations and disturbances of red cell metabolism could be found. In vitro study with tetrametansulfonyl-D-mannitol revealed increased fragility of chromosomal structures explained by the changed structure of DNA. The increased chromosome fragility is probably an important symptom in Fanconi’s anemia because these patients have increased susceptibility to leukemia and malignant tumors. No aberration was found in the two fathers (supposed to be heterozygotes). The serious pancytopenia could not be influenced by intravenous administration of phytohemagglutinin.—S.R.H.


Bone marrow histology was done in 52 cases of polycythemia vera. In 19 patients cellular hyperplasia existed; in these cases the spleen was usually only moderately enlarged and showed no signs of myeloid metaplasia. In 26 patients, the bone marrow showed cellular hyperplasia together with intensification of the reticulin network, but normal architecture. In these cases, notable splenomegaly was present and myeloid metaplasia was very probable. In seven patients, a fibrotic transformation of the bone marrow was observed, besides an increase of reticulin fibres. A true osteomyelosclerotic transformation, as in cases of primary myelosclerosis in the course of polycythemia vera is rare, however, in the experience of the authors.—M.C.V.


Capillary microscopic investigations were performed in 35 instances in 25 patients suffering from polycythemia vera. In the periods of exacerbation (19 investigations) vasodilatation, relative increase in the number of small blood vessels and a significant decrease of circulation time were observed. These anomalies partially regressed during remission (16 investigations).—S.R.H.


The therapeutic effect of Myelobromol in 23 patients suffering from polycythemia vera during a period of 5 years was investigated by 63 treatments. Hematological and clinical symptoms showed complete remission in each case. In relapse, complete remission was obtained by repeated treatment with Myelobromol. The average duration of remission after the first treatment was 14.7 months, and 6–9 months following repeated treatments. A dose of 250 mg. per day of Myelobromol, totaling an average dosage of 10,000 mg. in the first treatment and 8250 mg. in the following treatments, was necessary for obtaining remission. Hematologic studies revealed no considerable side effects.—S.R.H.


A 44-year-old white male with malignant thymoma developed ovalocytosis. Red cell agenesis and ovalocytes developed after thymectomy and were associated with recurrence of the malignant thymoma. There was no response to cortisone, chlorambucil or testosterone. It is postulated that the ovalocytosis was a manifestation of the disturbed erythropoiesis with malignant thymoma.—J.E.U.


The effect of MgCO₃ and Amberlite IRA-401 on the iron-transferrin and iron-conalbumin complexes has been studied, by the method of Bothwell. Amberlite eliminates the free iron. The iron binding capacity of serum was approximately the one theoretically calculated for both proteins. MgCO₃ removes the free iron and some of the fixed iron from the iron-transferrin and, especially, from the iron-conalbumin complex.—E.S.

PANCYTOPENIA WITH HYPERCELLULAR HEMATOPOIETIC TISSUE. M. Schiller, E. A. Rachmilewitz and G. Izak. Hematology Research Laboratory and the Department of Medicine B, Hadassah University Hospital and Hebrew University—Hadassah Medical School, Jerusalem, Israel. Israel J. Med. Sci. 5:69, 1969.

The clinical and laboratory features of 28 patients with chronic pancytopenia and normo- or hyperplastic bone marrow are described. The marked similarity of the hematological findings presented here seems to justify the description of these patients as a group, although the etiological factors responsible for this picture were probably manifold. The relationship between this disease and that referred to as primary hypoplastic anemia, refractory anemia, and by a variety of other terms has been discussed.—B.R.

LEUKOCYTES

Blood and bone marrow changes were observed in over 1000 animals, including guinea pigs, rats, hamsters and cats, following treatment by cyclofosfan, imitifs and vincristine (cytotoxic drugs). Initially there occurred necrosis of the myeloid cells followed by accelerated maturation. Reticulum cells seemed to be resistant to the drugs and later differentiated into giant neutrophils. From the third or fourth days large monocytoid blastic cells were observed, these apparently having developed from the reticulum cells; erythroid elements also developed from the reticulum cells. During the third and fourth weeks a more normal pattern of marrow regeneration supervened.—J.V.

VIRAL INDUCED ACUTE LYMPHATIC LEUKEMIA IN GUINEA PIGS RESEMBLING HUMAN LEUKEMIA. S. R. Opler. From Department of Pathology, Stanford University School of Medicine, Stanford, California. Haematologia 3:157–162, 1969.

An acute lymphatic leukemia of guinea pigs which arose spontaneously was found to have a morphologically distinctive virus as seen under the electron microscope in ultrathin sections of lymphoid tissues, megakaryocytes and plasma pellets. The natural host for the Opler leukemia virus is an inbred line of guinea pigs known as Strain 2. This virus has not been observed in non-leukemic animals. Transmission of the leukemia has been accomplished with cell-free techniques, placing this animal model which morphologically resembles human leukemia, as an additional example of viral-induced leukemia. This system warrants further investigations in the broad field of experimental hematology.—S.R.H.


Nine adult patients with acute leukemia (7, lymphoblastic; 1, myeloblastic; 1, erythromyeloblastic) on treatment with various therapeutic agents including prednisolone, 6-mercaptopurine, methotrexate and vincristine) were actively immunized by the transfusion of 200–300 ml. plasma and leukocytes from leukemic donors, this transfusion being repeated after 12–14 days. Clinical and hematologic remissions were noted in five cases. Compared with leukemic patients on drugs alone, these immunized patients
showed a higher incidence of remissions and an extended life span.—J.V.


The ability of peripheral blood lymphocytes to respond to phytohemagglutinin (PHA) in vitro was studied in patients with Down’s syndrome. The response was measured by the increase in DNA polymerase activity and the rate of incorporation of tritiated thymidine by the cultured lymphocytes. These activities were significantly lower in PHA-stimulated lymphocytes from patients with Down’s syndrome, compared with age- and sex-matched, mentally retarded patients without Down’s syndrome from the same institution and the normal healthy volunteers. The impairment in response to PHA does not seem to be related to the presence of Australia antigen in patients with Down’s syndrome or to institutionalization itself. In contrast to DNA polymerase activity and thymidine-3H uptake, there was no significant difference in the percentage of blast transformation in the three groups studied. The poor response of the lymphocytes from patients with Down’s syndrome to a mitogenic stimulus could reflect an impairment of cellular immune functions in these patients which may be one of the factors contributing to the vulnerability of these patients to repeated or persistent infections.—J.E.U.


Chromosomal studies were performed on 61 adult patients with “typical chronic myelocytic leukemia.” The Philadelphia (Ph1) chromosome was found in 43 patients, with equal sex distribution and with a median age of 48 years. The 18 Ph1-negative (Ph1−) patients had a median age of 69 years and were predominantly male. Absent leukocyte alkaline phosphatase was noted with equal frequency in both groups. The Ph1− patients had lower white blood cell and platelet counts and did not respond as well to antileukemic therapy. Basophilia and abnormally high platelet counts were fairly common among Ph1 positive (Ph1+) patients. The incidence of blastic crisis was approximately 50 per cent in both groups but appeared earlier in the Ph1− group, only 8 months. Karyotypic analysis furnishes a subclassification of myelocytic leukemia with significant prognostic and therapeutic implications.—J.E.U.


Destructive bone lesions were observed in six patients from a series of 205 patients with chronic granulocytic leukemia (CGL). In three patients, bone involvement occurred during the blastic phase of the illness, and in two of these cases the bone lesion was the initial manifestation of blastic transformation. Two other patients with osseous lesions remained in the chronic phase of CGL. The osteolytic lesions were rapidly progressive and tended to occur at the ends of the long bones. Numerous Gaucher-like histiocytes were observed in the lytic areas and bone marrow of two patients, probably the result of rapid granulocyte turnover and membrane lipid accumulation in these cells. In this series, local radiotherapy produced effective palliation and halted progression of the lytic process in three patients.—J.E.U.


Activities of β-glucuronidase and of β-acetylglycosaminidase were compared cytochemically in plasma cells of normal persons or of patients with reactive plasmacytosis and in plasma cells of patients with multiple myeloma. Multiple myeloma plasma
cells displayed higher activities. This is in accordance with the elevated activities of acid phosphatase and of unspecific esterase in such plasma cells. On the other hand, the activity of adenosine triphosphatase was found to be diminished.—K.B.


Minor congenital abnormalities and the dermatoglyphics of 25 leukemic children were studied. The leukemic patients presented different patterns than the normal children and also a higher incidence of congenital cutaneous spots was registered. Cutaneous spots were found in six of the 14 cases studied and were present from the time of birth, according to the mothers’ information. The other abnormalities occurred with a lower incidence. A patient presented eight of the abnormalities studied. Theoretical considerations are discussed concerning the value of these findings, in that inherited genetic characteristics are permanently present and differ between normal and leukemic individuals. It is pointed out that these characteristics might be useful in distinguishing people with a propensity for leukemia.—M.A.J.

THE ACTION OF INTERFERON IN TRANSPLANTABLE LYMPHATIC GROSS LEUKEMIA. A. Stelmachowska, Z. Błach-Olszewska and Z. Skurska. From Department of Immunology of Tumors and Department of Virology, Institute of Immunology and Experimental Therapy, Polish Academy of Sciences, Wroclaw, Poland. Arch. Immun. Ther. Exp. 18: 16-22, 1970r.

Subcutaneous administration of $8 \times 10^7$ leukemic cells of Gross virus lymphoma into mice of inbred AKR strain caused rapid development of the neoplastic process and death of 100 per cent of animals in 2-3 weeks. Treatment with interferon obtained from mice inoculated with Newcastle virus prolonged the survival of mice by 5-6 days. Interferon was administered together with leukemic cells and, then, twice daily in doses of 512–1024 PDD 50 during 14 days. The results indicated that interferon did not protect the animals from generalization of the neoplastic process, but prevented the development of tumors at the site of its inoculation.—M.K.

THE EFFECT OF CYCLOPHOSPHAMIDE ON BLOOD CELLS. S. Sadliński, J. Stadnik, D. Włodarczyk and H. Biskupek. From 3rd Department of Surgery, Silesian School of Medicine and from Department of Internal Medicine, Municipal Hospital, Bytom, Poland. Pol. Tyg. Lek. 24:1811-1813, 1969.

Influence of cyclophosphamide on peripheral blood was examined in patients with neoplasms and in rabbits. In patients 200 mg., in rabbits 3 mg/Kg. of cyclophosphamide were injected intravenously every day during 20 days. Blood examinations were performed before and after 1, 4, 7, 10, 14 and 20 days of cyclophosphamide treatment. The changes observed in patients and rabbits were very similar. Slight drop in leucocyte count mostly pronounced on the tenth day was due to lowering of absolute counts of lymphocytes. Absolute numbers of granulocytes rose in this period. Moderate increase of eosinophilic granulocytes was observed. Qualitative changes consisted in the presence of toxic granules, vacuolization of plasma and nuclei, and nuclear swelling. Moderate lowering of the platelet count took place in patients as well as in rabbits reaching, on the 20th day, 70 per cent and 80 per cent of the initial values, respectively.—M.K.


Classical Gaucher’s cells, identified by their histochemical and ultrastructural characteristics, were found at autopsy examination of the liver and bone marrow of a child who died of acute leukemia.—J.B.S.

OSTEOPOROTIC FRACTURES SECONDARY TO METHOTREXATE THERAPY OF ACUTE LEUKEMIA IN REMISSION. A. H. Ragab, R. S. Frech, and T. J. Vietti. Washington Uni-
ABSTRACTS

ROENTGENOGRAPHIC CHANGES IN CHILDHOOD

Four of 11 children with acute lymphoblastic leukemia on long-term methotrexate therapy developed severe bone pain in their distal extremities. All were in good clinical and hematologic remission. Radiologic examination revealed severe osteoporosis with associated fractures in five of these 11 children. A trial of local radiotherapy was attempted in one patient with no response. Methotrexate therapy was stopped in four patients with marked improvement in their bone pain and osteoporotic lesions.—J.E.U.


The results of total body irradiation for 17 consecutive patients with symptomatic chronic lymphocytic leukemia who had received no prior therapy are reviewed. All patients were given an induction course of treatment and subsequently observed without maintenance therapy to determine the duration of remission. Using a described classification to measure therapeutic response, 16 of the 17 patients were evaluated to have had favorable clinical responses. Eight of these responders had complete disappearance of palpable disease and symptoms, correction of anemia when present, and restoration of the white blood count and differential count to normal following completion of therapy (Type I response). The remaining eight responders had less complete but definite improvement both objectively and subjectively (Type II response). The duration of unmaintained remission was measured from completion of induction therapy to relapse, the latter being defined as any evidence of recurring disease, even though asymptomatic. The median duration of remission was significantly longer for the Type I (19 months) as compared to the Type II (4 months) responders. The ability to achieve these high quality remissions offers some prospect of materially improving both the quality of life and survival time of chronic lymphocytic leukemia patients in the future.—J.E.U.


In a study of 50 patients with cirrhosis, analyses of serum proteins were made and, in 47, sternal marrow was also examined. There was an increase in reticulum cells and plasma cells in the marrows, primarily due to large numbers of young cells, particularly proplasmacytes. Serum protein paper electrophoresis demonstrated the hypoalbuminemia and hyperglobulinemia of this disease, the latter being clearly related to the increase in marrow plasma cells. Immunoelectrophoresis of the sera showed subtle changes among which was a consistent elevation of all three immunoglobulins (M, A and G). The authors regard the cellular reaction and immunoprotein changes as the result of an autoimmune reaction in cirrhotic patients with hyperplasia of the reticulum cells, differentiation of which into plasma cells leads to an increase in immunoglobulin production.—J.V.

STEM CELL LEUKAEMIA IN MYELOMATOSIS. E. Andersen and A. Videbaek. From the Medical Department C (Chief, Aa. Videbaek), Gentofte Hospital, Copenhagen/ Hellerup, Denmark. Scand. J. Haemat. 3:201-207, 1970.

In four of 19 nonselected cases of myelo-
matosis, acute myeloblastic leukemia developed at the end. These four cases showed severe myelomatosis of relatively long duration; all four had been treated with cytoxic agents. X-ray treatment was given in two of the four cases. The development of acute leukemia in systemic blood diseases in general is discussed. Abstractor's comment: In the last few years several articles have been published dealing with myeloblastic leukemia developing in patients with lymphogenic leukemia and vice versa. A close evolutionary connection exists between the stem cells of different blood cell lines, a reason why perhaps, in the long run, one form of systemic blood disease may become "transformed" into another.—M.C.V.


A 52-year-old female patient fell rapidly ill with cerebral symptoms, pronounced anemia (5.5 Gm. %) and thrombocytopenia. In the spinal fluid, 1730 cells per jsl. were found, mostly atypical mononuclear elements. The bone marrow showed absence of normal homopoietic cells and the presence of a uniform population of reticular cells, negative in the peroxidase reaction, in the chloracetate-esterase-reaction and in the a-naphthylacetate-esterase-reaction. Cytostatic therapy with cyclophosphamide was without effect. Autopsy showed malignant reticulosis with massive involvement of the mesentery as well as of the meninges, spinal cord and optical nerves.—K.B.

HEMOSTASIS

The authors present further evidence that the platelet release reaction occurs by the secretion of specific substances from subcellular organelles rather than by simple cell lysis. They found that five acid hydrolases associated with α-granules as well as serotonin and metabolically inert adenine nucleotides were released from platelets by thrombin. By contrast, none of nine enzymes associated with the soluble fraction, membranes or mitochondria were released. The authors concluded that the substances released by thrombin originate from α-granules and very dense bodies, whereas constituents of other subcellular compartments appear to be retained by the cell.—H.J.W.


14C-adenosine is not incorporated in guinea-pig platelets which are insensitive to adenosine with regard to the inhibition of ADP-induced aggregation. Rat platelets are also insensitive to adenosine, but 14C-adenosine is incorporated by these platelets. In these two animals, plasmatic adenosine deaminase is different from the ones from human, rabbit and dog and the author's postulate that both plasmatic adenosine deaminase and adenosine incorporation in platelets are of great importance for platelet behaviour in primary hemostasis.—J.C.


The hemolytic activity of Naja nigricollis venom seems to be enzymatic and is, at least, partially due to lecithinase activity. In human serum, there exists a thermolabile antilecithin the identity of which is discussed by the authors with regard to the antihemolytic activity of the serum.—J.C.

The regulation of platelet production and maturation was studied by labeling the megakaryocyte cytoplasm with $^{35}$S and following its subsequent appearance in the circulating platelets. By using a bioassay system that employed platelet-hypertransfused rats, the author demonstrated the presence of a thrombopoietic-stimulating activity in normal rat plasma. This activity was increased in thrombocytopenic rats and was undetectable after platelet hypertransfusion. The author concludes that this circulating factor regulates platelet production and that the amount of available stimulator is governed by the requirements for circulating platelets.—H.J.W.


"Old" and "young" rat platelet populations, each prepared by two different methods, were labeled with $^{51}$Cr, and their survival patterns were observed in normal recipients. Old platelets were removed much more rapidly than normal in each instance while "young" platelets were removed at a rate much less than normal during the first 2 days after injection. These studies indicate that "senescence" is an important and probably major factor governing the removal of platelets from the circulation. Discrepancies between the results of these studies and those of some previous studies with other isotopes may be due to differential loss from the platelets of the sites at which various labels attach.—H.J.W.


The authors studied 26 children during the active phase of the hemolytic uremic syndrome. Thrombocytopenia was a constant finding. A variety of changes in other clotting factors suggested to the authors that intravascular coagulation had occurred. They concluded that thrombosis was superimposed upon a more basic vascular lesion. The authors suggested that heparin might be useful, but do not report on its use in their study. Abstractor’s comment: Although this disorder in children resembles thrombotic thrombocytopenic purpura in some respects, there is no convincing evidence that the latter disorder is associated with intravascular coagulation.—H.J.W.

IMMUNOHEMATOLOGY


The immune globulins of a patient having primary "acquired hypogammaglobulinemia" and his family were studied. Reduced serum concentrations of one or more factors were found in most family members. Individual globulin synthesis is regulated by autosomal nonallelic genes. The recessive allele which manifests variable expressivity causes marked reduction of globulin synthesis in the homozygous state. "Acquired hypogammaglobulinemia" occurs when synthesis of all three globulins is inhibited in the same individual. A heterozygous carrier frequency of 1:5.6 for each globulin may be necessary to maintain "acquired hypogammaglobulinemia" in the general population. In certain healthy family members, the similarity of the immune globulin pattern to those found in patients having dysgammaglobulinemia suggests that synthesis of other infection resistance factors besides the immune globulins may be impaired in the dysgammaglobulinemias.—J.E.U.


An evaluation of the immune status in patients with Burkitt’s lymphoma was performed. Cellular immune responses were normal in both untreated patients and in patients in remission. An impairment of primary antibody response and low IgM levels were noted in untreated patients with Burkitt’s lymphoma but not in patients in remission. A defect in humoral immunity as-
sociated with the presence of active Burkitt's lymphoma is postulated; this defect is possibly related to IgM metabolism.—J.E.U.


Lymphopenic agammaglobulinemia is described in a brother and sister, each of whom died of respiratory infection before the age of 9 months. Laboratory and pathologic studies were typical of the "Swiss type" of congenital immunoglobulin deficiency. This autosomal recessive form of agammaglobulinemia has been reported infrequently in the United States.—J.B.S.


An 11-year-old boy with Bruton type of congenital agammaglobulinemia developed and, 7 months later, died of Hodgkin's disease.—J.B.S.

MISCELLANEOUS


Basal cells of hairless mouse interfollicular epidermis are self-perpetuating to 85 per cent, with generation times about 3.5 days. After methylcholantren, the "proliferation rate" was three times normal. Homogenized epidermis contains the glycoprotein chalone (molecular weight 30-40,000), capable of reducing the "mitotic rate" by 50 per cent. Indications for a "granulocyte chalone" exist. Action is assumed to be on DNA synthesis in G1.—P.G.R.


In 22 patients with cirrhosis and anemia transfusions of fresh blood (up to 48 hours old) led to an improvement in the general clinical condition, arrest or reduction of the anemia and hemorrhagic manifestations, and improvement in liver function. After the course of transfusions, electrophoretic and immunoelectrophoretic studies of the sera demonstrated a reduction of the dysproteinemia with restoration of albumin levels and correction of abnormalities in the other fractions. After repeated transfusions blood amino acid levels tended to regain normal values.—J.V.


Adenosine deaminase was determined in heparinized platelet-rich plasma by using a radiochromatographic method which can measure the degradation of 0.016 μmoles adenosine in inosine. This enzymatic activity is suppressed by heating at 56°C for 30 minutes and by high concentrations of urea, but it is not affected by dipyridamol, an inhibitor of adenosine incorporation by platelets. Abstractor's comment: This technique can be useful in the detection of plasmatic abnormalities responsible for abnormal platelet behavior, for instance, in chronic myeloid leukemia.—J.C.