ABSTRACTS

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ERYTHROCYTES

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ERYTHROCYTES


A fascinating and carefully studied case of refractory, microcytic, hypochromic anemia with increased iron stores, fever, splenomegaly and bone-marrow plasmocytosis in an 8-year-old boy who had, in addition, mediastinal lymphadenopathy which seemed to be responsible for his anemia, since all signs and symptoms disappeared after surgical removal of the nodes. The authors speculated that clones of antibody-producing cells were lodged in mediastinal lymph nodes, presumably by fetal inoculation with lymphocytes from the mother. Attempts to demonstrate reactivity between maternal tissue and patient’s cells and serum were unsuccessful. Chromosome studies and skin transplantation were not performed.-A. J. E.


In 12 patients with nephrosclerosis and chronic uremia, a severe hypochromic, normocytic anemia with erythoblastic hyperplasia, slow maturation, hypersideremia and bone marrow hypoplasia, absence of reticulocytosis and signs of increased hemolysis, increased erythrocyte free protoporphyrin (in 9 cases), normal excretion of coproporphyrin, and minimal modification of urinary excretion of other porphyrin precursors were observed. A reduced utilization of iron was postulated and the diagnosis of sideroachrestic anemia was suggested.—P. d. N.


Significant decreases in skeletal muscle myoglobin, cytochrome c concentration and oxygen uptake, using succinate as the substrate, were demonstrated in rats with severe nutritional iron deficiency. Low cytochrome c levels in diaphragm and kidney were also noted. Brain and heart cytochrome c were minimally reduced, as was the myoglobin content of heart and diaphragm.—J. B. S.

ERYTHROPOIETIN AND MODIFICATIONS OF HEMOGLOBIN SYNTHESIS AND OF ERYTHROCYTE

Fractions were prepared from 8 cases of polycythemia, from a subject with secondary polycythemia (silicosis) and from a normal subject, according to Liman and Bethell with some modifications. An increase in erithrocytes and reticulocytes and of Fe59 incorporation as compared with controls, a decrease in mean diameter, mean corpuscular volume and mean corpuscular hemoglobin content in newly formed erythrocytes, and an increase in mean corpuscular thickness and sphericity index according to Cazal were found after injection of fractions from polycythemic patients, but not with fractions from the other subjects.—P. d. N.


Acid hemolysis was studied by using trypsin, papain and bromelaine treated erythrocytes. Analogies between acid hemolysis of artificially altered erythrocytes and hemolysis of erythrocytes in paroxysmal nocturnal hemoglobinuria were demonstrated.—P. d. N.


Serum citric acid levels were significantly lower in thalassemic children than in their healthy peers, as was the 24-hour urinary excretion. Low normal levels of serum calcium, phosphorous and alkaline phosphatase were noted. The authors concluded that the osteoporosis of thalassemia is due not to bone marrow hyperplasia, but to an abnormality in the metabolism of ossification.—J. B. S.


A Thai family is described in which the father carries the trait for a new hemoglobin (hemoglobin Siriraj) and the mother has β-thalassemia minor. One of their children has hemoglobin Siriraj-β thalassemia. The red cells containing the abnormal hemoglobin do not sickle. It is a β-chain variant in which lysine has replaced a glutamic acid residue at position 7.—P. B.


Infections in patients with sickle-cell disease appear to predispose to sickling crises. Protection against infection might, therefore, confer clinical benefit. Because the commonest early infections in Uganda, where the study was carried out, are malaria and respiratory infections, the prophylactic value of chloroquine and a long-acting penicillin was assessed. Patients under 6 years old, followed for 10 months or more and who were treated, had a significantly lower rate of dactylitis and higher average hemoglobin values than patients in a control placebo group. The authors recommend that children with sickle-cell anemia in malarious zones should be treated with chemoprophylactic doses of chloroquine.—P. B.


Patients with sickle cell hemoglobinopathies demonstrated a normal pattern and extent of immunoglobulin response to Salmonella vaccines. These patients also had normal isohemagglutinin and antitoxin antibody titers.—J. B. S.


An extract, enriched in catalase, was obtained by gel filtration on Sephadex G-100 of normal and acatalasic hemolysate. Catalase, with a molecular weight of about 220,000 appeared in a peak separate from hemoglobin. Catalase was concentrated by dialysis against polyethylene glycol.
to yield a final product enriched 15-fold in catalase, as measured by enzymatic activity. Immunochemical detection of catalase was done by double diffusion and was compared with the pattern given by a normal hemolysate. By comparing the precipitin lines, it was found that the acatalasic hemolysate had about 75 to 150 times less catalase than the normal. The catalase of the acatalasic subject appeared to be immunochemically normal.—G. M.


Two cases were presented in which the hemolytic process was mild. The degree of enzyme deficiency was not very extreme and the results of autohemolysis studies were those of Type I (Selwyn and Dacie). Red cell ATP content was decreased and 2,3-diphosphoglycerate content was markedly increased. The rates of red cell glucose utilization and lactate formation were decreased in both. These patients and the 47 hitherto reported cases were reviewed and the clinical picture was summarized.—K. F.


Sixteen cases were observed in 240 instances of severe jaundice in newborn males (approximately 6.67 per cent). In 6 cases, maternal isoimmunization was ruled out.—P. d. N.


The intra- and extracellular hydrogen ion concentrations of umbilical cord blood were 82.8 ± 7.3, and 52.9 ± 5.9 nanoequivalents per liter with a difference of 30.0 ± 6.3 nanoequiv. per liter. This difference was identical to that previously reported for red cells from adults. Maternal and fetal bloods, exposed to varying pCO₂ tensions, had similar intracellular hydrogen ion concentrations.—J. B. S.


Respiratory distress with consequent respiratory acidosis again was shown to be an important factor in producing neonatal hyperbilirubinemia, particularly in small premature infants with elevated pCO₂ levels were described in whom kernicterus appeared, followed by death within 12 hours of the onset of symptoms. In four infants, the peak bilirubin was below 19 mg. per cent.—J. B. S.

**Hemostasis**


Careful examination of the pulmonary arteries in a series of 61 consecutive adult autopsies reasonably representative of the total autopsy population at the hospital revealed the presence of old or recent thromboemboli in 64 per cent. This figure was considerably higher than the incidence usually recorded and its magnitude was in large part due to the inclusion of organized and organizing traces usually overlooked, unless extreme care is taken and the index of suspicion is high. During a study in dogs of the fate of experimentally induced autologous peripheral venous thrombi released to the pulmonary circulation, the authors noted that some of the residue observed weeks or months after the embolic episode was identifiable only as traces of organized thrombi. The inclusion of such lesions was responsible for the high percentage of thromboemboli found. Previously reported experimental observations demonstrating the effectiveness of thrombolysis in disposing of emboli in the lung suggested that the reported figure may represent a minimum value. Many of these episodes appeared to be multiple and of little clinical significance, attesting to the efficiency of the disposal mechanisms, but even more severe episodes frequently were unrecognized during life.—R. G.

Studies of 117 patients on long-term sodium heparin therapy disclosed cumulative evidence of a relationship between prolonged use of large amounts of heparin and osteoporosis. No symptoms of osteoporosis developed in 107 patients who received 10,000 units of concentrated sodium heparin once daily or less for 1 to 15 years. Of 10 patients treated with larger doses (15,000 to 30,000 units) for 6 months or longer, spontaneous fractures of vertebrae or ribs developed in six; osteoporosis was present, and biopsies disclosed soft bony matrix. Parathyroid function studies in one patient were normal. Serum calcium, phosphorus and alkaline and acid phosphatase values remained within normal limits on a low calcium, low phosphorus diet. Urinary excretion of calcium remained within normal limits on a low calcium, low phosphorus diet. Urinary excretion of calcium tended to be low, while total hydroxyproline excretion was normal. Studies of rats on sufficient heparin therapy to triple clotting times for 10 days indicated that collagenolytic activity of rat bone-cell homogenate increased two to four fold and the stability of the lysosome-like bodies in cells which contain collagenase appeared to be decreased. The authors postulated that hyperheparin states, whether spontaneous or man-induced, may take their place in the category of lysosomal disease.—R. G.


An active 41-year-old man with angina pectoris developed slight generalized osteoporosis and multiple spontaneous fractures (left and right 7th and 8th ribs and compression fractures of the 6th, 8th, and 10th vertebral bodies) while receiving daily subcutaneous injections of 20,000 units of sodium heparin over a 13-month period. Following cessation of heparin therapy, no new fracture developed in 6 months and he experienced no new episodes of pain. Good callus formation occurred.—R. G.


Two cases were reported. In one patient, the prothrombin time was markedly prolonged, but in the other it was in the so-called therapeutic range. The onset of circulatory collapse in a formerly stable patient treated with anticoagulants should suggest the diagnosis, despite the absence of the classical clinical signs of pericardial tamponade. Pericardiocentesis is the most reliable diagnostic procedure and it usually affords adequate therapy as well.—R. G.

**ABSTRACTS**


In 84 cases of liver cirrhosis, prolongation of k-values and reduction in the maximal amplitude represented the most significant and frequent findings. The author considered these modifications to be the consequence of alterations in the thrombin-fibrinogen reaction.—P. d. N.


Activated Hageman factor, when injected into the rabbit ear chamber, produces a delayed and prolonged inflammatory response, characterized by prominent sticking and emigration of leukocytes. In contrast, preformed bradykinin evokes an immediate and more transient response in which leukocyte emigration occurs less frequently. It is concluded that either Hageman factor produces its inflammatory effects by mechanisms other than kinin release or bradykinin released endogenously has effects quite different from those resulting from a single injection of the exogenous material. —R. G.


Case report of a 31-year-old woman who, one month after the second pregnancy, developed severe manifestations and who had a complete, spontaneous remission after 2 years. No therapy was effective during the acute phase. The inhibiting effect could be demonstrated in a dilution of 1:100 with specific inhibition of antihemophilic globulin and, possibly, of some factors of the
prothrombin complex. The anticoagulant was not adsorbed by barium sulfate, was present in serum, was not neutralized by protamine sulfate or toluidine blue, was thermostable and could be kept unaltered at low temperatures.—P. d. N.


Incubation of citrated normal human plasma with 1–2 \( \mu \text{g} \)/ml. of Malayan pit-viper venom resulted in apparently complete conversion of plasma fibrinogen to fibrin without loss of Factor VIII activity. This observation offers a method for the preparation of fibrinogen-free Factor VIII preparations.—P. B.


A controlled clinical trial with orally administered EACA was carried out on 10 severe haemophiliacs. There was appreciable reduction in spontaneous bleeding episodes during EACA therapy. None of the patients showed a rise in Factor VIII, nor were any significant changes observed in plasminogen and fibrinogen levels. The results were considered to warrant further large-scale clinical study of EACA in hemophilia.—P. B.


By the use of EACA titration, SK-activated plasminogen in the plasma and euglobulin fraction was measured. Plasminogen activity was expressed as per cent EACA versus activated plasmin. Antiplasmin was calculated from the difference between plasmin activities in the plasma and the euglobulin fraction. Half the patients with acute hepatitis were hyperplasminemic. Cases with chronic hepatitis and liver cirrhosis had decreased EACA titration values for plasma plasmin activity, in contrast with elevated plasmin values in the euglobulin fraction. This finding reflected the increase of antiplasmin in these diseases. In some cases with nephritis, there was hypoantiplasminemia. In cases with arteriosclerosis, there was no hyperplasminemia, except with cerebral hemorrhage.—K. F.


Patients with systemic poisoning from Malayan pit-viper bites develop incoagulable blood which is associated with increased fibrinolytic activity. Treatment with specific antivenom promptly abolishes both the coagulation defect and the increased lytic activity. EACA treatment depresses fibrinolysis, but does not rectify the coagulation defect. It was, therefore, concluded that increased fibrinolytic activity is not the predominant cause of the "defibrination" syndrome following this type of snake bite. No harmful effects follow EACA therapy. Malayan pit-viper venom has a highly specific direct action on fibrinogen which prevents its conversion to fibrin. The other clotting factors and platelets are unaffected by venom and this distinguishes snake-bite "defibrination" from other defibrinating syndromes. Despite incoagulable blood, hemorrhage is conspicuously absent in many patients with the snake bite syndrome and the condition resembles congenital afibrinogenemia. The author suggests that the fibrinogen-fibrin reaction by itself may play little part in the normal control of spontaneous or minor hemorrhage.—P. B.


Two observations of acute monoblastic leukemia were reported. The first was accompanied by a hemorrhagic syndrome and extreme hypofibrinogenemia, symptoms which appeared when the number of monoblasts in the blood exceeded 100,000 per cu. mm. The syndrome became more severe with an increase in the number of cells and even more acute when the cells were rapidly removed by therapy. Epsilon-amino-caproic acid did not arrest the fall in fibrinogen, even when 1 Gm./Kg. was given. Hypofibrinogenemia was cor-
rected by injection of fibrinogen (fraction I.A., National Blood Transfusion Center) and by the Kunitz inhibitor. The second patient did not have fibrinogen deficiency, but hemorrhagic symptoms appeared each time there was a brisk fall in the number of circulating monoblasts without relation to the platelet count. These observations suggested that there was a relationship between the hemorrhagic syndrome, the monoblast count and the rapidity of destruction of these cells. Leukocytes were concentrated by sedimentation, either with or without dextran. The cells were washed and tested in fibrin plaques, either warmed or not warmed. Isolated monoblasts formed 85-90 per cent of the leukocytes and were tested intact, after treatment with saponin or after rupture by freezing and thawing three times. An activity resembling that of a plasminogen activator was demonstrated. Ruptured monoblasts had a direct fibrinolytic activity. These properties resembled those of granulocytes and promyelocytes. It was not possible to demonstrate such activity in lymphoblasts obtained from a patient with acute lymphoblastic leukemia. Lymphoblasts differ from monoblasts in their almost complete absence of cytoplasmic granules which are considered to contain large quantities of hydrolytic enzymes. A study is now in progress to examine the role of the fibrinolytic activity which has been observed and to determine its relationship to the normal fibrinolytic system in the plasma.—G. M.

**CONGENITAL DEFICIENCY OF FIBRIN-STABILISING FACTOR. A REPORT OF THREE UNRELATED CASES.** M. S. Losowsky, R. Hall and W. Goldie.


The authors report the first cases in Great Britain of congenital deficiency of fibrin-stabilizing factor (FSF). In each case, recalcified plaques were soluble in 5 M urea. The half-life of FSF appeared to be about 7 days. The clinically normal parents of two of the patients showed minor degrees of FSF deficiency.—P. B.

**VON WILLEBRAND'S DISEASE. USE OF A PLATELET-ADHESIVENESS TEST IN DIAGNOSIS AND FAMILY INVESTIGATION.** H. S. Strauss and G. E. Bloom.


Von Willebrand's disease has been characterized by a prolonged bleeding time, reduced plasma level of Factor VIII, reduced in vivo platelet adhesiveness and an autosomal mode of inheritance with variable expressivity. An in vitro test for platelet adhesiveness (modification of the Salzman technic) was performed on 14 affected propositi and 219 members of their families. Bleeding times and Factor VIII levels were also determined. In 6 families, occurrence of the disease was familial and in 8 it was sporadic. Fifty-one had reduced Factor VIII levels. Bleeding phenomena depended largely on the degree of Factor VIII deficiency. Although the bleeding time was often normal and Factor VIII levels fluctuated and were occasionally normal, platelet adhesiveness was reduced consistently in the affected patients. Forty-four individuals with normal Factor VIII levels exhibited reduced platelet adhesiveness as the only abnormality. These individuals had no bleeding tendency and were considered "carriers." Patients with hemophilia A and B or with congenital deficiencies of Factors V–VIII, VII, XI, and fibrinogen had normal platelet adhesiveness. Severe anemia and renal disease have been found to be associated with reduced platelet adhesiveness. Reduction of platelet adhesiveness was diagnostic of Von Willebrand's disease only when associated with reduced plasma levels of Factor VIII, with or without prolonged bleeding times. The test reliably differentiates Von Willebrand's disease from classic hemophilia in cases of Factor VIII deficiency associated with normal bleeding times, and in which the family history does not establish the pattern of inheritance.—R. G.

**STUDIES ON THROMBOPOIESIS. II. ASSAY OF HUMAN PLASMA THROMBOPOETIC ACTIVITY.** I. Schulman, C. F. Abildgaard, J.-A. Cornet, J. V. Simone, and Z. Currimbhoy.


Significant thrombopoietic effects on rats given daily intraperitoneal injections of filtrates prepared from acidified, boiled fresh human plasma were demonstrated. Both the dose and the number of injections influenced the eventual platelet response which was maximal 3 days after the last of 3 or 5 doses and which persisted for 6 days. The platelet response was significantly greater in splenectomized rats. Filtrates prepared from plasma of a child with congenital thrombopemia were ineffective in this test system, while filtrates from children with acute or chronic ITP were at least as effective as those prepared from healthy donors. Fresh normal human plasma in a dose of 30 ml/Kg induced a permanent remission in 7 of 13 children with acute ITP. The administration
of fresh plasma, either by vein or by mouth, induced rises in platelet count to normal levels in the child with congenital thrombopenia; this response has remained consistent over a 10-year period. Although heat stable, human thrombopoietin was storage labile, particularly at or above 4 C. Lyophilization may preserve activity. Human thrombopoietin appears to be an acidic glycoprotein similar to, but probably not identical with, erythropoietin.—P. B.

LEUKOCYTES


Older children and adolescents seemed to fare better when informed that they had leukemia as soon as the diagnosis was confirmed. Secretive behavior on the part of parents and physicians increased, rather than allayed, anxiety. The authors pointed out that the leukemic patient eventually learns of the diagnosis and is left without adequate psychologic support when everyone in his environment denies what he knows or suspects to be true.—J. B. S.


The author investigated regional differences in leukemic infiltration of pulmonary tissue, i.e., pulmonary pleura, bronchial adventitia and pulmonary lymphoid tissue, to determine the infiltrative pathway of leukemic cells. The material consisted of 34 autopsies of various leukemias and related diseases. (1) In the mediastinal area of the pulmonary pleura, the density of infiltration decreased gradually from the hilum and pulmonary ligament toward the periphery. (2) The marginal regions of the overhanging pleura at the sites of right S₃, middle lobe and lower lobes were likely to show dense leukemic infiltration. (3) Regional differences between central bronchial adventitia (3rd to 5th bronchus) and peripheral ones (6th to 8th bronchus) were statistically insignificant. (4) Leukemic infiltration of the bronchial lymph nodes started at the perivascular areas and leukemic cells invaded them from their peripheral zones toward the centers. (5) A statistically significant correlation in the degree of leukemic infiltration of the hilar lymph nodes and bronchial lymph nodes was shown by $\chi^2$-test only in CML and reticulosaoma.—K. F.

Following intravenous injection of methotrexate into leukemic children in doses of 3 mg./Kg., urinary FIGLU excretion increased rapidly, usually to levels of over 50 mg. per 8 hours. Maximal levels were usually seen on the third day and, by the 10th day after injection, urinary FIGLU returned to the pretreatment level. In a patient who did not achieve significant remission, no rise in FIGLU excretion was observed. In a few patients on oral methotrexate, urinary FIGLU levels did not exceed 20 mg. per 8 hours.-J. B. S.


Intraperitoneal administration of VLB to pregnant Wistar rats from the 7th to the 12th day of pregnancy in a dose of 0.25 mg./Kg. produced a 40 per cent mortality and a 9 per cent incidence of gross malformation among the surviving offspring. These effects were not reversed by the simultaneous administration of glutamic acid. Four hours after administration of 1 mg. i.V. to the mother there was a 6-fold increase in the number of mitotic figures in all preparations derived from the fetus.-J. B. S.


Increased fetal resorption, intrauterine death, runtting and skeletal anomalies were seen in the offspring of Swiss-Webster mice given intraperitoneal injections of Imuran between the 3rd and 11th day after conception.-J. B. S.


The "Swiss-type" of agammaglobulinemia is reported in two sisters whose parents are first cousins. The disease is characterized by an inevitably fatal course, severe lymphopenia, severe hypoplasia of the whole lymphoreticular system, especially of the thymus, absence of all three immunoglobulins and a recessive autosomal mode of transmission. Because the thymus appears to be essential for the normal development of immune competence and because the disease is invariably fatal, thymus tissue was transplanted into the patients. A transient rise in the lymphocytes followed, but both children later died of pulmonary infections.-P. B.


Human leukocytes contain an enzyme system which degrades thymidine to thymine and dihydrothymine, breakdown products which are not used by the cells for DNA synthesis. In normal peripheral blood, these enzymes are considerably more active in polymorphonuclear leukocytes than in lymphocytes. Phytohemagglutinin (PHA) stimulates human leukocytes to transform into large dividing blast cells. An investigation of the changes in this enzyme system which occur in lymphocytes when they are induced to change from the resting to the activated state by incubation with PHA is described. The change in metabolism of lymphocytes is associated with a considerable increase in the rate of degradation of thymidine. Unlike normal cells, the degradation by PHA-stimulated cells proceeds very slowly between thymine and dihydrothymine, which would indicate a disproportional increase in thymidine phosphorylase activity. There is no direct relation between the activity of thymidine degrading enzymes and the number of cells synthesizing DNA. A failure to be aware of the degradation of thymidine-H3 in experiments in which PHA-stimulated cells are labeled over a long period with a single dose of the isotope can lead to errors in the interpretation of kinetics and chromosome labeling patterns.-G. M.


A 48-year-old woman was admitted with symptoms and signs of hepatic cirrhosis. The white
blood cell count was 10,000–30,000 with meta-
myelocytes and rare promyelocytes and myelo-
cytes. It was noteworthy that 80 per cent of
neutrophil leukocytes had a nuclear anomaly in
peripheral blood; 50 per cent in bone marrow.
Drumstick-shaped projections were seen and the
nuclear anomaly consisted of abnormal regular-
ulgar formation of the segmented nucleus, thin mem-
branous protrusions from part of the nucleus,
connection of two segmented nuclei with a thin
nuclear substance and variation of the nuclei. On
electron microscopic study, almost all of these
cells revealed more rapid maturation of the
nucleus than of the cytoplasm. Myelocytes, meta-
myelocytes and mature neutrophil leukocytes had
abnormal protrusions of the nuclear membrane,
although of lesser degree in bone marrow. This
protruded portion was covered by two folds, inner
and outer nuclear membranes, between which was
a uniform layer of nuclear substance of high elec-
tron density and containing cytoplasmic elements,
vesicles, mitochondria and specific granules. On
occasion, one or several parts of the protruded
portion had small bilateral bulgings in which the
nuclear substance was found to be uniformly dis-
persed, along with some vesicles. The chromosomes
of neutrophils, both in peripheral blood and bone
marrow, did not reveal any abnormality. More-
over, in her family, no other instances of nuclear
anomaly were detected.—K. F.

NUCLEAR PROJECTIONS OF NEUTROPHILS IN THE
13–15 TRISOMY SYNDROME. R. N. Fine, M. Y.
F. Woo Wang and C. W. Heath, Jr. From the
Boston University Medical Center, Boston, Mass.

Normal neonates and an infant with 13–15
trisomy demonstrated nuclear projections in num-
bers which did not differ significantly. Thus, the
presence of these projections does not appear to
be a valuable test for 13–15 trisomy.—J. B. S.

EXPERIMENTAL STUDIES ON THE LIFE SPAN OF
THE NEUTROPHIL. DETERMINATION OF THE
LIFE SPAN OF THE NEUTROPHIL BY SERIAL IN-
JECTIONS OF COLCHICINE. K. Chiba. From the
University of Tokyo, Toyko, Japan. Acta

Rabbits were injected intravenously with 1
mg./Kg. of Colcemid every 4 hours for 72 hours.
Following the initial injection, the neutrophil
counts started to increase within 4 hours and
reached a steady state by 8 hours. This level was
maintained until 24 hours. Thereafter, it fell off
in an exponential curve and practically dis-
appeared at around 52 hours. A single intravenous
injection of antileukocyte antibody at the be-
ginning of the descending phase caused a rapid
disappearance of the neutrophils in 15 minutes,
eliminating the possibility of influx of neutrophils
either from the bone marrow or extravascular
space. In an experiment with leukocyte transfu-
sion, Colcemid per se did not seem to influence
neutrophil survival. Thus, a half disappearance
time of peripheral neutrophils of 8.9 ± 2.4 hours
was obtained by analyzing the exponential curve.
Cytokinetics of the neutrophil in the intravas-
cular phase were discussed from the results in ex-
periments where rabbits were treated with tur-
pentine, splenectomy or epinephrine in addition
to Colcemid or with ACTH. This life span of the
neutrophil should be understood as survival in the
intravascular phase under the administration
of Colcemid, rather than in the normal condition.
—K. F.

EFFECT OF DEFICIENCY OF DIFFERENT AMINO-
ACIDS ON EOSINOPHIL PRODUCTION AND DIS-
TRIBUTION IN BLOOD AND GASTRO-INTESTINAL
MUCOSA. ROLE OF ADRENAL CORTEX IN DE-
PRIVATION EOSINOPENIA. A. Aschkenasy. From

Young male rats (97) were divided into 13
groups, subjected for 25 days to the following
diets: (1) 18 per cent casein; (2) a complete
mixture of 19 amino-acids; (3) no protein or
amino-acids; (4) 13 diets with 17 per cent in-
complete amino-acid mixtures. While diets de-
prived of lysine or sulfur amino-acids were devoid
of noxious effects on eosinophils, the majority
of incomplete amino-acid diets induced eosino-
penia which was sometimes even more pro-
nounced than after complete nitrogen depriva-
tion. Deprivation of valine provoked a maturation
arrest of marrow eosinophils and an almost
complete disappearance of these cells from the
blood and gastrointestinal mucosa. After depriva-
tion of histidine, eosinopenia was much less
pronounced than with valine or isoleucine-defici-
ent diets. All these diets induced relative hyper-
trophy of adrenals and involution of the thymus;
both effects were maximal in rats deprived of
isoleucine or valine. Young male rats (34) were
divided into two groups and received either a
diet with 18 per cent casein or no protein for 21
days. In each group, certain animals were
adrenalectomized and received amounts of salt
and cortisone just sufficient to permit survival.
Blood eosinopenia and thymus atrophy induced by protein deprivation were exactly the same in intact and in adrenalectomized rats maintained on constant hormonal intake. In casein-fed controls, eosinophil levels and thymus weights remained unchanged in both groups.

Thus, both reduction of eosinophil levels and thymus atrophy of protein deprived rats appeared to depend, at least in part, on an abnormal sensitivity of these cells and this organ to the catabolic action of endogenous glucocorticoids.—G. M.


A review of lymphoreticular tumors in 942 biopsy and necropsy samples from West and Central French-speaking Africa indicated the peculiar incidence of facial hematosarcomas during childhood. More than 400 Burkitt facial tumors, clinically detected by otorhinolaryngologists, ophthalmologists and stomatologists, have been published in Africa. In a survey (1950–1962), bone localization of tumors affected children and adults in 43 and 14 per cent, respectively. The patients were 82 boys and 47 girls under 15, most between 4 and 8. This disorder rarely affected adults, was observed in all Negro-African races without any tribal predilection in tropical Africa and affected White African races, such as Maures and Targui. Reported facial lymphoreticular tumors include 33 similar cases in France, 30 in New Guinea and rare cases on the Asiatic and American continents. In tropical Africa, the facial site was the best known and it could remain the only symptom during the entire course. This localization, however, was also associated with other long bone tumors, abdominal nodules, retroperitoneal masses and paraplegia. Clinical manifestations were pleomorphic. Postmortem studies revealed other sites; ovaries, thyroid, adrenals, peritoneum and digestive tract, kidneys and heart. The integrity of the lungs, the absence of adenopathy and the abnormal peripheral blood were elements of some value for a clinical diagnosis. Bone marrow was sometimes invaded. Spontaneous evolution lasted from several weeks to a few months. In most cases, therapy was of no value, for death occurred within a few weeks. In exceptional cases, radiotherapy and chemotherapy allowed a remission of 2 years. Histologic patterns were uniform: a round cell sarcoma with undifferentiated tumor cells, although cytologic variations may be seen. Present research in histochemistry, electron microscopy, immunoelectrophoresis and tissue culture were reviewed. A viral etiology was suggested and serologic studies directed toward the arbovirus were detailed.—G. M.

ABSTRACTS


Variation in a specific serum alkaline phosphatase component, detected by starch gel electrophoresis, is genetically controlled. Expression is masked or inhibited in individuals of blood group A and in nonsecretors of ABH blood group substances. The nonsecretor effect is apparently not directly related to the Lewis blood group system. No association with other blood group or serum protein group systems or with sex has been detected. Among group B and O secretors, in which phosphatase expression is not inhibited, a significant age effect has been defined. The mean level of the specific phosphatase in adults is approximately 50 per cent higher than that in children below the age of 16. Twin studies and family studies of this variation strongly indicate that it is genetically controlled.—H. H. F.


Using antisera against haptoglobins 2-2, 2-1, and 1-1, it was found that hemoglobin blocked several antigenic determinants of the native haptoglobins. Many of the specific antigenic determinants of Hp2-2, however, remained free, so that the anti-Hp2-2 sera could still distinguish the Hp2-2 hemoglobin complex from Hp1-1. One mole of hemoglobin was necessary to block the maximum number of antigenic determinants of one mole of Hp1-1.—H. H. F.