HEMOSTASIS


In 36 cases, Factor XI was measured and in 35 cases, Factor XIII was studied. Factor XI values were very low during the first week and reached normal values during the fourth week. Partial thromboplastin times behaved similarly, but were not completely normalized after four weeks. Factor XIII was within normal limits.-P. d. N.


Thirty cases of leukemia with hyperfibrinolysis were compared with 12 patients with leukemia, but without hyperfibrinolysis. No significant differences in fibrinogen concentration were found, nor was there a correlation between fibrinogen concentration and hyperfibrinolysis.-P. d. N.


The method involved estimation of plasma clotting activity in a situation intermediate between in vitro and in vivo. Aortic intima of guinea pig and pig had the ability to promote the clot formation of platelet-poor plasma with Ca++ in the absence of brain extracts. Saline extracts from the intimal surface, containing a small amount of B+ adsorbed plasma, were more active for plasma clotting than was saline alone. Clot promoting activity of large vein intima of pig was quite similar to that of pig aortic intima, whereas with guinea pig venous intima, an inhibitory effect was observed. —K. F.


Eighty-eight patients with acute leukemia and 32 patients with chronic leukemia, aged 2 to 73, were studied and no correlation between leukocytosis and fibrinolysis was found.-P. d. N.

A young girl with post-varicella purpura fulminans and early signs of gangrene was treated with heparin. An initial intravenous dose of 50 units/Kg. was followed by 100 units/Kg. every four hours by IV drip. Preheparinization coagulation studies revealed a platelet count of 114,000, prothrombin of 0, and fibrinogen of 15 mg./100 ml. Two days after the onset of therapy, coagulation studies were essentially normal and the child made a complete recovery, except for residual scarring of the gangrenous area of her leg.—J. B. S.


Intestinal intramural hematomas constitute a rare complication of anticoagulant therapy. The clinical manifestations are those of progressive intestinal obstruction, sometimes with hemorrhage, meteorism and, occasionally, a palpable mass. The presence of associated cutaneous or visceral hemorrhages and the history of anticoagulant therapy should lead to the diagnosis which is confirmed by a very abnormal Quick’s time. Confirmation is also provided by barium studies with evidence of segmental stenosis, often jejunal, especially with a bristled or palisade-like picture. Regression of the hematoma occurs in the absence of sequelae. Surgery is hazardous but may be justified in case of uncertain diagnosis or with fear of an associated lesion.—J. C.


Abnormal circulating anticoagulants were found in 22 patients, especially in connection with intraocular hemorrhages. A similar finding was observed, however, in other patients with recurrent conjunctival or retinal and vitreous hemorrhages secondary to other diseases. This finding was not considered to be characteristic of Eales disease. In this condition, vascular lesions and the “reactive” production of circulating anticoagulants were considered important for the onset of hemorrhages. These concepts should justify corticosteroid therapy.—P. d. N.


Fresh platelet-free plasma was infused into 17 children with acute ITP, 30 ml./Kg., in divided doses over 24 hours. Twelve patients had an increase in circulating platelets 12 to 48 hours after completion of the infusions. The remissions thus produced apparently persisted in each case. Among the other six patients were three who improved clinically and a week later had platelet counts which were normal or near-normal.—J. B. S.


Three youngsters with chronic ITP which had been unresponsive to steroids during 3 to 18 months of therapy were essentially unresponsive to 6 MP and, later, vincristine. Vincristine produced a partial response in each patient but was associated with significant toxicity. Fresh platelet-free plasma infusions were ineffective. All three children responded to splenectomy.—J. B. S.


Five young children with severe iron deficiency anemia had significant thrombocytopenia. Bone marrow megakaryocytes were variable: in two instances, the number was decreased and in three specimens there was no evidence of platelet budding. Platelet counts rose steeply after institution of iron therapy and remained at supranormal levels for a short time.—J. B. S.


Nine patients with polycythemia and 5 patients with hemorrhagic thrombocytopenia were treated. With only one exception, a patient who died from mesenteric thrombosis, remissions lasting from 5 to 48 months were obtained in the former disease and from 3 to 62 months in the latter. In three patients with polycythemia, relapses were controlled by additional treatment. No significant undesirable side-effects were observed.—P. d. N.

Changes in Oxyindole Acetic Acid Excretion
**ABSTRACTS**


In 67 cases, the thrombocyte count was reduced during the use of therapeutic doses of zitofenton. In seven cases where the thrombocyte count was not reduced, oxyindole acetic acid excretion did not change. In cases where the thrombocyte count was lowered, oxyindole acetic acid excretion was also diminished.—S. R. H.

ERYTHROCYTES


Hemoglobin Cja a ß-chain abnormality with an electrophoretic mobility slightly anodal to Hb C on starch gel at pH 8.6, resembles Hb S in that it gels on deoxygenation and is relatively insoluble in the deoxygenated state. Peptide and amino acid analysis of the isolated peptide chain reveals the presence of two substitutions, ß6 (or ß7 Val) and ß73 Asn. The authors suggest that such a molecule might arise by either: 1) a second mutation in an individual already carrying a ß*a* locus, or 2) crossover between chromosomes carrying a ß6 mutation and a ß73 Asn mutation. (Abstracter’s comment: Gerald and Rath have recently reported a similar, possibly identical, situation in Hb Cjia, p. 73.)—T. F. N. (Editor’s Note: In the more complete report, J. Biol. Chem. 242:248, 1967, this hemoglobin was demonstrated to have the structure ßa* ßβ6 val, ß73 Asn.)


These two abnormal hemoglobins with the structure ßa* ß and ß56 Asp have been found in a family in Thialand and a Hakkanese Chinese family. No hematologic abnormalities have been associated with this hemoglobinopathy.—T. F. N.


Children with sickle cell anemia had fluid intakes and urinary outputs significantly greater than normal. These increases were associated with the low serum and urine osmolality. The authors interpreted the low serum osmolality as evidence that the increased intake, was, at least in part, the cause and not the result of increased urine flow. They believed that the excessive drinking was habitual and resulted from parental urging. This concept assumed that polydipsia appeared after the family was aware of the diagnosis. No evidence was presented to support this supposition.—J. B. S.


Alkal-resistant hemoglobin (Hb F) was found in concentrations of over 3 per cent in 63 per cent of 62 Negro patients with sickle cell disease. Approximately 80 per cent of the females of all ages had increased levels of Hb F. A similar percentage was found in males below the age of 14. In males age 15 or older, however, increased levels were found in only 26 per cent. The author suggested that males with sickle cell anemia cease producing Hb F at puberty and postulated that one or more of the female hormones may play a role in the regulation of Hb F production.—T. F. N.


A 40 year old Negro male had sickle cell trait, a refractory anemia characterized by pancytopenia and hemolysis and a superimposed nutritional anemia responsive to folic acid (no serum or red cell folate levels reported). At the time when the bone marrow showed megaloblastic features, the level of Hb F was 58 per cent. Following therapy, the level fell to 40. (Abstracter’s comment: This case represents another example illustrating possible environmental influences on the rate of synthesis of specific polypeptide chains.)—T. F. N.

The test was used in 35 normal subjects and 120 patients and it gave satisfactory results as a complementary diagnostic aid.—P. d. N.


Morphologic and biochemical changes in the spleens of transfusion-induced polycythemic mice were followed serially after administration of partially purified urinary erythropoietin. A few large basophilic erythroblasts appeared 24 hours after injection in splenic imprint specimens from polycythemic mice in which erythroid precursors had completely disappeared. Forty-eight hours after erythropoietin injection, numerous small erythroblasts with polychromatophilic cytoplasm were noted. Changes in radiiodin uptake, activity of ALA synthetase, ALA dehydrase, iron-protoporphyrin chelating enzyme, heme synthesis, and globin synthesis in the spleen were followed. Activity of ALA synthetase, which had been deficient in hypertransfused mice, started to appear 8 hours after erythropoietin injection. Increased radiiodin uptake, ALA dehydrase activity, globin synthesis, heme synthesis and iron-protoporphyrin chelating enzyme activity were observed 8, 12, 24 and 28 hours after erythropoietin injection. Erythropoietin-induced stem cell differentiation was demonstrated both morphologically and biochemically. In the process of differentiation, a series of enzyme activities related to heme synthesis were induced before the appearance of morphologically recognizable erythroblasts. The appearance of ALA synthetase activity after erythropoietin injection was thought to be the result of DNA-directed RNA synthesis which was completed 6 hours after the injection.—K. F.


Mean daily blood loss into the intestinal tract in healthy infants and in a group of infants with acute infections differed insignificantly, being 0.64 ml./day and 0.43 ml./day, respectively. Among infants with acute diarrhea, the mean loss was significantly higher, 1.85 ml./day. There was no correlation between the observed daily blood loss and the infant’s hemoglobin level. The daily loss, in healthy infants more than 0.5 ml./day, was higher in terms of body size and red cell mass than the daily blood loss reported for healthy adults.—J. B. S.


The ages, presenting signs, symptoms and clinical courses of a large group of youngsters with acute iron overload were described. The correlation between serum iron levels and the presence of shock or coma was noted. All 144 patients who did not develop shock or coma survived, and three deaths occurred in the 28 children who developed coma and/or shock. The recommended regimen of deferoxamine therapy was discussed.—J. B. S.


The authors review the pathophysiology of the thalassemias and suggest that many of the clinical symptoms are due to the precipitation and removal of chains which are made in excess (the alpha chains in beta-thalassemia, beta chains in Hb H disease), rather than to a deficiency of the affected chains. They suggest that therapy should be directed towards suppression of the excess chain production.—T. F. N.


In a case of hydrops fetalis associated with Hb Bart’s in a Chinese family, hemoglobin electrophoresis on starch-urea gel indicated the complete absence of alpha-chains in the fetus. Family studies led to the suggestion that the father was heterozygous for both alpha- and beta-thalassemia; his disease was similar in severity to beta-thalassemia trait. In neither the father nor the mother, both presumably heterozygous for alpha-thalassemia, could Hb H be demonstrated, either by electrophoresis or by incubation of erythrocytes with brilliant cresyl blue. (Abstractor’s comment: This family points up the extreme difficulty inherent in establishing the diagnosis of alpha-thalassemia. If one accepts the hypothesis originally pro-
A 3.0 per cent, MCH 22) and hereditary per-
Rb S genes for alpha-thalassemia, beta-thalassemia and
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semia in the adult patient?—T. F. N.

THE INTERACTION OF HEREDITARY PERSISTENCE OF
FETAL HEMOGLOBIN AND BETA THALASSEMIA.
G. A. Becker and E. C. Rossi. From Marquette
University School of Medicine, Milwaukee, Wis.

A Negro female, aged 30, was found to be
heterozygous for both beta thalassemia trait (Hb
A_2 3.0 per cent, MCH 22) and hereditary per-
sistence of fetal hemoglobin (HPFH) trait (Hb
F 64 per cent with homogenous distribution on his-
tologic examination). The patient had a Hb of
13.7 Gm per 100 ml. Three offspring had either
beta thalassemia trait or HPFH trait, but not
both, suggesting that these two mutations were
closely linked, if not allelic.—T. F. N.

ALPHA-BETA THALASSEMIA IN A NEGRO FAMILY.
H. A. Pearson. From University of Florida Col-
lege of Medicine, Gainesville, Fla. New Eng. J.

In a Negro family whose members possess
genes for alpha-thalassemia, beta-thalassemia and
Hb S alone and in various combinations, three
members are believed to have alpha-beta thalas-
semia. Despite the presence of two thalassemia
genes, these patients have hematologic findings no
more severe than in either trait alone. Segregation
analysis of the children provides further evidence
confirming the non-allelism of alpha-thalassemia
and beta structural loci. (Abstractor's comment:
Neither Hb H nor Hb Bart's was found in any of
these patients. As pointed out before, clinical and
biochemical criteria for the diagnosis of alpha-
thalassemia are sadly lacking and differentiation
from other forms of familial hypochromic
anemia becomes increasingly difficult).—T. F. N.

STUDY OF A ANTIGEN IN ERYTHROCYTES AND SA-
LIVA IN THALASSEMIA. E. Gandini, C. Menini
and N. Ricci. From the University, Ferrara,

Two cell populations were identified in the
Peripheral blood of Cooley's anemia patients by
means of appropriate dilutions of a saline extract
of Dolichos biflorus (lectin anti A_1). The non-
agglutinated cells (depressed A_1 antigen) con-
tained predominantly Hb F and only fetal antigen
i; agglutinated cells (antigen A_1, normal) contained
predominantly adult Hb and both fetal antigen i
and adult antigen I. In saliva, a higher inhibitory
titer was found when tested with group A_1 cells
from Cooley's anemia patients when compared
with titers obtained with normal A_1 cells. In
saliva from normal secretors, a similar inhibitory
titer was found with both types of A_1 cells.—
P. d. N.

HAPTOGLOBIN AND HEMOLYTIC DISEASES. D. Rizzi,
F. Daniele and U. Gillardi. From the University,
Bari, Italy. Progr. Med. (Napoli) 22:766-777,
1966.

In 63 cases of hemolytic anemia, there was a
correlation between the severity of the hemolytic
phenomenon and the level of haptoglobin.—P. d. N.

MICRO-ANGIOPATHY AND THE UREMIC-ANEMIC
SYNDROME IN INFANTS. REPORT OF 5 NEW CASES.
R. Mallet, M. Ribierre, F. Bonnenfant, L. Rey-

An account of 5 new curable cases and a review
of 62 others, almost exclusively in infants, were
presented. Renal biopsies suggested that the syn-
drome represented a special evolutionary form of
microangiopathy. The term curable uremic-anemic
syndrome should be replaced by microangiopathy
with favorable evolution. An infectious origin,
probably viral, was suspected.—J. C.

POPULATION GENETIC STUDIES IN THE CONGO. I.
GLUCOSE-6-PHOSPHATE DEHYDROGENASE DE-
FIENCY, HEMOGLOBIN S, AND MALARIA. A. G.
Motulsky, J. Vandepitte and G. R. Fraser. From
the University of Washington, Seattle, Wash.

A survey for G-6-PD deficiency, Hb S and
malaria was carried out on 1594 male natives
from the Congo. Comparison of malarial infestation
rates, parasite counts, gene frequencies related to
mean age of boys with Hb S, G-6-PD deficiency
and a combination of the two led to the suggestion
of protection by sickling and G-6-PD deficiency
traits against malaria. The data on age distribution
and gene frequency at different ages indicated that
the strength of protection afforded by G-6-PD de-
fiency was much less than that provided by
sickling trait. There was some evidence of additive
protection when both traits were present. The
frequency of the combination, sickle trait and


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G-6-PD deficiency, was somewhat higher than expected by chance alone, but was not statistically significant. -T. F. N.


The correlation between hemolytic anemia accompanying severe infection and the presence of G-6-PD deficiency which these authors described in 1959 was not found when the association was investigated in a larger group of youngsters. -J. B. S.


Dr. Itano reiterates the structure-rate hypothesis for the regulation of hemoglobin synthesis first suggested by Itano and Pauling several years ago. In its simplest form, this hypothesis states, "The structural genes for the chains of hemoglobin control not only the amino acid sequence, but also the net rate of synthesis of the chains." A model is proposed in which completed polypeptide chains are released from the polysome complex at a rate determined by the slowest step in their assembly. Decreased synthesis can result from a mutation that results in a slower step anywhere along the mRNA. An increased rate of synthesis, however, can only result from a change in a step which is initially rate limiting. The net quantity of a specific hemoglobin in the peripheral blood depends not only on the rate of synthesis of the component polypeptide chains, but also on the rate of destruction of one or another hemoglobin present in the peripheral blood. Balanced chain production, i.e., alpha vs. non-alpha chains, is probably maintained by selective pressure against gross imbalances in production which lead to hemolytic anemia or death. -T. F. N.


Hemoglobin A1,, a normal minor hemoglobin component in human adults, is identifiable only by column chromatography and its significance in normal or pathologic erythrocytes is still uncertain. The component is a condensation product between the N-terminus of one of the two beta chains of Hb A and one molecule of a ketone or aldehyde; this appears to be the only structural difference between Hb A,, and Hb A1,, the major fraction of Hb A. Hb A,, a normal constituent of all red cells, irrespective of physiologic age, appears to be synthesized in vitro concurrently with Hb A. Whether or not it is in a dynamic equilibrium with the latter remains an open question. -T. F. N.


The authors have established the amino acid sequence of the gamma chain of bovine fetal hemoglobin and discuss the variations in the structure of the human beta and alpha chains as compared to those found in cattle and horses. Examination of the amino acid residue differences with respect to their localization in the three-dimensional molecule reveals that the invariant sections of the non-alpha polypeptide enclose the heme group, are present between the closer heme groups of different chains and are at the points of contact between identical chains. These areas may play a major role in determining the proper function of the hemoglobin molecule. (Abstracter's comment: This paper represents an excellent summary of what was known about the relationship between structure and function in the hemoglobin molecule at the beginning of 1966). -T. F. N.

PASSENGEMCGLOBLININTOUREENERYLYMPH:

In thoracic duct and cervical trunk lymph, the maximum L/P concentration ratios of total extracorpuscular hemoglobin and 35,000 Mol. weight dextran were found to be practically identical and significantly higher than the values for 131I-albumin. Equilibration between plasma and lymph of injected hemoglobin was, however, slower than equilibration of dextran and dextran was excreted more readily by the kidney. Hemoglobin probably passed more readily than plasma albumin through capillary membranes, the visceral and peripheral capillaries being nearly as permeable to hemoglobin.
as they were to 35,000 Mol. weight dextran. This phenomenon might be due to the dissociation of the hemoglobin tetramer to the dimeric species and to a rapid reassociation on the outer side of the membrane. The same phenomenon might account for the filtrability of hemoglobin in the glomerular capillaries and for its excretion with urine.—S. R. H.

LEUKOCYTES

VINCRISTINE IN THE TREATMENT OF ACUTE LEUKEMIA IN CHILDREN. R. M. Heyn, Chairman, Writing Committee. From the Stritch School of Medicine, Chicago, Ill. Pediatrics 38:82-91, 1966.

Vincristine induced complete marrow remission in 43 per cent of children with acute leukemia refractory to other drugs. The mean duration of disease prior to this therapy was 13.5 months. Appearance of remission was quite rapid and persisted for 2 months with or without maintenance therapy. Toxicity was seen in half the children and included alopecia (48 per cent), gastrointestinal disturbances (43 per cent), and neurologic abnormalities (weakness, decreased tendon reflexes, neuralgia and/or paresthesias) in 53 per cent. Vincristine did not prevent onset of meningeal leukemia. The dose was 0.075 mg/Kg, intravenously every 7 days for a month. Unresponsive patients received 2 additional weekly doses of 0.1 mg/Kg., but these additional doses did not increase the remission rate.—J. B. S.

INDUCTION OF REMISSION IN ACUTE LEUKEMIA OF CHILDHOOD BY COMBINATION OF PREDNISONE AND EITHER 6-MERCAPTOPURINE OR METHOTREXATE. W. Kricit, C. Brubaker, J. Hartmann, M. L. Murphy, M. Pierce and G. Thatcher. From the University of Minnesota Medical School, Minneapolis, Minn. J. Pediat. 68:965-968, 1966.

Complete or good partial remission in acute lymphocytic or stem cell leukemia of childhood occurred in approximately 90 per cent of cases treated with daily oral administration of prednisone, 60 mg./M.² plus either methotrexate, 3.3 mg./M.² or 6-MP, 70 mg./M.². The response among children with other types of acute leukemia was below 50 per cent on either regimen.—J. B. S.

LONG-TERM REMISSIONS IN CHILDHOOD ACUTE LEUKEMIA; USE OF INFREQUENT INFUSIONS OF METHOTREXATE; SUPPORTIVE ROLES OF PLATELET TRANSFUSIONS AND CITROVORUM FACTOR.


Methotrexate (MTX), 175 to 525 mg./M.², was given intravenously on 2 consecutive days to 15 children with acute leukemia. Sixty per cent of the dose was given in 5 minutes and the remainder over 4 hours. Antibacterial and antifungal mouth washes were used prophylactically and citrovorum factor was given orally 4 times a day for several days to some. Platelet transfusions were given to sustain platelet counts greater than 50,000/mm.³. Steroids and other antimetabolites were used to supplement the MTX. Half the patients were given 1 gm. cyclophosphamide/M.² 14 days after MTX and this MTX-cytotoxic cycle was repeated every month. All patients in this group remained in remission for at least one year. The other patients received cyclic therapy with MTX plus prednisone, cytoxan and 6MP with similar results. Thrombocytopenia and leukopenia were frequent, but the general condition of the patients remained satisfactory. There was an apparent decrease in buccal ulcers using either folic acid or antibiotic mouth washes. Transient elevation of SGOT was seen often during the week following MTX infusion.—J. B. S.


A patient with warm antibodies had been described previously, but the findings deserved further comment. The apparently paradoxical situation of an autoimmune condition with deficient antibody formation was explained on the basis of the gradual replacement of the normal, immunologically competent lymphocytic clones by a neoplastically mutated clone which was able to produce autoantibodies, but no others.—P. d. N.

VITAMIN B₁₂ STUDIES IN MYELOGENOUS LEUKEMIA OF CHILDHOOD. G. Skaff, J. T. Louman, R. Bruning and W. Kricit. From the University of Minnesota School of Medicine, Minneapolis, Minn. J. Pediat. 68:890-894, 1966.

Serum B₁₂ levels were elevated in 5 of 8 children with CML and in 1 of 4 with acute myelogenous leukemia. In the six patients in whom plasma radioactive vitamin B₁₂ clearance was evaluated, the clearance was prolonged.—J. B. S.

Two of three siblings with Downs syndrome and 15/21 chromosome translocation demonstrated evidence of abnormal hemopoiesis shortly after birth. One infant presented with hepatomegaly and plethora, associated with marked peripheral blood erythroblastosis, mild leukocytosis and mild thrombocytopenia. Peripheral blood and bone marrow smears suggested chronic myelogenous leukemia. The infant died at 10 days of apparent cardiorespiratory failure and post-mortem examination revealed infiltration of nodes and viscerum by immature myeloid elements. The second infant presented with hepatosplenomegaly and hemolytic jaundice on the first day of life. The peripheral blood smears were similar to those obtained from his sister, although leukocytosis and thrombopenia were more pronounced. Over the ensuing two years, intermittent hepatosplenomegaly, anemia and thrombocytopenia were noted. The peripheral blood changes suggesting leukemoid reaction progressively improved and were gone at last examination when the infant was 2 years old.—J. B. S.


The analyses revealed three essential facts: 1) the frequency and kind of infections which almost exclusively attacked the breathing apparatus; 2) the existence of a deficiency in immunity whose characteristics are still difficult to define; and 3) the relatively frequent evolution to a malignant tumor of the lympho-reticular system. The most constant immunologic abnormalities were: absence of development of the entire lymphoid system, lowering or complete disappearance of gamma-A-globulin, subnormal levels of circulating antibodies, reduction in blood lymphocytes, absence of the reaction of delayed hypersensitivity and increased tolerance to cutaneous homografts. Respiratory infections may exist without abnormalities in circulating immunoglobulins, abnormalities of the lymphocytes seem to play a predominant role and these cells behave very abnormally when they are cultured. The abnormality in the circulating immunoglobulins may not affect the gamma-A-globulins, but the gamma-G-globulins. The deficiency in immunity and the neurologic manifestations are hereditary.—J. C.


Salmonella typhi bacteria were infused into the afferent lymphatic duct of a popliteal node of a sheep and all the efferent lymph was collected. Under these circumstances, no systemic immune response was observed. Cells collected in a similar fashion, however, could adoptively confer an immune response to another sheep. The ultra structure of these cells was described in detail. The authors proposed that, although these cells derived from the efferent lymph may merely have differentiated into antibody forming cells in distant sites in the animal, it was also possible that these cells acted indirectly by transferring "information" to other cells which would then produce antibody.—I. G.


An adequate dose of irradiation, at least 2-3,000 r skin dose, altered the shape of the lymphocyte curve produced by adrenaline. Failure of mobilization of the lymphocyte reserve and an absence of lymphocytosis were observed as a result of direct inhibition of the lymphoreticular system. This test seemed to be suitable for establishing the presence of irradiation damage or for excluding with certainty a lesion due to irradiation.—S. R. H.


Morphologic differentiation of granulocytes in rat bone marrow was studied with the electron microscope and some cytochemical reactions were applied to the glutaraldehyde-fixed sections, which were postfixed in OsO4, ATPase was detected on the plasma membrane of the neutrophilic myelo-
cytes and in a few granules. Na+ was found mainly along the plasma membrane. TPPase was present in the Golgi apparatus, AC phosphate in the Golgi sacs and in a few granular components, and ALPase in acidophilic granules. The reaction product was found in the entire matrix of the immature granule, but, as maturation proceeded, it became concentrated in the central crystalloid portion. The mature crystalloid, however, seemed to be negative. Peroxidase was demonstrated in the neutrophilic and acidophilic granules, but not in the basophilic granule. The reaction was strongest in the peripheral matrix of each granule and was negative in the central crystalloid of the eosinophilic granule. Certain stages of neutrophilic and eosinophilic myelocytes showed a reaction in the granular endoplasmic reticulum and Golgi complex. The reaction was strongest in the eosinophilic myelocyte. The possibility of regarding granulocytes as unicellular glandular cells was discussed.—K. F.


Long-term heparin treatment caused inhibition of the human and rat suprarenal gland. Elevation in the number of circulating eosinophils of human subjects occurred 60 minutes after a single i.v. dose of heparin. Spironolactone also increased the number of eosinophils. The combined effect of a single heparin injection and simultaneous administration of ACTH, cortisone or aldosterone moderated, but did not eliminate, the specific heparin effect. Decrease in adrenocortical function occurred only after long-term heparin treatment, but increased eosinophils were found after a single dose of heparin. The effect of heparin could not be mediated through inhibition of adrenal cortical activity. Heparin may cause quick mobilization of eosinophils from other tissues and may play a significant role in the regulation of eosinophils in peripheral blood.—S. R. H.


A low molecular weight, dialyzable extract of leukocytes from humans with delayed sensitivity was prepared. This factor, when added with PPD to leukocyte cultures of tuberculin negative individuals, caused leukocyte stimulation. A similar extract prepared from leukocytes of tuberculin negative individuals did not have this property. The authors raised the question as to whether or not this material and the leukocyte response elicited by it were related to in vivo passive transfer of PPD sensitivity with cell free transfer factor.—I. G.

MISCELLANEOUS


By means of observations of phagocytosis of metal colloid particles by mouse ascites macrophages, the mechanism of transport of substances was studied. Adsorption of the particles to specific areas of the cell surface was the first and indispensable step. Ehrlich ascites tumor cells adsorbed the particles and phagocytized them when they were incubated with histone or protamine. Small rhopheocytic vesicles formed at the area where the particles were adsorbed and developed into large phagocytic vacuoles. Finally, a number of phagocytic vesicles formed, but most of them were found to interconnect with the cell environment by canaliculi through which the particles were incessantly transported, probably by the membrane flow of the outer half layer of the cell membrane, i.e., the flow of the outer lipid monomolecular layer and the protein layer on the cell surface. The possible changes in molecular arrangement of cell membrane and cytoplasm induced by adsorbing the charged colloid particles and the possible mechanism of transport of ions and charged organic compounds through the membrane were discussed from the viewpoint of higher molecular physics.—K. F.


Patients given ordinary gamma globulin intravenously usually develop severe reactions. An adult with agammaglobulinemia had unfavorable reactions to I.M. gamma globulin. He was, therefore, treated with pepsin-modified gamma globulin given I.V. This preparation was tolerated, if given slowly. The author suggested that the absence of the Fc component in the pepsin-modified gamma globulin molecule was the explanation for the lack of reaction.—I. G.

A group of 129 children who had experienced neonatal hyperbilirubinemia with indirect reacting serum bilirubin levels exceeding 20 mg per 100 ml. was examined 5-6 years later. The mean IQ was 104.8. Seven children had some neurological deficits, each had mild to moderately severe hearing loss, three had mild athetosis and three were mildly to moderately aphasic. Only one child, with a peak bilirubin of 30.8 mg per 100 ml., demonstrated significant mental retardation. Six of the children received streptomycin and the seventh was given chloramphenicol.—J. B. S.


Four of 7 isolated cryoglobulins were anticomplementary; even gamma quantities could inhibit the hemolytic effect of complement. The anticomplementary effect was heat sensitive (56 C, 30 minutes) except in one cryoglobulin. Even immunologically identical cryoglobulins were not uniform in respect to anticomplementary effect. There was no close correlation between anticomplementary effect and rheumatoid factor activity, although cryoglobulins with rheumatoid factor were more frequently anticomplementary. Cryoglobulin was primarily responsible for the anticomplementary effect of these sera. The complement of normal serum could be abolished by isolated cryoglobulin.—S. R. H.


In a group of 42 youngsters with rheumatoid arthritis, 14 of 25 girls, but only 2 of 17 boys, demonstrated the presence of antinuclear factors.—J. B. S.


The composition of hemolymph in older insects reflects the kinds of changes which are known to occur with aging. Specific volumes of hemolymph, withdrawn from inbred hosts, have been replaced by equivalent volumes of hemolymph from donors of the same genotype, but of varying ages. The reduction in the life span of hosts injected with hemolymph from older females is explained by the presence of deleterious factors in the hemolymph of older individuals.—O. P. J.


Isolated subfractions of normal gamma G globulins and gamma G type myeloma proteins were studied by starch gel electrophoresis. The polypeptide chains of the proteins were also analyzed after reductive cleavage. The electrophoretic mobility of H chains was found to correspond to that of the intact protein. No correlation was found between electrophoretic patterns of intact gamma G myeloma proteins and their L chain patterns.—S. R. H.