ABSTRACTS

Theodore H. Spaet, M.D., Editor

ABSTRACTERS

Ernest Beutler, M.D., Duarte, Calif.
T. H. Bothwell, M.D., Johannesburg, South Africa
T. E. Brittingham, M.D., St. Louis
I. Chanarin, M.D., London, England
J. B. Chatterjea, M.D., Calcutta, India
Amor I. Chernoff, M.D., Knoxville, Tenn.
G. C. deGruchy, M.D., Melbourne, Australia
Pietro deNicola, M.D., Pavia, Italy
Ludvik Donner, M.D., Prague, Czechoslovakia
A. J. Erslev, M.D., Philadelphia
Theodor M. Fleidner, M.D., Upton, L. I., N. Y.
H. Hugh Fudenberg, M.D., San Francisco, Calif.
Katsuhiro Fukutake, M.D., Tokyo, Japan
Robert Goldstein, M.D., New York City
Victor Herbert, M.D., Boston
Susanna R. Hollan, M.D., Budapest, Hungary
G. Watson James, III, M.D., Richmond, Va.
Michel Jamra, M.D., Sao Paulo, Brazil
Alan Johnson, M.D., New York City
Oliver P. Jones, M.D., Buffalo
Joseph Kasirsky, M.D., Moscow, U.S.S.R.
Sven-Age Killmann, M.D., Copenhagen, Denmark
E. Kowalski, M.D., Warsaw, Poland
Miguel Layrisse, M.D., Caracas, Venezuela
H. Martin, M.D., Frankfurt/Main, Germany
Georges Mathé, M.D., Paris, France
W. J. Mitus, M.D., Boston
Bracha Ramot, M.D., Tel Aviv, Israel
Peter G. Reizenstein, M.D., Stockholm, Sweden
Richard E. Rosenfield, M.D., New York City
Julian Schorr, M.D., New York City
C. Wasastjerna, M.D., Vasa, Finland

ABSTRACTS OF SPECIAL INTEREST


A procedure for the detection of cancer cells is described that does not depend on ordinary morphologic criteria for identification. By this technic cancer cells can generally be found in the blood of patients having early as well as advanced malignancy. In view of these findings it is suggested that the technic would be of value in cancer detection programs. The following method was used: (1) Whole blood (5 ml.) is collected from a peripheral vein and transferred to a centrifuge tube containing 1 mg. of heparin; (2) 0.2 ml. of Phytosemanglutinin isolated from black beans (or bovine fibrinogen) are added and mixed; (3) the sample is then centrifuged at 500 rpm for one minute; (4) the supernatant fluid is transferred to another centrifuge tube and centrifuged fourteen minutes at 3,500 rpm; (5) the supernatant fluid is discarded, and smears made with the sediment are fixed in alcohol-ether (1:1) solution for at least 15 minutes; (6) the slides are stained with acridine orange in accordance with Bertalanaffy’s technic; (7) the final preparations are then examined microscopically under UV light; (8) in counting the number of cells with bright red fluorescence, an indirect method was used based on the number of leukocytes per cu.mm. The peripheral blood of 50 cancer patients was examined for red fluorescence, and neoplastic cells were observed in numbers ranging from 150-840 cells per cu.mm. of blood. Twenty-five patients with non-neoplastic diseases and 15 normal individuals were studied as controls. In these, no similar fluorescent cells were found.—M. J.


The suggestion that an extrinsic factor could explain the modification of agglutinability observed in the A red cells in some patients with acute leukemia has led to a study of the serologic behaviour of normal A red cells transfused into such patients. In the three reported cases, after 9 days, 18 days and 2 months, there was no detectable alterations in normal A red cells as to agglutinability with anti-A or anti-A1 antibody. These observations suggest that only the patient's red cells are modified and the hypothesis of a mutation remains possible; but these facts do not exclude a phenotypic alteration of the erythroblasts.—G. M.

Essential lymphocytophilsis or “infantile lymphoplasmocyte aplasia with alymphocytosis and hypo-γ-globulinemia” occurs in the first few months of life. It presents with persistent, severe phoplasmocyte aplasia with alymphocytosis and M. necroses of buccal and intestinal mucosa, (which despite fatal in the second or third month of life and hypo-γ-globulinemia. The condition is hypo-γ-globulinemia” occurs in the first few LYMPHOCYTOPHTHSIS: representing a very severe impairment of immune entity, separate from other hypo-γ-globulinemias, shows an extreme lymphoplasmocyte aplasia. Be- form intractible diarrhea, skin and lung involvement. Investigations show a well marked lymphocytopenia and hypo-γ-globulinemia. The condition is fatal in the second or third month of life despite γ-globulin administration. Besides 12 typical cases from the literature, there is also an atypical form and a form revealed by progressive gangrenous vaccinia. In all cases, postmortem examination shows an extreme lymphoplasmocyte aplasia. Because of the clinical, hematologic and pathologic features, this disease should be accepted as an entity, separate from other hypo-γ-globulinemias, representing a very severe impairment of immune response.—G. M.

ERYTHROCYTES


The effect of x-rays on heme synthesis in the peripheral blood of hens was investigated with the aid of C14-labeled glycine. This synthesis was found to be increased in red cells given 750 or 1000 r. Doses of 10,000 r and higher progressively inhibited heme synthesis. Aqueous extracts of various organs and serum, and hemolysates of the blood of various animals, both normal and ir- radiated, were investigated in relation to their effects on heme synthesis. The presence of serum enhanced heme synthesis in the system investigated.—E. K.


Erythropoiesis and basal metabolic rate (BMR) were followed in adult rats deprived of proteins.

Additional studies were done on the effect of alimentary deprivation or loading of iodine as well as the action of thyroxine, or cortisone and of 2,4- dinitrophenol (DNP). One group of protein-starved rats was put on a casein diet and the effect of such a repletion diet on tissue oxidation and on anemia was investigated. During the first two weeks of protein deprivation, there was a simulta- neous fall in reticulocyte levels and basal metabolism rate. Iodine added to the diet reduced these effects slightly; repeated injections of thyroxine or cortisone prevented them entirely. DNP had no effect on the reticulocytes, but increased the BMR for some hours following injection. After longer protein deprivation, the BMR, in animals not receiving hormone injections, continued to fall, whereas the reticulocytes again increased as the anemia progressed. The anemia was largely prevented or delayed by thyroxine and by cort- isone, both of which increased the oxygen consumption. During protein repletion, a discharge of reticulocytes into the blood was observed on the sixth day simultaneous with a rapid increase in BMR. Thus, both in the early stages of protein depletion and repletion, there was a direct relationship between oxidation and erythropoiesis. The parallelism was absent both in advanced protein depletion and in the later stages of repletion and, also, immediately after DNP injections. Even when parallelism between erythropoiesis and oxida- tion existed, there was no evidence that erythro- poiesis depended directly on oxygen consumption. Both the reduced reticulocytosis and the fall in BMR early in protein depletion could be due to a general depression of the metabolism of tissues, of which the bone marrow would represent only a small fraction. On the other hand, the opposite changes observed at the beginning of protein repletion could reflect an increase in the whole tissue metabolism. Thus, the changes in BMR would be the effect rather than the cause of changes in erythropoiesis. The anti-anemic action of thyroxine and cortisone could be due not only to their action on oxidation but, also, to a direct stimulation of the erythropoiesis.—G. M.

ANEMIA ASSOCIATED WITH CHRONIC RENAL FAIL- URE, WITH SPECIAL REFERENCE TO KINETICS OF THE ERYTHRON. T. Kuroyanagi. From the School of Medicine, University of Tokyo, Tokyo, Japan. Acta Hemat. Jap. 24:156–175, 1961.

(1) Clinical and statistical studies were car- ried out on 302 cases of kidney disease. The incidence of anemia was 47 per cent. Normochromic anemia was most frequently observed. Serum NPN, serum creatinine, GFR and RPF showed good
correlation with the degree of anemia. Reticulocytosis was frequently found, showing a negative correlation with RPF. Red blood cell half-life correlated with serum NPN, demonstrating an increase in vivo hemolysis in chronic renal failure with azotemia. (2) The bone marrow of uremic patients showed an increase in immature nucleated red blood cells and a relative decrease in mature cells. (3) Both hemosiderin iron and ferritin iron was decreased in the liver of uremic patients. (4) Red cell iron turnover was markedly reduced while plasma iron turnover was slightly elevated in chronic renal failure with elevated serum NPN values, indicating that there is an increased ineffective and a decreased effective erythropoiesis in chronic renal failure with azotemia. (5) Daily red blood cell production rate as measured by Fe59 method was 2-3 times normal, while the red blood cell iron turnover was less than normal. Anemia associated with chronic renal failure is progressive. (6) Prolongation of the period required before reaching the plateau was demonstrated in uremic patients, suggesting prolongation of intermitotic interval. This prolongation was confirmed by a decrease in H3-thymidine uptake by uremic erythroblasts in vitro. (7) An increase in ineffective erythropoiesis is believed to be due to an increase in "abortion" rate in the bone marrow. (8) Shortened life span of red blood cells and increased random cell destruction were demonstrated in uremic patients. (9) Reduced cholinesterase activity in erythrocytes was found in chronic renal failure. (10) Inhibition of metabolism of ADP, 2,3,DPGA and ATP in erythrocytes was demonstrated in uremic patients. This inhibition may be induced by an inhibitory effect of serum NPN on enzyme systems in erythrocytes. It was speculated that inhibition of the metabolism of organic acid soluble phosphate in erythrocytes will result in increased random cell destruction. (11) Administration of NPN of uremic serum reduced Fe59 uptake in starved rats. (12) It may be concluded on the basis of above results that a combination of erythropoiesis inhibition and increased hemolysis is mainly responsible for the development of anemia associated with chronic renal failure. The elevated NPN might influence these processes.—K. F.


Ineffective erythropoiesis was marked in thalassemia major, extramedullary hemopoiesis, refractory normoblastic anemia, megaloblastic anemia, and di Guglielmo disease.—V. H.


In 17 subjects, 7 Gm. of calcium lactate did not correct vitamin B12 malabsorption. This confirms the prior report that 7 to 10 Gm. of calcium lactate did not correct vitamin B12 absorption in 10 subjects with steatorrhea (Herbert, V.: The Megaloblastic Anemias. New York, Grune & Stratton, 1959, p. 32).—V. H.


Intrinsic factor action required calcium (for which magnesium could partially substitute), and a pH of 5.9 or higher.—V. H.


Supraphysiologic (50 µg.) quantities of radioactive vitamin B12 are better absorbed by normal men when administered after a meal. Other workers have reported that food does not enhance the absorption of physiologic quantities of vitamin B12 by normal subjects or patients with pernicious anemia (Deller, Germar, and Witts. Lancet 1: 574, 1961).—V. H.


The plasma vitamin B12-binding protein (B12BP) of man was soluble in sulfosalicylic acid and migrated anodally when it was electrophoresed at pH 4.5 on a starch block. The plasma B12BP of the Rhesus monkey, guinea pig, and rat were only minimally soluble in sulfosalicylic acid and did not migrate anodally when electrophoresed at
ABSTRACTS

pH 4.5. When the vitamin B12-binding capacity of these plasmas was saturated with Co60 vitamin B12, no evidence for an unsaturated, anodally migrating B12BP was found. Chicken plasma contained a protein capable of binding huge amounts of added Co60 vitamin B12. The protein was soluble in sulfosalicylic acid and had an electrophoretic mobility at pH 4.5, similar, but not identical to the human B12BP.—V. H.


An isotope method basically similar to that of Barakat and Ekins (Lancet 2:25, 1961), who used serum rather than intrinsic factor as their vitamin B12 binder.—V. H.


One-tenth to 0.25 mg. of folic acid daily may be hematopetically active in the presence of a low serum vitamin B12 level. The author therefore feels that folic acid should be removed from multivitamin preparations in medically advanced communities with adequate diagnostic facilities, but should be retained in areas where malnutrition is prevalent and medical care inadequate. The subject is debatable, as indicated in an editorial comment appended to the paper. It is likely that multivitamins containing not more than 100 μg. of folic acid per daily dose will eventually prove acceptable to most students of megaloblastic anemia, provided the bottle is labeled “Caution: not more than 100 μg. of folic acid should be taken daily without medical advice.”—V. H.


Serum iron copper has been examined in 89 patients with liver diseases, in 3 patients with nonhepatic diseases and in 84 healthy subjects. In patients with acute hepatitis, iron was found increased an average of 100 cc. to 197 μg. in women, and to 231 μg. per cent in men. The peak of iron increase occurred at the end of the second and at the beginning of the third week of illness in infectious hepatitis, but before the second week in serum hepatitis. In chronic hepatitis, increased plasma iron indicated persistent activity of the disease. Increased plasma iron in chlorpromazine hepatitis indicated the liver cell injury. In cirrhosis, plasma iron changes were of no diagnostic significance. It was found to be normal in 52 per cent, increased in 21 per cent, and decreased in 27 per cent of these patients. A significant increase of plasma copper was found in biliary obstruction; either in primary or secondary biliary cirrhosis, and in jaundice due to obstructions of extrahepatic biliary ducts. Increased plasma copper was also found in chlorpromazine jaundice. In acute hepatitis, plasma copper was slightly increased, not reaching the diagnostic value in the first and second weeks of the disease, steadily decreasing afterwards. In cirrhosis, it was normal in 43 per cent, decreased in 30 per cent, and increased in 28 per cent. Thus simultaneous determination of plasma iron and copper is able to help distinguish jaundice of acute infectious hepatitis from that due to obstruction.—L. D.


Single doses of orally administered ferrous sulfate, containing 1 to 3 μc of Fe59, were given to infants who had moderately severe iron deficiency anemia. An inverse relationship between iron dosage and percentage of utilization for hemoglobin formation was found. The most efficient utilization occurred at an elemental iron dose of 2 mg. per Kg. The mean iron utilization at this dosage was approximately 0.27 mg. per Kg. Since hemoglobin regeneration in iron deficient states
utilizes 0.8 mg. per Kg. iron daily, it is assumed that iron absorption is a limiting factor in the iron utilization from a single dose. In order to supply maximal amounts of iron daily, the author suggests a dosage schedule of 2 mg. per Kg. of elemental iron given three times daily.—J. B. S.


A family is described in which two male first cousins manifested episodes of hemolytic anemia, usually in association with upper respiratory infections. Between episodes they demonstrated mild anemia and moderate reticulocytosis. Both boys had low reduced glutathione levels before acetyl phenylhydrazine (APH), and marked GSH-instability after addition of APH. The levels of G-6-PD activity were very low, and a comparison of the rates of G-6-PD activity at varying concentrations of glucose-6-phosphate, and TPN, and at varying pH, demonstrated qualitative differences from normal G-6-PD. The mothers of the boys were sisters, and each exhibited a reticulocytosis and a moderately depressed level of reduced glutathione in their erythrocytes after incubation with APH. Qualitatively the G-6-PD in the one sister studied was indistinguishable from normal.—J. B. S.


The growth status of 48 children with sickle-cell anemia was compared with their normal siblings and with standards for normal white children. The children with sickle-cell anemia, as a group, weighed less, were shorter, and demonstrated a thinner body build. Skeletal maturation did not appear to be delayed, except perhaps during early adolescence. There was no apparent correlation between growth retardation and the frequency or severity of clinical crises, average degree of anemia, or degree of peripheral arterial oxygen unsaturation. Evidence pertaining to the possibility that a euonchoid habitus is present in these patients was equivocal.—J. B. S.

HEMOLYTIC SUSCEPTIBILITY DUE TO G-6-PD DEFICIENCY OF ERYTHROCYTES AMONG ASHKENAZIC JEWS. Ch. Sheba, A. Sheinberg, B. Ramoth, A. Adam and I. Ashkenazi. From the Tel-Hashomer Hospital, Israel. Harefuah, Vol. LX, No. 12, June 15, 1961.

The frequency of G-6-PD deficiency among Ashkenazic Jews has been reinvestigated, and 13 cases with the abnormality are presented. Three of them were discovered during a survey of 800 male blood bank donors, indicating a frequency of about 0.4 per cent.—B. R.


The authors call attention to slight hemolytic post-transfusion reactions without failure of renal function, and they report six cases of their own. Five of these showed neither shock nor a fall in blood pressure: in one patient brief unconsciousness occurred. In no patient was the hemolytic accident caused by a severe incompatibility in the ABO or Rh systems. In one case the cause was found to be a “dangerous universal donor”; in another the antibodies were from the recipient, but were not identified; in another patient disintegration of homologous blood cells occurred during paroxysmal nocturnal hemoglobinuria; and in three patients the cause of the hemolytic reaction could not be elucidated.—L. D.


A new notation for the Kell blood group system is based upon the sequential numbering of the available kinds of antisera: Anti-Kell = anti-K1; anti-Cellano = anti-K2; anti-Penney = anti-K3; anti-Raunenberg = anti-K4; anti-Peltz = anti-K5. This permits phenotypes and complex alleles to be coded in the exact terms of the results observed with those kinds of antisera that have been employed. An example is revealed in the following abstract.—R. E. R.


The serum of a woman having the phenotype K1:-1,2,-3,-4, was found to agglutinate all test
cells except others of the same phenotype. Antibodies were successively absorbed and recovered in eluates from K:1,2,3,-4 cells, K:1,-2,3,4 cells, and finally from K:1,2,3,4 cells. The final eluate agglutinated 525 random test cells but not cells of type K:1,2,3,4. The new antibody was termed anti-Ku (and thus becomes anti-K5). Complex cross-reactions could not be excluded to account for the K5 serologic specificity. A new Kell phenotype, K:1,w2,-,w4,-,5, is also reported.

—R. E. R.

THE FIRST EXAMPLE OF THE RH PHENOTYPE R0
R0, P. Levine, R. E. Rosenfield and J. White.

A blood specimen found to be R0 (G) positive, was also found to be negative for Rh0 (D), rh (C), rh" (E), and hr (c). A family study was strongly suggestive that the propositus was homozygous for the r0 allele. A complete typing study revealed that the propositus was positive for hr" (e), although not for hr*, for both Hr and Hr, as well as for rh" (C). These red cells were found to lack additionally hr (f), rh1 (Ce), hr' (V, ce*), rh*1 (C*), rh+ (C), and hr*. Of particular interest was the rh' (C) status: a series of anti-rh' (C) reagents, all lacking anti-Rh0 (D), reacted weakly, and failed to lose titer when absorbed with the rh0 erythrocytes; one serum, however, yielded an eluate from these cells, and this was termed anti-C0 to distinguish the specificity from that expected of anti-rh' (C).—R. E. R.


An example of reagent rabbit anti-M serum was found to cross-react with Henshaw (He) positive type N red cells despite the fact that the rabbit had not received He positive erythrocytes. Eluates from He positive type N cells yielded agglutinins with M specificity which, although insensitive to absorption with He negative type N cells, could be absorbed readily by He positive type N cells as well as by He negative type M or MN cells. Most of the anti-M in this reagent was found to cross-react with the He antigenic determinant, and this specificity was termed anti-M* to distinguish it from usual anti-M which does not cross-react with He. Failure to consider such complex immune responses could lead to errors in MN typing and false exclusions of maternity in medicolegal cases.—R. E. R.


An interesting genetic analysis of the quantitative precipitin reactions obtained with chicken “anti-O” and the saliva of members of 13 families. The chickens had been immunized to type O pseudomucinous cyst fluid. Type AB saliva precipitated insignificantly, and three grades of secretors appeared to depend upon three differing O alleles.—R. E. R.


DEAE column chromatography adapted to small (1 ml.) volumes of serum permitted the elution of 7S γ2 globulins with 0.02M phosphate buffer, pH 6.3, and the recovery of other serum proteins in 1 M NaCl eluates. Each fraction was then studied by acacia technic for anti-A and anti-B agglutinating activity. Paired specimens of maternal and cord sera revealed identical amounts of protein and identical titers of ABO isoagglutinins in the 7S γ2 globulin containing fractions. Whereas maternal serum isoagglutinins were encountered chiefly in the macroglobulin-containing 1 M NaCl eluates, cord serum isoagglutinins were found only in 7S γ2 globulin fractions. Of 110 prenatal sera, 28 type A and 9 type B mothers lacked 7S γ2 globulin ABO isoagglutinins. On the other hand, 53 of 73 type O mothers revealed 7S γ2 globulin ABO isoagglutinins. On the other hand, 53 of 73 type O mothers revealed 7S γ2 globulin ABO isoagglutinins and a higher frequency of anti-A than of anti-B. When the 53 examples of 7S γ2 globulin ABO isoagglutinins were tested for resistance to inhibition by hog A and horse B specific soluble blood group substances (SSBGs), only five were found to be non-inhibitable with concentrations of SSBGs approximately equivalent to average secretor saliva. Four of these mothers subsequently delivered type O newborn. but one delivered a type B child with erythroblastosis requiring exchange transfusion therapy. No other newborn in the small series was affected. Six other women with histories of having had children with characteristic ABO disease, and five donors stimulated with similar
LEUKOCYTES


A 13-year-old girl with Chediak-Higashi syndrome showing giant granulations of the white blood cells, albinism and frequent infections is reported. The leukocytic anomaly has been observed for two years. Leukopenia with 60 per cent lymphocytes was always present. Slight enlargement of the submaxillar nodes, hemorrhagic diathesis and psychomotor troubles were also noted.

—E. K.


A case of acute leukemia with atypical chromosomal constitution is described. Examination of 200 cells from the bone marrow gave chromosome counts varying between 45 and 54, with a modal number of 53. The chromosomes showed no structural modifications. The excess chromosomes, which varied in number from cell to cell, belonged to the different recognized groups (large, medium, small). Polyploid cells were also seen in which the number of chromosomes was not a multiple of the normal haploid cell. The mechanism giving rise to this anomaly and its possible role in the pathogenesis of the disease is discussed.—M. J.


Icterus was noted in 16 of 58 cases of acute leukemia and four cases of chronic leukemia in the acute stage. The icterus appeared usually several days before death and was accompanied by high fever which commonly began a few days earlier. Clinical-laboratory, epidemiologic and postmortem studies of all cases showed that the cause of the icterus was not uniform, but very often complex. Probably the most important cause was sepsis (5), marked leukemic infiltration of the liver with intrahepatal mechanical obstruction (2). Posthepatic residua were found in two cases and tuberculous dissemination in the liver also in two cases. In only one case was chemotherapy (6-mercaptopurine or possibly 6-azauracil) suspected as a toxic agent for the liver, and once the cause was homologous serum hepatitis.—L. D.
ABSTRACTS


The incidence of the factors Gm\(^a\), Gm\(^b\) and Gm\(^c\) have been studied in 79 patients with multiple myeloma and 705 normal subjects. In subjects showing hypo-\(\gamma\) globulinemia by electrophoresis, five types of Gm \((a,x,b)\) were shown to be present.—G. M.

HEMOSTASIS

FORMATION OF THROMBIN. H. Kowarzyk and E. Marciniak. From the Institute of Immunology and Experimental Therapy, Wroclaw, Poland. Polski, Tygodnik lek. 16:1,847, 1961.

Prothrombin derivatives are present in citrate thrombin preparations. These derivatives added to Ca\(^++\) and cephalin induce and accelerate the formation of thrombin in normal and hemophilic A and B plasma as well as in the plasma of hen or carp. A quantitative study of the preparations of citrate thrombin and of biothrombin suggests a new prothrombin derivative called autoprothrombin C. Unlike autoprothrombin I and autoprothrombin II of Seegers, autoprothrombin C with Ca\(^++\) and cephalin elicits prothrombin consumption in hemophilic A and B plasmas. It normalizes the prothrombin consumption in the plasma of even the most severe hemophilia A cases. It was shown that autoprothrombin C acts only when factor V is present. Autoprophrothrombin C activity appears and increases successively during the conversion of prothrombin to thrombin in sodium citrate solution, as well as during biothrombin formation in human plasma. The existence of autoprophrothrombin C strongly supports the concept that thrombin formation is a chain reaction.—E. K.


A method of isolation and purification of the thermostable fraction, FDG, from a fibrinogen lysate is described. FDG possesses only one N-terminal group and this together with the results of the electrophoretic and sedimentation analysis \((S_{20} = 2.8 S)\) indicate the homogeneity of this preparation. Further analysis of FDG revealed a high content of hexoses and hexosamines.—E. K.


The effect of a macroglobulin has been studied on the concentration of prothrombin and proconvertin in a normal plasma. The macroglobulin was found in association with a reticulum cell sarcoma in a patient with a persistent, well-marked prolongation of the Quick test. The latter was defined as due to a deficiency of true prothrombin and of proconvertin. After addition of different dilutions of the macroglobulin to normal plasma, the prothrombin and proconvertin times were greatly prolonged. This suggested an inhibiting effect of the macroglobulin on the plasma activators. This effect may explain some clotting defects in the dysproteinemias.—G. M.


(1) The incidence of hemophilic syndromes in Japan is 0.8 per 100,000 males and in some localized areas it ranges from 1.2 to 2.6 per 100,000 males. The relative incidence of hemophilia and PTC deficiency is 4:1. (2) The clinical and hemostatic findings of eight cases in five families with a syndrome of AHF deficiency and hemophilia and von Willebrand's disease are presented. In agreement with Nilsson's opinion, it is confirmed that there is a lack of plasma factor, i.e. anti-bleeding factor. (3) Nishimine factor deficiency combined with thrombopathy is described. Nishimine factor, which is considered to involve thromboplastin formation, was located at the \(\gamma\) globulin area while AHF migrated with \(\alpha\)-globulins when starch zone electrophoretic studies were carried out on normal BaSO\(_4\) plasma. Separation of Nishimine factor from fibrinogen and AHF was accomplished with DEAE-cellulose column chromatography. Nishimine factor was separated by these methods from fibrinogenemic plasma or hemophilic plasma. (4) Tatsumi factor deficiency combined with thrombopathy is described. Tatsumi factor, which is considered to participate in thromboplastin formation, was located at the \(\beta\)-globulin area and separated from PTC and factor X by starch zone electrophoretic of normal serum. Separation of Tatsumi factor from PTC and factor X was demonstrated with DEAE-cellulosc column chromatography. Tatsumi factor was prepared by these methods from PTC-deficient serum. The patient's prolonged bleeding time seems to be due to a
deficiency of platelet anti-bleeding factor. (5) Thus, the following combined defects: AHF deficiency and prolonged bleeding time; Nishimine factor deficiency combined with thrombopathy; and Tatsumi factor deficiency combined with thrombopathy.—K. F.


A group of newborn infants was examined for the presence of petechiae during the first day of life. Petechiae were found in one or more regions of the body in 44 per cent of the 250 infants studied. They were more commonly seen in boys and there was a positive correlation between the presence of petechiae and increasing weight, height or head circumference. The petechiae were usually limited to the part of the body which leads the descent through the birth canal, and they were thought to be due to a local or regional increase in venous pressure as a result of temporary compression of superficial veins.—J. B. S.


In a group of 45 children with anaphylactoid (Henoch-Schönlein) purpura, 36 per cent showed evidence of present or recent streptococcal infection. This is not a very different incidence from that found in children hospitalized for nonstreptococcal illness, and is a much lower incidence than is found in children with acute rheumatic fever or glomerulonephritis. Skin biopsy specimens from 12 children showed perivascular infiltration of the dermis. Leukocyte-platelet thrombi were observed in many small blood vessels. Necrosis of blood vessel walls was seen in some areas of marked inflammatory response. Erythrocyte extravasation was always associated with perivascular infiltration and there was a rough correlation between the severity of the inflammatory reaction and the degree of red cell extravasation. Muscle biopsy specimens were essentially normal. Kidney biopsy specimens from 11 children revealed a variety of abnormalities, but with certain fairly characteristic changes. The most common microscopic change consisted of focal areas of glomerular endothelial hypercellularity associated with fibrinoid deposition. Normal glomeruli were seen immedi-ately adjacent to glomeruli showing typical focal lesions. No abnormalities of blood vessels other than the glomerular capillaries were observed. The changes in the renal tubules consisted of mild to moderate atrophy, cloudy swelling and tubular casts.—J. B. S.


Three fatal cases of heatstroke with afibrinogenemia and multiple hemorrhages are described. In one case marked fibrinolytic activity could be demonstrated. The possible role of fibrinolysis and afibrinogenemia in the clinical picture of heatstroke are briefly discussed.—B. R.

PSEAS MUSCLE HEMORRHAGE AS A COMPLICATION OF ANTICOAGULANT THERAPY. H. Strašilová, Z. Matúška and J. Vícký. From the Second Medical Department, Hospital Bulovka, Prague, Czechoslovakia. Vnit. lék. 7:1131-1133, 1961.

Hemorrhage into the psoas muscle affecting four patients is described. One of them had the typical clinical signs of psoas muscle involvement of different origin ("psoas syndrome"). The tendency to hemorrhage in these patients was due to atherosclerosis, and in three of them also due to anticoagulant therapy. A probable precipitating factor was trauma arising from psoas strain when the bedridden patients attempted to sit up.—L. D.

MISCELLANEOUS


Sera of 508 patients and 168 normal subjects were studied. Among 329 patients who had received at least one blood transfusion, leukoagglutinins were found in 26 thromboagglutinins in 17 and hemoagglutinins in 4 per cent. Thromboagglutinins appear mostly together with leukoagglutinins. Hemoagglutinins were found only in sera containing both leuko- and thromboagglutinins. Isoagglutinins were found frequently in women, their appearance being related to pregnancy. Leukoagglutinins were found more often in patients with leukopenia than in patients with
ABSTRACTS

a normal number of white blood cells. No such relation was noted between the number of blood platelets and thromboagglutinins. Pyrogenic reactions after blood transfusion occurred more often in patients with leuko- and thromboagglutinins than in persons in whom isoagglutinins were not found.—E. K.


Exchange transfusion was performed in rabbits to assess the electrocardiographic and clinical changes associated with the use of citrated versus heparinized blood. The effects of raising the potassium content and lowering the temperature of the donor blood were also observed. At normal potassium levels there was no apparent difference, but at potassium levels of 25 mEq per liter, cardiac arrhythmias, ventricular fibrillation, and death ensued in almost all the recipients of citrated blood. These effects were rarely seen in the rabbits transfused with heparinized blood. Although the incidence of death was unchanged, the rabbits had a greater tolerance for hyperpotassic citrated blood at 1 C. than at 20 C. Intermittent injections of calcium heptogluconate also increased the tolerance to hyperkalemic citrated blood, but the final outcome was not significantly affected.—J. B. S.


The red and white cell count and the differential blood count of old men correspond to normal values for the adult population. With advancing age, no significant changes of these values were found. Neither physical training carried out throughout life nor a sedentary life influenced the values of the blood count. A difference was found only in hematocrit values following stress after 5, 15 and 45 minutes. An increase of the hematocrit during the 5th minute and a decrease during the 45th minute were more marked in those men who were engaged in physical training during their whole life.—L. D.


Proflavine (3,6-di-arnino-acridine) migrates with the albumin fraction of blood serum, as shown by cellulose acetate electrophoresis.—E. K.


The method described for isolating human y-globulins is composed of three steps: (1) obtaining a protein preparation with globulins concentrated by zinc fractionation, (2) removal of contaminating proteins by aluminum fractionation, (3) final concentration of y-globulins by zinc fractionation. The four following parameters were varied independently for studying fractionation by means of zinc: zinc concentration, protein concentration, pH and ionic strength. Aluminum concentration and pH were varied independently for studying fractionation by means of aluminum. A procedure for y-globulin isolation is suggested.—E. K.


A study of 287 sera from Japanese subjects in Tokyo showed that the Gma factor was present in all sera. Twenty per cent of them were Gm (b+), 30 per cent Gm (x+). Among that population, the Gm-like factor was absent and the Inv factor present in 48 per cent. For the seroanthropologist, most interesting is the fact that when the frequencies are compared, all the Negroes appear to be Gm(a+, b+, x–), whereas the Chinese and Australians are Gm(a+, b+). The Gm-like factor present in Negroes is absent in the Caucasian populations as in the Japanese. The present study confirms Steinberg's hypothesis that three genes, Gm+ Gm* and Gm* control the presence of these factors. The Inv factor is independent of the Gm factors.—G. M.


The mean properdin value found in 103 volunteer blood donors, the male figure of 7.3 and
female of 8.1 properdin units per ml. serum, was in complete agreement with the average properdin level in the serum of healthy subjects (4-10.0 properdin units/ml. serum). The normal properdin levels were found in nearly 50 per cent of the sera from the blood donors investigated; the remaining were divided by almost equally between properdin titers below and above the stated normal averages. It may be concluded from the results obtained that properdin levels in the blood need not be influenced by age or sex. Properdin levels failed to show any correlation with blood groups O and A (both Rh+) in either men or women.—L. D.

ERRATUM

In the list of authors’ affiliations following “Erythroblastopenia (Pure Red Cell Aplasia) in Childhood in Djakarta,” which appeared in the February BLOOD (Vol. XIX, No. 2, pp. 168-180), it was erroneously stated that Dr. S. Thajeb was formerly Director of the Army Blood Center in Djakarta. We have been informed that Dr. Thajeb still holds that position.