ERYTHROCYTES

THE SILENT CARRIER OF BETA THALASSEMIA.


A mild form of beta thalassemia major is described in which two siblings, of Albanian descent, had hepatosplenomegaly and persistent anemia (Hb between six and eight Gm. per 100 ml.) with typical morphological changes of the erythrocytes. Neither had ever required transfusions and the older girl, age 12, was in the 75th percentile for height and weight and in the early stages of pubertal development. The mother showed changes typical of high A2-beta thalassemia with classic alterations of red cell morphology, decreased red cell osmotic fragility, and an elevated HbA9 (5.4 per cent). The father, on the other hand, showed no such characteristic changes, either in red cell morphology, osmotic fragility or in the proportions of the various hemoglobins present in the peripheral blood. Incubation studies using the peripheral blood from various members of this family revealed a moderately severe depression in the relative rate of beta chain synthesis in both of the siblings. The presence of a gene for beta thalassemia in the father was documented by finding a moderate, but significant, depression in the relative rate of beta chain synthesis. Because of the depression in the relative rate of beta chain synthesis associated with normal hematologic indices (and normal values for HbA9 and F) the author has chosen to call this the "silent carrier of beta thalassemia" in analogy to a similar situation in the a-thalassemia syndromes. 

Abstracter's comment: This family is of considerable interest in that these findings may provide an explanation for a small but puzzling group of patients presenting with "thalassemia intermedia." In addition, this study points once again to the heterogeneity of the thalassemia syndromes, an observation that is of extreme interest not only to the in-
vestigator studying the pathophysiology of the disease, but also to the clinician who must advise on the prognosis and management of these patients.—T. F. N.


A hitherto undescribed hemoglobin variant was identified in a Japanese male and several members of his family. Erythrocyte morphology was normal and no illness was associated with the presence of this variant. It moved slightly more rapidly than HbA on starch gel at pH 8.6. Hemoglobin Yoshizuka is of interest in that at a given pO2, the per cent oxygen saturation is significantly decreased when compared to that of HbA. The side chain of the β G10 residue is thought to interact by hydrogen bonding with the corresponding histidine residue on the alpha chain. Interference with this α-β interaction may be associated with decreased oxygen affinity but normal heme-heme interaction.—T. F. N.


Four cases of fulminant pneumococcal infection are described in children with sickle cell hemoglobinopathy. All children died within several hours from the onset of symptoms and, in addition, two had laboratory evidence suggestive of disseminated intravascular coagulation. Three of the patients had splenomegaly at autopsy and, thus, the fulminant course of the disease could not be attributed to autosplenectomy. The authors suggest that reticuloendothelial blockade by engulfed sickle erythrocytes, as well as hemosiderin, might produce functional disability of the spleen, as well as contribute to the development of intravascular coagulation as seen in the generalized Shwartzman reaction in experimental animals. A retrospec-

ive study by the authors suggests that pneumococcal meningitis is 20 times as frequent in patients with sickle cell disease than in children without hemoglobinopathies. This association does not appear to be limited to sickle cell disease per se but may also be extended to Hb SC disease and sickle cell-thalassemia. Because of the rapid course of the disease, early treatment of acute febrile illnesses with penicillin in these patients is advocated. In addition, the authors suggest that heparin may be useful in selected patients. Abstractor's comment: Although not a new observation, this association is worthy of being re-emphasized, especially to house officers responsible for emergency-room care in hospitals with a large Negro population. The lack of prompt treatment of such patients may lead to tragic results and even a seemingly mild infection such as a sinusitis should immediately be treated with antibiotics until the results of bacterial cultures have been determined.—T. F. N.


Iron dextran was given intravenously to 30 patients with iron deficiency anemia. No side effects were observed in the dose range from 1 to 3 Gm. as a single infusion. The hematological response was evaluated in terms of estimates of the reticulocyte maturation time. It was found that the reticulocyte maturation time, normally 1.4 days, was increased to 2.8 days in the recovery phase of iron deficiency. This prolonged maturation should be accounted for, if the activity of the bone marrow is assessed by means of reticulocyte data. In addition, the microcyte survival time was evaluated by means of electronically plotted volume distribution curves, and a T/2 of 50 days was found. Abstractor's comment: The data illustrate that highly useful information can be obtained from a simple clinical model and by careful clinical observation alone. Nevertheless, some of the calculations tend to oversimplify the complexity of the problems involved and are in want of further confirmation.—H. J. H.
ABSTRACTS

COMPARATIVE STUDIES ON erythrocyte hemolysates, HbA1 and A9 were of hemoglobins in the cord blood did not from the corresponding zones were estimated by sedimentation and diffusion constant, and greater than one in every sample. However, the pattern on disc-electrophoresis of hemoglobins in the cord blood did not show such a separation of HbA components and it is presumed that A1 and A2 in the cord blood are identical in nature with A1" and it is presumed that A1 and A2 in the adult blood, respectively. A possibility that A1 (a9 Test) to be about 65,000 for each. There-fore, such a possibility can be neglected. —K. F.

CLINICAL STUDIES ON FIVE PATIENTS WITH MYELOFIBROSIS, WITH SPECIAL REFERENCE TO REDUCED GLUTATHIONE OF ERYTHROCYTES. M. Matsukawa and T. Ishida. From Niigata Univ. School of Medicine, Niigata, Japan. J. Clin. Hemat. 11:30, 1970.

Reduced glutathione (GSH) of erythrocytes was increased in five cases. The elevated level of erythrocyte GSH was assumed to result from an increased synthesis of GSH in erythrocytes of extramedullary origin, and it is suggested that the determination of erythrocyte GSH and leukocyte alkaline phosphatase would be useful in differentiating myelofibrosis from chronic myelogenous leukemia.—K. F.


Two further hitherto unreported hemoglobin variants have been identified among approximately 100,000 apparently normal Chinese residents of Taiwan. Although detailed studies were not carried out, neither variant appeared to be associated with obvious signs of anemia.—T. F. N.


An interesting hemoglobin variant is described in a Negro family. Members heterozygous for this hemoglobinopathy were clinically asymptomatic and showed no signs of hematologic abnormalities. This hemoglobinopathy could be recognized by the presence of two (rather than one) new bands (between HbA and S) on starch gel electrophoresis at pH 9.0. Extensive investigation revealed that the “extra” band was due to the formation of asymmetrical hybrids of the $\alpha A_2 R X$ type. Such a molecule appears to be unique among the hemoglobin variants thus far characterized and may shed additional light on alpha-beta chain interaction in the hemoglobin molecule. Structural analysis revealed that Hb Richmond is characterized by replacement of the asparagine residue at position $\beta G 4(102)$ by a lysine residue.—T. F. N.

LEUKOCYTES


RF mice, each bearing a transplanted myeloid leukemia, and A mice, each with a transplanted lymphoid leukemia, were irrad-
ated with a sublethal dose of X rays and immediately thereafter received injections of allogeneic spleen and bone marrow cells obtained from C57BL mice; control mice received syngeneic cells after irradiation. On days 1, 2, 3, 4, 5, 6, and 7 following this treatment, groups of mice were splenectomized and the minced spleens were transferred into syngeneic recipients. Each spleen was individually processed, i.e., each splenectomized animal provided spleen for a single recipient. The incidence of leukemia in both the spleen donors and recipients was followed. Spleens obtained 4 or more days after irradiation and injection of allogeneic cells failed to transfer leukemia, and donors of these spleens died with graft-versus-host reaction rather than with leukemia. The possible mechanisms involved in causing disappearance of leukemic cells are discussed and the early graft-versus-host reaction by injected allogeneic cells is considered to be the most likely explanation.—G. M.


Mice of the AkR strain are generally thought to be tolerant to the antigens of the murine spontaneous leukemias. Such a concept is essentially based upon the fact that Gross virus is vertically transmitted in mice belonging to this strain. We have experimentally tested this theory. The survival time of nonleukemic, 2-month-old, AkR mice receiving $10^2$ to $10^7$ K36 cells intraperitoneally is inversely proportional to the number of injected cells, and animals may escape death from leukemia more frequently when the number of tumor cells is smaller. Pretreatment of the animals by BCG delays and reduces the mortality of normal, 2-month-old, AkR mice receiving $10^5$ K36 cells intraperitoneally. Furthermore, immunization of 2-month-old AkR mice with E G2 cells (a leukemia induced in C57Bl/6 mice by Gross virus) reduces and delays the mortality produced in these mice by grafting $10^5$ K36 cells. Serum from nonleukemic, 2-month-old, AkR mice treated by BCG and E G2 cells or by K36 cells contains antibodies active in immune adherence against K36 cells. Moreover, it is possible to obtain in normal 2 or 5-month-old AkR mice, the formation of antibodies which are cytotoxic for cells from a leukemia induced in C3H mice by Passage A (Gross) virus and for cells from a spontaneous AkR leukemia, following a prolonged immunization with syngeneic K36 cells. These direct serological arguments, as the indirect arguments previously mentioned, lead us to object to the idea which has become traditional, that AkR mice are tolerant to cells from leukemias induced by Gross virus.—G. M.


Various erythrocytic abnormalities were investigated in 94 cases of leukemia: acute lymphoblastic, myeloblastic and undifferentiated leukemia, myelomonocytic leukemia, chronic lymphocytic and myelocytic leukemia, and also in polycythemia vera. The parameters studied were: in bone marrow: sideroblastosis and PAS staining in the erythroblasts; in blood: erythrocytes carrying Hb F, G-6-PD, acetylcholinesterase, lactic dehydrogenase and its isoenzymes. Hypersideroblastosis, PAS+ erythroblasts, Hb F erythrocytes, increased activity of G-6-PD and of the isoenzyme 5 of the LDH were found to be frequent in leukemia. These abnormalities were most frequent in acute leukemia. Myeloblastic and especially myelomonocytic leukemia were particularly abnormal; this last type seems similar to a "refractory anemia with partial myeloblastosis." The search for correlations between these various abnormalities is relatively disappointing; this fact is an argument for both random disturbance and multiple populations.—G. M.

LEUKEMIA IN INFANCY. H. J. Plüss. From the Universitätskinderklinik, Zürich, Swit-
Six cases of infantile leukemia were studied. Three cases showed initially the morphological, though not the histochemical, characteristics of acute lymphoblastic leukemia, but they all converted soon to monocytoid forms; there were two non-differentiated blast cell leukemias, two morphologically and cytochemically typical monocytic leukemias and one infantile chronic leukemia. The mean survival was 13 months. The data suggest that leukemia of the first and part of the second year of life is quite different from acute lymphoblastic childhood leukemia as far as the prevalence of atypical cells and the poor prognosis are concerned.—H. J. H.


Localized leukocyte mobilization was studied in 52 healthy volunteers and in 64 patients with acute and chronic leukemias, using a plastic skin chamber device. In contrast to the skin window method described by Rebuck, the exudates observed with the chamber method remained predominately granulocytic for at least 24 hours. Leukocyte mobilization averaged 68,400 x 10^6 leukocytes/sq. cm./24 hours. It was greatly reduced in acute leukemias, but acute myelogenous leukemias in complete remission showed normal results. Acute leukemias with normal peripheral granulocyte counts had, nevertheless, low mobilization values. This finding suggests that these cells, although morphologically normal, behave abnormally as far as their entrance into tissues and exsudates is concerned. Chronic myelogenous leukemias showed normal mobilization values, but the figures were low in chronic lymphocytic leukemias.—H. J. H.


The hypofibrinogenemia which is often seen in acute promyelocytic leukemia is not well understood. The authors performed coagulation studies in four such patients. In one of the patients, treatment with heparin resulted in increased fibrinogen values and widespread thrombi were seen in post-mortem examination. In another patient, active fibrinolysis was present and no post-mortem thrombi were seen. The authors concluded that both fibrinolysis and intravascular coagulation may occur in patients with this disease.—H. J. W.


In this survey of the literature and review of 10 patients with macrofolicular lymphoblastoma, problems of diagnosis, classification and treatment of this disease are discussed. Macrofolicular lymphoblastoma is a form of reticuloblastosis with a distinct benign phase lasting from several months to many years; in the early stage it may be impossible to differentiate it from reactive lymphadenitis. Finally, the disease becomes reticulosarcoma but it is doubtful that it ever is transformed into Hodgkin's disease.—J. V.


In the study of 24 patients with various proliferative diseases of the reticuloendothelial system, the daily urinary excretion of uric acid, purines, and their methyl deriva-
ties was measured together with serum Vitamin B<sub>12</sub> levels. Patients with increases in serum cyanocobalamin levels also showed increased excretion of uric acid, purines, and derivatives.—J. V.

**HEMOSTASIS**


The authors described a method for detecting fibrinogen and fibrin degradation products (FDP) by their ability to clump certain strains of coagulase-negative *Staphylococcus aureus*. The method detected concentrations of fibrinogen as low as 0.03 µg. per ml. (slide test) or 0.38 µg. per ml. (tube test). Early FDP (fractions X and Y) gave positive clumping reactions, whereas no clumping was obtained with late FDP (fractions D and E). Clumping was also obtained with soluble complexes composed of fibrin monomers and FDP. Increased clumping titer was obtained in patients with cirrhosis and in those treated with streptokinase, and a positive correlation was found between the staphylococcal clumping titer and the plasma thrombin time.—H. J. W.


The authors studied the possibility that homotransplantation of a normal spleen might permanently increase the factor VIII levels in hemophilic dogs. Preliminary studies demonstrated the necessity for administering cryoprecipitate during the operation to insure survival of the animals. In the critical experiments, a splenic homotransplant was performed on two hemophilic male dogs. Postoperatively, the animals received azathioprine and methylprednisolone. Factor VIII was assayed using hemophilic dog plasma as substrate and results were
expressed as per cent of normal dog values. Despite evidence of continuing function in the transplanted spleens, factor VIII activity remained undetectable for 2 weeks after splenic transplantation. The dogs’ "native" spleens were removed at 2 weeks and immunosuppressive drugs were discontinued 5 weeks after splenic transplantation. Eight months after transplants, no factor VIII was detectable in their plasmas. The studies do not support the conclusions of other studies which suggested that the spleen is the primary site of factor VIII synthesis. The authors urged that this site be further identified before the hypothesis of organ homotransplantation as a 'cure' for hemophilia is further pursued.—H. J. W.


Artificial heart ventricles were implanted into dogs and calves and the blood coagulation system studied in a series of 83 experiments. The materials used for the prostheses and the formation of thrombi upon their surfaces is discussed. The authors conclude in the presence of a cardiac prosthesis there develops a moderate thrombocytopenia and fibrinopenemia without any marked coagulation changes whether or not anticoagulants are used concurrently. Heparin administration reduces the incidence and extent of thrombus formation on the prosthesis but does not prevent it. Graphite-benzalkonium-heparin complex possesses marked anti-thrombogenic properties compared with other materials used in the manufacture of prosthesis.—F. V.


The effect of plasmin on human platelets was studied in vitro. The results obtained were as follows: (1) The anti-plasmin activity of platelets seemed to function by a biological mechanism which can be demonstrated by the diminished activity of platelets after disruption in a glass homogenizer. (2) Platelet-rich plasma treated with high doses of plasmin showed an extremely reduced ma in the TEG, whereas platelets treated with low doses of plasmin did not indicate any changes in the TEG. (3) Platelet-rich plasma homogenized with a glass homogenizer caused a marked decrease of ma in the TEG when compared with intact platelet-rich plasma. (4) It might be assumed that plasmin could directly affect platelet aggregation by ADP. However, both streptokinase and FDP did not show any influence on the aggregation of platelets with ADP. (5) Platelets incubated with plasmin had reduced activity of platelet factor 1. This effect of plasmin on platelets could not be prevented by the presence of AMCHA in high doses.—K. F.


Normal human blood platelets were fixed under possible native conditions with glutaraldehyde and osmic acid. Serial ultrathin sections about 800 Å thick were prepared and studied by electron microscopy to elucidate the three-dimensional ultrastructure of platelets. Conclusions obtained from 10 completely reconstructed platelets and many partially reconstructed platelets were as follows: (1) The ordinary shape of normal human platelets was discoid, but it was sometimes nearly spherical. (2) Microtubules made a circular running at the rim of the disc-shaped platelets, but they ran circularly around the equator in spherical platelets. (3) Each of the 10 reconstructed platelets had 50 to 150 spherical α-granules, a few rod-shaped γ-granules, and one to 11 drum-stick shaped granules. (4) One platelet had seven to 33 mitochondria. (5) The blood platelets had many vesicles resembling the smooth-surfaced endoplasmic reticulum. A three-dimensional reconstruction revealed that vesicles were connected with each other, forming a tubular system with irregular course and ramifications. The
tubules had some openings on the cell surface, and were connected with a vacuole limited by a single unit membrane. (6) The formation of blood platelets as a result of the maturation process of megakaryocytes is discussed with respect to formation of the α-granules, formation of the demarcation membrane with liberation of platelets, formation of centrioles and the possible relationship between spindle fibers and microtubules.—K. F.


The surface structure of human blood platelets and the alterations produced by thrombin were studied by scanning electron microscopy. Platelets collected by careful centrifugation partly showed a few long pseudopods projecting from the margins of the platelets and interpreted as being elongations of the spiny projections. About 78 per cent of the collected platelets were discoid in form, 12.5 per cent spheroidal, 5.5 per cent spherical and 4 per cent irregular. The addition of thrombin to citrated platelet-rich plasma caused the following changes: At first, small protrusions appeared on the entire surface of the platelets except on their margins. The discoid platelets, then, transformed into spheres covered by grown and rounded protrusions and became aggregated, while the formation of pseudopods progressed. Fibrin strands about 0.1 μ in thickness appeared to form networks into which the aggregated platelets were trapped more and more firmly.—K. F.


The test involves the use of citrated platelet-rich plasma (P.R.P.) obtained by spontaneous sedimentation of blood at 5°C for 120–240 minutes. Then, 1 ml of P.R.P. is placed in a siliconized glass tube, sealed with Parafilm and rotated at 16 rpm for 2 minutes. The tube is left vertical and undisturbed for 20 minutes, then 0.1 ml. of the top layer of plasma is diluted with 1.9 ml. of 3.8 per cent sodium citrate, and a platelet count is performed by phase-contrast microscopy or by an automatic counter. Results are expressed by the per cent difference between the platelet counts obtained before and after rotation. Results in 30 normal subjects were 25–78 per cent. Normal values were obtained during anticoagulant treatment with Dicumarol, in hemophilia, factor VII deficiency, acquired anticoagulant, hyperlipidemia and in diabetes. Increased values were obtained in artherosclerosis, stress, and during heparin treatment. Decreased values were obtained in hypofibrinogenemia, diffuse intravascular coagulation, Glanzmann’s disease, renal failure, thrombocytopenias and with isolated prolonged bleeding time. Three patients with Von Willebrand’s disease gave normal results. Abstracter’s comment: The technique described herein may measure a different parameter than that measured by the usual platelet aggregation or adhesiveness tests. It is not known what is actually determined, but perhaps the changes induced in platelet shape and volume by chilling are in part responsible for the results, as has been described by Salzman et al., Amer. J. Physiol. 217:1330, 1969. It seems that the wide variation in the response of normal subjects is an important shortcoming. Also it is not possible to measure the release of platelet constituents at that temperature.—E. S.

Studies of Platelet Metabolism in Relation to Platelet Function. M. Saito and T. Karuayanagi. The 3rd Department of Internal Medicine, Faculty of Medicine, University of Tokyo, Tokyo, Japan. Acta Haemat. Jap. 32:23–30, 1969.

The significance of neuraminic acid, transport ATPase, Mg-ATPase and ATP metabolism was studied with respect to platelet adhesiveness and the release reaction. Platelet adhesiveness was determined by the modified method of Hellem. The adhesiveness index determined at 4°C was similar to that determined at 37°C suggesting that adhesion of platelets occurs independently of platelet metabolism. No change
in the content of ATP, ADP and AMP was demonstrated during platelet adhesion. Treatment of platelets with ouabain produced a marked decrease in the transport ATPase activity and in the adhesiveness index, suggesting a significant role of transport ATPase in the mechanism of platelet adhesion. Neuraminic acid content of non-adhesive platelets separated by the above mentioned glass bead method was much lower than that of total platelets (nonadhesive and adhesive platelets). Both adhesiveness index and neuraminic acid contents of platelets were markedly decreased in hemorrhagic patients with ITP, SLE, aplastic anemia, uremia and liver cirrhosis. A significant correlation between adhesiveness index and neuraminic acid content of platelets was demonstrated in these patients. Treatment of rabbit platelets with ouabain had no effect on the release of acid phosphatase, ADP and AMP during viscous metamorphosis. Treatment of rabbit platelets with N-ethyl maleimide resulted in marked inhibition of Mg-ATPase activity and of the release reaction. These results point to the significant role of Mg-ATPase for the release reaction of platelets.—K. F.

THROMBOPATHY IN MAY-HEGGLIN ANOMALY

E. A. Beck and H. R. Baumgartner.

Data on a new family with May-Hegglin anomaly (giant platelets, thrombocytopenia, basophilic inclusions in the cytoplasm of granulocytes) are presented. In contrast to earlier reported cases and to the Hegglin’s original case (which was reexamined), this family showed normal platelet function tests. The findings suggest that in the May-Hegglin anomaly, pathological platelet functions are not necessarily present.—H. J. H.


Thrombocytopenia associated with acute alcoholism has been described from several centers during the past five years. This has been unrelated to liver disease or vitamin deficiencies and platelet counts rapidly returned to normal after cessation of drinking. The authors administered a synthetic whiskey, containing 43 per cent ethanol, to three volunteers for 12 days. Two separate effects were observed. In all three subjects a transient decrease in the platelet count occurred 5 hours after the onset of drinking. A second, and more pronounced, thrombocytopenia occurred 1 week later. The authors suggest that the initial decrease in platelets may have been due to platelet sequestration. The later, gradual development of thrombocytopenia suggested a different mechanism, but the studies could not distinguish the mechanism of platelet destruction from that of bone marrow depression.—H. J. W.


30 patients with chronic posthemorrhagic anemia of from 1 to 16 years duration were given 125 transfusions (three to four to each patient) of thawed red cells. All transfusions were uneventful and numerous tests of blood coagulation function were made before transfusion, and at 2 hours, 24 hours, and 3–5 days after transfusion. No consistent changes in fibrinogen, Factor VIII or heparin were noted but there was, in the first 24 hours, a transient fall in proconvertin and an increase in fibrinolysis which subsequently returned to normal. These changes could be related to the release of factors from the destruction of transfused red cells. After the transfusions, (3–5 days), there was a consistent increase in the prothrombin index and proconvertin concentration, suggesting an improvement in hepatic function.—J. V.

IMMUNOHEMATOLOGY

MATERIAL MODIFICATION OF TISSUE ANTIGENICITY AND THE GRAFT-VERSUS-HOST
ABSTRACTS


Differences in time of onset and severity of a spontaneous graft-versus-host reaction were demonstrated in lethally irradiated reciprocal hybrid mice inoculated with marrow from parental strains. The preferential advantage observed as a less severe graft-versus-host reaction when the marrow donor strain was syngeneic with the maternal strain of the hybrid cross was interpreted as resulting from a modified expression of paternal antigens induced by the maternal environment during fetal development. This modified antigenicity affected not only recovery from the graft-versus-host reaction but apparently also influenced the ability to prevent the secondary disease syndrome.—G. M.


On isolated human monocytes distinct receptor sites for IgG (in particular of its antigenic subclasses IgG 1 and IgG 3) and for C'3 have been demonstrated in vitro by means of sensitized erythrocytes. There was no interaction between monocytes and IgM covered erythrocytes unless complement was added. Macrophages, isolated from liver and spleen showed similar reactions as did monocytes. Results with other serum proteins were consistently negative.—H. J. H.


Studies were done on the low titer of serum IgG in germfree rats. It was rare to find the typical complete form of the so-called germinal centers in the lymphoid tissue of germfree rats. However, by exposure of these rats to bacterial contamination, it was easy to induce typical germinal centers. The mass appearance of plasma cells in conventional rats occurred without direct relation to lymphoid nodules, namely at the junction where Peyer's patches are exposed to the intestinal lumen and at the medullary hilum of lymph nodes, while such sites in germfree rats revealed a simplified architecture consisting of a combination of reticuloendothelial cells and lymphoblast-like cells. However, according to the results of inoculation experiments with human gamma globulin using germfree animals, this simplified architecture seemed to maintain a high potential for inducing a dramatic production of plasma cells. In addition, demonstration with immunofluorescence indicated that these plasma cells contained antibodies corresponding to the injected antigen and most of the antibodies belonged to IgG.—K. F.


In a study of experimental burns in rats, the response of the animal to injections of paratyphoid vaccine was observed with and without the simultaneous administration of homologous serum. In the acute toxic stage of the burns, the immune response as judged by subsequent antibody titers and lymphoid tissue hyperplasia, appears to be depressed. The transfusion of serum immediately following the burn promotes, in great measure, a more normal immunologic response.—J. V.


The etiopathogenesis, the clinical and im-
munological characters of primary, chronic cold agglutinin disease were studied in one patient. Mercaptan reduction and immunoch­
emical examination of cold agglutinin eluate indicated that the cold agglutinins in the patient's serum belonged to the IgG class, consisted of light kappa-type chains and were characterized by anti-I blood group specificity.—S. R. H.


Quantitative determination of IgA, IgG and IgM in duodenumpancreatic fluid and bile were made using immuno­diffusion methods. IgA was observed in the highest concentrations (51.0 ± 16.0 mg. per cent); the concentration of IgM was 32.0 ± 9.0 mg. per cent, that of IgG 16.5 ± 11.0 mg. per cent. Presence of the transport piece was demonstrated as well and found to be associated with IgA globulin. Intestinal parasitosis was found in some cases to be associated with increased IgA levels, chronic pancreatitis showed an increase of IgM, and all immunoglobulins were raised in cases with acute and chronic hepatitis.—H.-J. H.

ABSTRACTS

MISCELLANEOUS


Detailed observations were made on 33 patients of which 13 were suffering from subacute and 20 from chronic hypoplastic anemia. The schemes of treatment included corticosteroids combined either with transfusions of ordinary cytrated blood or with those of noncitrated blood. The method of obtaining non citrated blood was based on the finding of calcium ions by cellulose adsorbants. Marked differences in the results were observed: In cases where transfusions of noncitrated blood were administered, the hemoglobin and erythrocyte values went up much faster (36 ± 4 days), than in those cases where transfusions of citrated blood had been resorted to (44.5 ± 7 days). In cases of chronic hypoplastic anemia, the effect of treatment was greater than in those with subacute hypoplastic anemia.—J. K.