ABSTRACTS
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ERYTHROCYTES


The production of erythropoietin (ESF) is controlled by the relative availability of oxygen to the tissues concerned with its synthesis. The kidney is the primary organ serving this function although other sites may also exist. It is known that the production of ESF involves the elaboration of a renal factor termed erythropoietin (REF) which by converting or activating an inactive normally circulating protein results in the formation of the ESF. The nature of this protein or proteins remains unknown. The plasma ESF activity, first evident 2 hr after REF injection was preceded by a significant rise in the concentration of the substrate for REF. Similar results were obtained when rats were subjected to hypoxia to stimulate the release of endogenous REF. Fluctuations in the level of circulating ESF and the substrate for REF suggest that the negative feedback action of the ESF on its own production is mediated through a decrease in the amount of available substrate. Hypoxia of only 1 hr duration was sufficient to evoke the reactions leading to the formation of the ESF. These events preceded independently of the hypoxic stimulus for at least 5 hr after its interruption and resulted in the production of significant quantities of ESF.—M.S.


Serial collections of plasma, bile, and urine were obtained from two infants with hyperbilirubinemia due to glucuronyl transferase deficiency who received continuous
transfusions of $^{14}$C-bilirubin while undergoing phototherapy. Analysis of these fluids provided evidence that light converts bilirubin in vivo to diazeneutral derivatives that are excreted fairly rapidly in bile and urine. These bilirubin derivatives qualitatively resemble those found in similar patients and in Gunn rats without treatment suggesting that light accelerates bilirubin breakdown by mechanisms similar to those normally operative in glucuronyl transferase deficiency.—J.B.S.


Intravenous infusions of large amounts of urea in invert sugar have recently been advocated as therapy for sickle cell crises. However, the blood concentrations achievable under clinical conditions are far below those at which urea is known to disrupt hydrophobic bonds. The authors therefore considered other possible actions of urea in the concentrations observed (200-400 mg/100 ml). Cyanate (-NCO) which is known to be present in urea solutions can react with the N-terminal residues of proteins to form carbamylate derivatives. This reaction is essentially irreversible. The authors show that low concentrations of cyanate inhibit sickling in vitro and that in contrast to the effects of high concentrations of urea, this effect is irreversible. The amount of bound cyanate required to inhibit sickling is in the range of 0.1–1 mole of cyanate per mole hemoglobin. Not only is the morphological phenomenon of sickling inhibited, but the gelling of hemoglobin S is also blocked. In their discussion, the authors point out that carbamylation of globin does not appear to affect the ability of the molecule to bind and release 0$_2$. Moreover, mice treated with sublethal doses of KCNO show no apparent ill effects while the hemoglobin isolated from such animals contains more than one carbamyl group per hemoglobin molecule (shown to be sufficient to inhibit sickling in vitro). Abstracter's comment: This is an exciting finding and deserves careful follow-up. Irreversible inhibition of sickling by an agent which is potentially tolerable in vivo would truly represent the ultimate in "molecular engineering" and could provide long-term relief presumably for all patients with this disease. A word of caution: a great deal of laboratory and clinical investigation must still be carried out before this can be considered ready for use in routine clinical situations.—T.N.


The authors reexamined a Chinese family in which the original propositus had what appeared to be classical hemoglobin-H disease. On careful reexamination however, a sister was found to have several minor hemoglobin components that could not be explained in terms of the current concepts of Hb-H disease and alpha thalassemia. These minor components had a mobility slower than Hb-A, H, or Barts, and on careful analysis were shown to contain abnormal $\varphi$-chains (similar perhaps to Hb-Thai). The authors discussed these findings in terms of the possibility of a triplicated $\varphi$-chain locus. However, in a note added in the proofs, the authors refer to a recent article by Milner et al. (Lancet 1:729, 1971), in which a similar Chinese patient with Hb-H disease was found to have a minor hemoglobin component which proved to be an elongated $\varphi$-chain consisting of 172 rather than 141 residues. Reevaluation of the minor components present in the patient studied in this report suggests that such an elongated chain may be present here also. Abstracter’s comment: The simplest explanation for the existence of such a component would not be a reduplicated $\varphi$-chain locus, but rather that the $\varphi$-chain, as originally synthesized, is significantly longer than that present in the completed hemoglobin molecule. These cases of $\varphi$-thalassemia may then represent a failure, at least in part, of the final stages of $\varphi$-chain “preparations,” with the release of abnormal alpha chains.—T.N.
Clinical Investigations on Renal Anemia. Ferrokinetic Aspects. H. Yoshimatsu. Department of Internal Medicine, Faculty of Medicine, Kyushu University, Fukuoka, Japan. J. Kyu. Hemat. Soc. 21:1, 1971.

Twenty-six patients with chronic glomerulonephritis were studied by the combined 59Fe and 51Cr methods. The patients were divided into two groups, the nonazotemic group (BUN below 25 mg/100 ml) and the azotemic group (BUN over 26 mg/100 ml). In the nonazotemic group, the plasma iron disappearance half-time (PIDT½) was 85.5 ± 28.7 min, the plasma iron turnover rate (PIT) 0.530 ± 0.252 mg/kg/day, the percent iron utilization by red cells (%RCUt) 93.4 ± 6.3%, the red cell iron turnover rate (RCIT) was 0.436 ± 0.192 mg/kg/day and the red cell half survival time (51Cr T½) was 24.3 ± 4.5 days. In the azotemic group, PIDT ½ was 103.9 ± 50.9 min; PIT, 0.541 ± 0.231 mg/kg/day; %RCUt, 71.3 ± 16.6%; RCIT, 0.384 ± 0.179 mg/kg/day; and 51Cr T½ was 20.2 ± 4.5 days. The difference between PIT and RCIT was 0.07 mg/kg/day in the nonazotemic group and 0.16 mg/kg/day in the azotemic group. The patterns of body surface counting were classified into four types. Type 1 was normal; Type 2 was characterized by normal to moderately decreased bone marrow uptake with increased liver and spleen uptake associated with prolonged plasma iron clearance seen in patients with anemia and renal insufficiency. In Type 3 the bone marrow uptake was low with increased liver and spleen uptake and rapid PIDT½ associated with the rapidly progressive type of chronic glomerulonephritis. Type 4 was similar to Type 2, but characterized by late rise of radioactivity in the liver and spleen and the shortening of 51Cr T½ seen in patients with anemia and renal insufficiency at the late stage.—K.F.


Red cell indices of 23 patients in whom osmotic diuretics were used during treatment for thermal burns showed that in those receiving urea, no morphologic changes were demonstrable, but in those receiving mannitol the mean diameter, volume, and surface area of the red cells were reduced, these changes persisting for up to 2 hr. The difference in effect between urea and mannitol is held to reflect the manner of distribution of these substances in the intracellular fluids and cells.—J.V.


A description of the authors' 17 cases and a review of published cases with this syndrome gives many clinical and laboratory details. It gives no new information but is a valuable source reference.—R.O.W.


A review of oxygen uptake and release by hemoglobin, particularly as it relates to erythrocyte content of 2,3-DPG. The authors appear to believe that fresh blood transfusion may have a role in managing infants with hypoxemia, and that massive transfusion with ACD blood more than a week after collection may be hazardous.—J.B.S.


An excellent review of 35 papers describing developments in the field during the 1960s. It is concluded that B12 requirements (1-2 µg/day) can normally be satisfied only if IF, "a part of a transport system developed for efficient catching and retention of a sparsely occurring biologically active substance," is present. Serum autoanti-
bodies block I.F. binding of B$_{12}$, and can thus be used to estimate I.F. in gastric juice and to estimate stimulatory effect of most secretagogues, as well as of food, on I.F. secretion. The I.F. output in the first hour after the subcutaneous injection of 40 $\mu$g histamine acid phosphate per kg body weight, is taken as an estimate of the gastric I.F. secretory capacity. Under these conditions normal subjects have an I.F. output of 5-25 $\mu$g B$_{12}$/hr, duodenal ulcer patients 10-40 $\mu$g/hr, gastritis patients 1-15 $\mu$g/hr, and patients with pernicious anemia less than 0.2 $\mu$g/hr. Gastric carcinoma often has a very low I.F. secretion. The I.F. secretion is correlated to the gastric secretion of acid.—P.G.R.

**LEUKOCYTES**


The colony-stimulating factor (CSF) contained in human urine and capable of stimulating the growth of granulocytic colonies when bone marrow is explanted in an agar medium has been previously shown to be resistant to treatment with preparations of RNAase and DNAase but to be activated by incubation with pronase. Among a large number of proteolytic enzymes, glycosidases, and phospholipases now tested, only $\alpha$-chymotrypsin and subtilisin and ficin caused loss of activity of CSF; the degree of inhibition being correlated with the length of incubation. This is evidence in favor of a peptide component of CSF. Its relative resistance to enzyme proteolysis suggests that controlled enzyme digestion may be a valuable tool for purification of CSF.—F.W.G.


Esterase activity on alpha-naphthyl and naphtol A5 acetates was normal in the polymorphs of patients with the Chediak-Higashi syndrome. Acetylichoronaphotol esterase activity was, on the contrary, very low or absent in the granulocytic series, more than in mature lymphocytes.—G.M.


Esterase activity on the substrate choronaphotol acetate ASD was studied in blood polymorphs of normal subjects. It was found to be present in all polymorphs but more so in young children than in adults and there was a frank rise in febrile patients.—G.M.


In 52 patients in three groups: (1) acute leukemia, (2) chronic aleukemic leukemia-reticulosis, and (3) chronic lympho-reticulosis, bone marrow smears were studied to detect the presence of polysaccharides, peroxidase, alkaline phosphatase, lipids, and glycogen in the reticular cells. A group of normal subjects was similarly studied. The greatest abnormality was noted in acute leukemia in which a distinct increase in peroxidase activity and in content of lipids and glycogen were seen, the glycogen being present in fine granules. These changes appeared to be characteristic of reticular cells destined to form myeloid elements. In lympho-reticulosis no peroxidase or lipid was seen in reticular cells but glycogen was
increased and was present in large granules, changes which appeared to be characteristic of lymphoid cell precursors. No alkaline phosphatase activity was noted in either kind of reticular cells.—J.V.


Acute leukemias were subclassified within the conventional cytological varieties. There was a good correlation between the duration of the first remission and the duration of survival, on one hand, and this classification based only on Giemsa staining, on the other. The actuarial curves of the first remission’s cumulative duration presented a plateau (which is the statistical expression for “cure expectancy”) in 50% or more of the patients, only for two types of acute lymphoblastic leukemia subjected to immunotherapy, the microlymphoblastic and the prolymphocytic. The actuarial curves of the cumulative survival duration presented a plateau for about or more than 50% of the patients for three types: the microlymphoblastic, the prolymphocytic, and the macrolymphoblastic. It seems possible, therefore, to foresee the prognosis at the time of the first perceptible phase of the disease: more than 90% of the patients with the microlymphoblastic type and younger than 20 yr of age presented the cumulative survival duration plateau phenomenon.—G.M.


This paper presents a patient with chronic granulocytic leukemia (C.G.L.) who exhibited one cell line with two Ph’ chromosomes throughout the repeated examinations of his direct bone marrow preparations. This aberration was present regardless of the clinical and hematological phase of the disease. The first cytogenetic analysis was performed during a partial remission and revealed a Ph’ disomy. Following a short interval the patient developed signs of meningitis but with no evidence of blast cell transformation in the peripheral blood, bone marrow, and spinal fluid (postmortem examination revealed however that this episode had been a consequence of meningeal involvement with blast cells). Hematological and clinical signs of acute transformation became evident after 4 mo. It has therefore been concluded that Ph’-disomy in the patient possibly reflected an impending acute transformation.—Z.R.


Neurological complications have become a more frequent component of acute leukemia in infancy in recent years. The causes for this are discussed and reference is made to the observation of meningeal involvement in 36% (26 cases) of infantile leukemia treated at the Torino University’s Pediatric Clinic during the last 3 yr. The series was composed of 25 acute lymphoblastic and one myeloblastic leukemia. A detailed account is presented of the latter case since scintigraphic visualization was obtained of serious meningoencephalitic involvement that occurred at about the fifth mo. Unusual nerve localizations during the course of this disease is reported. Cerebral scintiscans are of particular importance in this respect as a means of diagnosis and in order to control the course of the disease and follow the effect of treatment.—G.L.


Total or partial, though rarely long-last-
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Irreversible aggregation of platelets is always linked to the release of, among other components, biogenic amines and adenine nucleotides. Whereas thrombin, collagen, and immune complexes such as aggregated gamma globulin bring about release without the requirement of aggregation, ADP (in the presence of Ca²⁺ and fibrinogen) does so only if aggregation is allowed to occur. This leads to the suggestion that it is the close contact of the platelets in the first place which induces release. Evidence obtained on human platelets further substantiates this hypothesis. A pH 8, platelets brought in contact by mild centrifugation, released 14C-serotonin under conditions comparable to those with collagen or immune complexes as inducers, i.e., in the absence of Ca²⁺ and other cofactors. Only material contained in specific storage organelles was released, whereas cytoplasmic nucleotides were retained, as exemplified by the absence in the released material of tritiated nucleotides after pretreatment of platelets with 3H-adenine. The release reaction due to cell contact was inhibited by acetylsalicylic acid and phenylbutazone which were also effective when release was triggered by other means. Chlorpromazine and amitriptyline, both known inhibitors of second phase aggregation, failed to inhibit release due to packing of the platelets. It is speculated that blood platelets are induced to undergo the release reaction whenever they are brought into close contact by any mechanism for a certain critical time and under certain conditions. With respect to the mechanism of induction of the release reaction, an “inducing surface” is postulated which would be preformed in immune complexes and collagen, and which would be formed on the platelet surface under certain conditions.—M.C.B.

HEMOSTASIS

Heart-lung machines can effectively oxygenate blood, but their prolonged use causes difficulties not directly related to the patient's heart condition or the specific procedures used to correct it. In extreme cases, a patient may be seriously affected and may develop thrombosis, abnormal permeability of the vascular system (particularly in the lungs), blood clots, and, occasionally, brain damage. The results of previous studies by several investigators suggest that denaturation of blood protein at blood-gas interfaces in the heart-lung machine may be responsible for some of the postoperative difficulties. The authors' investigations were done on the denaturation of gamma globulin, albumin, and their mixtures in the disc oxygenator. They found that gamma globulin solutions subjected to the action of a disc oxygenator of a type often used in open heart surgery showed a concentration-dependent increase in turbidity followed by precipitation of the denatured protein. Furthermore, gamma globulin solutions exposed to the oxygenator were highly reactive with guinea pig complement when compared to control solutions which had not been subjected to the oxygenator. Further indications that gamma globulin is denatured in the oxygenator is also found in the recent reports that the sera of many patients who have had open-heart surgery contain antibodies to gamma globulins (Pretty, H. M. Fudenberg, H. H., Perkins, H. A., and Gerbode, F., Blood 32:205, 1968). The presence of albumin significantly reduced the turbidity and therefore the denaturation of the gamma globulin. In other experiments it was shown that when solutions of gamma globulin were placed in the oxygenator and samples taken at time intervals, its solubility in a 30% solution of ammonium sulfate decreased with time. Soluble gamma globulin disappeared from the solution by a process consistent with first order kinetics. The authors propose the following mechanism for the denaturation of gamma globulin in the disc oxygenator. The first step would involve unfolding of the gamma globulin molecule in the liquid-air interface. The second step is the interaction of the unfolded molecule with another gamma globulin molecule, either native or altered. In subsequent steps, unfolded gamma globulin molecules would interact with other aggregates to produce the observed large increases in turbidity and, ultimately, precipitation. When albumin is present in large excess, the unfolded gamma globulin molecules interact with albumin molecules to produce a complex which does not aggregate further. The formation of this complex would account for the observed slight increase in the turbidity of mixtures of these proteins and their altered solubility in ammonium sulfate solution. On the basis of these results, the authors suggest that long-term operation of the heart-lung machine may produce biologically active quantities of denatured gamma globulin or gamma globulin-albumin complexes and that these altered proteins, particularly the denatured gamma globulin, may activate the complement system and, thus, cause some of the observed medical complications following surgery.

—M.G.B.


Previous investigators have demonstrated that adhesion of platelets to glass requires the presence of fibrinogen. This has been demonstrated with washed platelets, platelets in citrated plasma, and platelets in native whole blood. The availability of a patient congenitally deficient in fibrinogen made possible a quantitative investigation of the need for fibrinogen in the platelet adhesion phenomenon. Platelet adhesiveness to glass was measured by a method devised by the authors. By this technique, an acid-washed glass rectangle measuring $75 \times 10 \times 1$ mm was quickly immersed for 2 min in the blood-fibrinogen mixture following removal of the syringe plunger. The glass rectangle was then twice immersed gently in 0.154 M sodium chloride, fixed in buffered 1.3% glutaraldehyde for 45 min, rinsed in Tyrode's solution and then in water, and air dried. Platelets adhering to the glass surface were counted by incident phase-contrast microscopy in 11 fields along the central longitudinal axis of the rectangle. The effect of fibrinogen concentration on the adhesion to glass of platelets...
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from a fibrinopenic patient was shown clearly by the results. In the absence of added fibrinogen, an average of seven platelets adhered to glass (1000 X magnification field). Fibrinogen concentrations of 0.2 mg/100 ml had no effect on platelet adhesion to glass. Increasing concentration of fibrinogen from 0.5 to 14.0 mg/100 ml produced progressively increasing platelet adhesion. Increasing the fibrinogen concentration above 14.0 mg/100 ml produced only a slight increase in platelet adhesion. It was, therefore, concluded that the extent to which platelets adhere to glass is dependent on the fibrinogen concentration. Platelet adhesion increased progressively in a linear fashion when an increase in concentrations of purified fibrinogen (0.5-14.0 mg/100 ml) were added to the blood of a patient with congenital afibrinogenemia. —M.G.B.


A postmature infant developed a hemorrhagic diathesis at 1 hr of age. Coagulation profile was characteristic of DIC. One hour after heparin therapy started, oozing stopped and within 12 hr coagulation studies indicated significant improvement. Heparin was continued for 5 days and the infant did well. By day 5, coagulation tests were normal except for moderate thrombopenia which persisted through the first week. The authors suggest that the DIC was initiated by liberation of thromboplastin from the placenta, which on examination revealed multiple areas of infarction.—J.B.S.


Intravascular coagulation, variable in severity from one patient to another, was found with the Merskey hemagglutination inhibition test, in 101 patients, including myelocytic leukemia, extracorporeal circulation, and during SK treatment. The authors confirm the value of the Merskey test even in the absence of fibrinogen decrease. —J.C.


Two boys and seven girls with Glanzmann’s disease were found in a strongly consanguineous group of gypsies. The five groups of siblings were related and belonged to three different generations. To our knowledge, the family tree is the largest published. The genetic trait existed in heterozygous form in two ancestral couples and lead to the homozygous patients in which the disease was transmitted as an autosomal recessive character.—J.C.


The level of factor VIII was determined in plasma samples from 28 normal subjects of both sexes, aged 20–30 yr. In addition, factor VIII level was determined in four mothers and four sisters of hemophiliacs. A modified Hardisty and Macpherson assay was applied (Bleeding Disorders, ed. 1. Philadelphia, Davis, p. 304). The statistical significance of the results was assured by the Fisher’s test. The study revealed a wide range of variations of factor VIII values in normal subjects; the factor VIII level ranged in normal females from 42 to 196 units per 100 ml and in normal males from 68 to 150 units per 100 ml. Interval of variation of factor VIII level in males and females was tested by comparing the variation of the standard deviation for different plasma dilutions. Highly significant variations of factor VIII level were found more frequently in females than in males. On the other hand, the difference in concentration of factor VIII between mothers and sisters of hemophiliacs and normal females was not statis-
tically significant. These findings lead to the conclusion that the wide variations of factor VIII level in normal females prevent detection of the female carrier in hemophilia families.—Z.R.


A 4-yr-old white boy, previously diagnosed as having hemophilia with antihemophilic globulin (AHG) deficiency (5% AHG concentration), developed acute lymphoblastic leukemia. At the time of the original diagnosis of leukemia, AHG levels were 75 and 100%; they fell to 20 and 49% of normal on two occasions during remission; and, during a subsequent relapse, they rose to 92% of normal. AHG activity has been demonstrated in the lysates of cultured normal white blood cells (WBC), while lysates of cultured WBC from hemophilic donors showed either no or very slight AHG activity. In general, the amount of AHG produced by cultured WBC from hemophilic subjects correlated with their plasma AHG concentration. In view of the previously reported elevations of AHG in normal persons with acute leukemia and these findings, it is postulated that the lymphocyte is a probable site of production of AHG, an important clue.—I.E.U.

IMMUNOHEMATOLOGY


The immunocytoadherence test (ICA) has been applied to the study of the immune response of mice during the development of leukemia. Groups of 24 BALB mice were challenged with a lethal dose of syngeneic leukemic cells followed a day later by an immunizing dose of sheep red blood cells (SRBC). Number of spleen rosette-forming cells, (RFC) as well as hemagglutination titers were determined daily, up to day 11 which coincided with death of the animal from leukemic colonization. This primary immunization led to the characteristic ICA curve in the controls with a first peak of 9.3/1000 RFC on day 5 followed by a second peak of 10 RFC per 1000 on day 7, while the leukemic mice gave consistently lower values with a single peak of 4.5 RFC per 1000 on day 5. After a second immunization with SRBC, the leukemic mice also showed a statistically significant drop in RFC with a peak of 6 RFC per 1000 on day 6, as compared to a control peak of 12 RFC per 1000 on day 3. No rosette formation could be observed on day 11 when the leukemic infiltration of the spleen was maximal. As for hemagglutinin titers, the leukemic mice gave slightly lower values than the controls. It can be concluded that the leukemic mice are immunodepressed and that the leukemic cells are incapable of forming rosettes.—M.J.


The specificity of the leukocyte migration inhibition method was tested, using PPD on tuberculin-sensitive and nonsensitive blood donors. Inhibition was never present in anergic subjects. In tuberculin sensitive subjects, there was marked inhibition in five of seven tests. The migrating cells were studied by staining the bottom of the chambers with May Grünwald-Giemsa and by adding trypan blue to the culture medium. It was possible to observe that both lymphocytes and monocytes migrated actively out of the capillary tubes. Partially purified extracts were prepared from human lymphomatous lymph nodes; these were assayed on the same patients and, simultaneously, on normal subjects, using direct and indirect techniques of inhibition. Direct tests were carried out usinguffy coat leukocytes packed in capillary tubes, as migrating cells.
Indirect tests were carried out in two steps: 24-hr lymphocyte culture supernatants of antigen-stimulated and nonstimulated cells were first obtained by the usual method; guinea pig peritoneal cells were then, packed in capillary tubes and were left to migrate in chambers containing the above mentioned supernatants as culture media. In both cases, the areas of migration in chambers with and without antigen were compared according to the following formula:

\[
\text{Migration index} = \frac{\text{Migration area with Ag} \times 100}{\text{Migration area without Ag}}
\]

Results revealed the presence of delayed hypersensitivity reaction in six patients with lymphomas, when tested against an auto-extract of their neoplastic lymph nodes. Correlation of migration index values, using both methods, showed that the migration test, carried out directly on human blood leukocytes, is a sensitive and reliable method for the detection of cellular immunity, and that migration inhibitory factor synthetized by human lymphocytes is able to act upon guinea pig macrophages.—M.J.


In a study of 229 patients with malignant lymphoma and chronic lymphocytic leukemia, in investigation of allergic symptomatology and immunoglobulin levels was conducted. Patients with chronic lymphocytic leukemia demonstrated a decreased incidence of allergic symptomatology that could be correlated with diminution of serum IgE levels. Of interest was the observation that some patients with lymphoma lost their symptoms of allergy prior to onset of the malignancy. History of allergic symptoms was elicited with equal frequency in both patients with malignancy and control population.—J.E.U.


The immunologic status of 146 patients with acute leukemia has been explored at different stages of the disease, before and during various treatments, and compared with clinical and hematologic status. Pre-established delayed hypersensitivity (D.H.) was studied by intradermal injection of four antigens; antibody response by injection of polio vaccine. D. H. reactions were found to be slightly impaired before treatment. During induction therapy, the immunodepression involved mainly antibody production, while D. H. was preserved in most cases. Abolition of D. H. reactions occurred during drug-induced aphasias, and strongly correlated with the severity of the aplasia. Maintenance treatments tested did not significantly affect immunologic reactivity of patients in remission.—J.E.U.


The authors describe a case of lethal pancytopenia with an abnormal karyotype in the blood and bone marrow (46, XY, C'). This abnormality, until now considered as peculiar to malignant blood diseases, is an argument in favor of a direct action of the virus on the cells.—J.C.


Prediction equations for blood and plasma volumes in children ages 3–15 yr are relatively inaccurate when based on weight, particularly in girls. The best predictions for intravascular volume were obtained from height by an exponential equation. Nomograms and regression equations are presented. In addition, the author describes a 7% increase in plasma volume within 10–15 min after children go from the erect to the horizontal position.—J.B.S.