ERYTHROCYTES

Erythrocyte Adenine Phosphoribosyltransferase in the Lesch-Nyhan Syndrome. P. Bashkin, O. Sperling, R. Schmidt, and A. Zeineberg. Department of Chemical Pathology, Chaim Sheba Medical Center, Tel-Hashomer and Tel-Aviv University Medical School, Rogoff-Wellcome Medical Research Institute, Beilinson Hospital, Petah Tikva and Rehabilitation Center for Children, Asif, Harofe Government Hospital, Zerifin, Israel. Isr J Med Sci 9:1553–1558, 1973

Lesch-Nyhan syndrome, a neurologic disease found in children, is associated with increased purine production. It is caused by an almost complete deficiency of the X-linked enzyme, hypoxanthine-guanine phosphoribosyltransferase. In dialyzed hemolysates from affected children, adenine phosphoribosyltransferase (APRT), a separate autosomal enzyme, exhibits increased activity and relative stability to thermal inactivation. APRT in the hemolysates of two children with Lesch-Nyhan syndrome was studied. The enzyme was normally sensitive to destabilization against heat inactivation by adenine, but resistant to destabilization by hypoxanthine. Other properties of the enzyme, including Km for substrates, optimal magnesium concentration, electrophoretic mobility, pH profile, and sensitivity to inhibition and to stabilization by 5-phosphoribosyl-l-pyrophosphate against thermal inactivation, were normal. The mechanisms underlying the abnormalities of APRT in the dialyzed hemolysates are discussed.—B.R.

Neonatal Hyperbilirubinemia and Glucose-6-Phosphate Dehydrogenase Deficiency. B. Milbauer, N. Peled, and S. Svirsky. Newborn and Premature Department of Chemical Pathology, Chaim Sheba Medical Center, Tel-Hashomer and Tel-Aviv University Medical School, Israel. Isr J Med Sci 9:1547–1552, 1973

During the course of a 1-yr study, all infants born at the Municipal-Governmental Medical Center, Tel-Aviv, were assayed for red blood cell glucose-6-phosphate dehydrogenase (G6PD) activity. Of 7520 infants, 360 were found to be G6PD-deficient. Hyperbilirubi-
nemia, with serum bilirubin values above 14 mg/100 ml, was present in 38 (14.3%) of 265 G6PD-deficient newborn males and in 260 (7.2%) of 3582 newborn male infants with normal G6PD activity. Statistical evaluation showed the difference to be highly significant. Neonatal hyperbilirubinemia in G6PD-deficient newborn infants was not associated with excessive hemolysis. — B.R.


Hemoglobin C was found in 44 of 79 members from the Bedouin tribe "El Heib" in northern Israel. Four subjects were homozygotes and 40 heterozygotes. Anemia with a hemoglobin level of ≤ 11 g/100 ml was present only in one homozygote, five heterozygotes, and three normal subjects, although marked iron deficiency was found in 57 of 73 subjects and folate deficiency in nine of 35 subjects. The presence of hemoglobin C seemed to have no effect on the survival and well-being of the members of this family. Intermarriage between close and distant relatives and a high birth rate are the main factors responsible for the high incidence of the hemoglobin variant. — B.R.


Four hundred and eighty-nine leprosy patients, lepromatous type, and another group of 518 individuals nonaffected by leprosy were submitted to screening tests for S hemoglobin. Among the leprosy patients, 401 were white (272 males and 129 females) and 88 were black (61 males and 27 females), while the control group was composed of 334 whites (112 males and 242 females) and 164 blacks (52 males and 112 females). The screening tests were performed by means of Sickledex, using half of the recommended volumes. These tests allowed the authors to demonstrate positive reactions among 9.09% of the black leprosy patients (five males and three females), 7.32% of the black controls (four males and eight females), and 0.75% of the white leprosy cases (two males and one female). Complete agreement between these positive reactions and the results of the standard sickling test with 2% sodium metabisulphite solution, after 30 min at room temperature, was found. The starch-gel electrophoretic examination of the hemolysates of the 23 ascertained Sickledex-positive individuals demonstrated that they exhibited the sickle cell trait (AS heterozygotes). The same investigation applied to the hemolysates of 78 Sickledex-negative black leprosy patients has shown that all of them were homozygous for the normal adult hemoglobin gene. All the leprosy patients with the sickle cell trait, as well as ten of the 12 AS heterozygous individuals of the control group, were submitted to detailed clinical examination and most of them also to radiologic and electrocardiographic investigations. Laboratory proofs included complete hemogram, protein electrophoresis, determination of serum bilirubin and transaminases (SGOT and SGPT), serologic reactions for Chagas' disease and syphilis, as well as urine and stool parasitologic examinations. The frequency of individuals exhibiting the sickle cell trait did not support the hypothesis that leprosy might have contributed to maintaining high frequencies of that allele by preferential selection of AA homozygotes, in spite of the similar geographic distribution of both leprosy and the gene for S hemoglobin in Africa and Asia. The data obtained are also not consistent with a secondary hypothesis that the gene for S hemoglobin might have a pleiotropic affect on susceptibility to leprosy infection. The sickle cell trait does not seem to affect the clinical evolution of leprosy or to intensify the signs and symptoms manifested by AS heterozygous leprosy cases. — M.J.


In four patients with pernicious anemia, urinary erythropoietin (bioassayed) fell from high levels before vitamin B₁₂ treatment to minimum levels 3–5 days after beginning of
vitamin B₁₂ treatment, although hemoglobins remained low. Bone marrow DNA synthesis normalization is invoked as an explanation.—P.G.R.


Literature reports of erythrocytosis secondary to tumors or renal disease have been collected. Reports of 118 hypernephromas, 50 cerebellar hemangiomas, and 49 hydrenephroses or cystic kidneys generally concerned hypertensive men with increased erythropoietin values. Some pheochromocytomas and kidney transplant receptors develop erythrocytosis. Reports of 64 liver tumors also concerned men, but erythropoietin levels were usually normal. The possibility of increased erythropoietin synthesis in the liver is discussed. Reports of 24 large uterine myomas, generally with normal erythropoietin values, are also cited. Animal studies show that renal anoxia can stimulate erythropoietin production. Abstractor's comment: Renal hypoxia and tumor production of erythropoietin or its precursors appear to explain most cases of erythrocytosis. However, catecholamines in pheochromocytoma may reduce the plasma volume and cause relative erythrocytosis.—P.G.R.


A 2½-yr-old girl with SCA developed diffuse osteomyelitis due to a species of Arizona bacilli, one of a group of Enterobacteriaceae closely related to Salmonella.—J.B.S.


Serum and erythrocyte folate levels were studied in children with SS, SA, and AA hemoglobin. Serum folate level was significantly reduced in half the children with SCA and in a third of each of the control groups. Hypersegmented neutrophils were significantly more frequent in SCA and somewhat more frequent than normal among children with SCT. Low erythrocyte folate was seen in two patients with SCA. Serum vitamin B₁₂ levels were normal in all patients and controls. A fourth of the children with SCA had evidence of growth retardation, but there was no correlation between this finding and the serum of RBC folate activity, nor between growth and hematocrit levels. Although folate administration to one child with low erythrocyte folate activity resulted in a 6% increase in hematocrit, no changes in growth were seen in three small sicklers given folic acid for almost a year. These findings contradict an earlier study from Africa which suggested that folate deficiency contributed to the growth retardation seen in SCA.—J.B.S.


In the group of chronic anemias resistant to usual treatments, primary acquired sideroblastic anemia is characterized by a macrocytic tendency, anisochromia, low reticulocyte count, raised serum iron, usually neutropenia, and a bone marrow rich in erythroblasts with megaloblastoid abnormalities, excess hemosiderin granules (sideroblasts), sometimes with a crown-shaped layout. Radioisotope investigations using ⁵⁹Fe and ⁵¹Cr demonstrate the inefficiency of erythropoiesis. The course lasts several years. There may be transfusion complications, e.g., hemochromatosis, serum hepatitis, and alloimmunization. One should distinguish this type of anemia from congenital sideroblastic anemia, the transmission of which is sex linked, and from secondary sideroblastic anemia in the course of epithelial carcinomas, malignant blood diseases, and poisoning and, also, from pyridoxine-responsive sideroblastic anemia, preleukemic states, and chronic erythemic myelosis.—J.C.

LEUKOCYTES


Leukotaxis, random migration, and NBT reduction were evaluated in children with cystic fibrosis when they were well and during respiratory infections. Leukotactic activity was

The ability of human, pig, and chicken polymorphonuclear leukocytes to phagocytose and kill Candida albicans was studied. Polymorphs of the pig and chicken were less effective than those of man. Since cells from these two species contain less myeloperoxidase, and since inhibition of myeloperoxidase by KCN reduced the degree of killing, the authors suggest that myeloperoxidase may be involved in candidal activity. — A. A. M.


Escape of granule constituents from white cells occurs during phagocytosis. The authors demonstrate release of myeloperoxidase by phagocytosing granulocytes into the medium. This was shown by extracellular iodination of plasma proteins with incubation medium of phagocytosing white cells, hydrogen peroxide, and iodine were incubated. Large particles such as killed yeast cells induced larger release of iodinating capacity than the smaller, Escherichia coli. These studies indicate that bacteria may also be killed by extracellular action of the antimicrobial myeloperoxidase and hydrogen peroxide without necessarily being interiorized by phagocytes. — M. S.

Homology Between Burkitt Herpes Viral DNA and DNA in Continuous Lymphoblastoid Cells from Patients With Infectious Mononucleosis. E. Kieff and J. Levine. Department of Medicine and the Committee of Virology, University of Chicago, Chicago, Ill. Proc Natl Acad Sci USA 71:355, 1974

More than 90% of the sequences of purified in vitro labeled DNA from Epstein-Barr (EB) virus which was prepared from Burkitt's lymphoblastoid cells were homologous to the DNA of the herpes virus contained in cell lines derived from patients with infectious mononucleosis. Thermal stability of the homologous and heterologous hybrid DNA molecules could not be differentiated, indicating at least 97% matching of base pairs between DNA of EB virus and the herpes viral DNA contained in the lymphoblast from patients with infectious mononucleosis. — M. S.


This unique observation in an 18-yr-old girl treated for 2 yr by contraceptive drugs showed a blockade of the C3 classic activation while the alternate pathway was normal. The granulocytes of the patient did not phagocytose latex particles in the presence of patient's serum, but the defect was corrected by the addition of normal fresh human serum. C4 esterase inhibitor was normal and the patient never developed hereditary angioneurotic edema in which C4 level is very low during crisis. — J. C.


Alveolar macrophages were obtained by bronchopulmonary lavage from four cigarette smokers and one nonsmoker. A small percentage, 0.35%-1.25%, were in DNA synthesis, as shown by incorporation of $^3$H-thymidine. Cells having the morphologic and functional characteristics of macrophages persisted for several weeks in tissue culture, and a low degree of labeling was observed in such cells. The authors conclude that pulmonary macrophages in man may be long-lived cells, capable of replication and thus perhaps sustained by local cell proliferation in addition to migration of precursor cells from the marrow. — A. A. M.

The cytoplasmic granules of leukocytes contain a number of basic proteins having antibacterial activity. The authors studied urine from seven cases of chronic myelocytic leukemia undergoing therapy and in two cases detected a cationic protein which was distinguishable from lysozyme. The same protein could be identified using specific antisera in leukocyte lysates from chronic myelocytic leukemia patients. — A.A.M.


Patients with ALL and their sibs have an excess of Sydney palmar flexion creases. Males with ALL also have an excess of digital whorl patterns. — J.B.S.


Four children with ALL presented with severe back pain as the predominant complaint. All had mild to moderate anemia and thrombocytopenia, with normal white blood cell counts, and fewer than 5% blasts. Bone marrow in each child revealed complete replacement with lymphoblasts. Treatment with diverse anti-leukemic agents was accompanied by a rapid relief of symptoms, which was not, even over a prolonged period of follow-up, associated with roentgenographic evidence of improvement in the pretreatment findings of generalized osteoporosis with collapse of several thoracic vertebrae. — J.B.S.

**Agranulocytosis After Treatment With Procainamide.** A. Hansen. Department of Internal Medicine, St. Elizabeth’s Hospital, Hoorlem, The Netherlands. Ned Tijdschr Geneeskd 17:624–625, 1974

Two female patients, 80 and 71 yr old, taking 1500 mg procainamide daily during 40 and 60 days, respectively, developed agranulocytosis. The oldest, with signs of angina tonsillaris, was treated with prednisolone and antibiotics, but died. The other patient was treated with antibiotics alone and recovered. Until now 11 other cases have been reported in the literature, of which four had a fatal outcome. Agranucytosis was discovered between the 16th and the 78th day after starting treatment. — K.P.


Surface receptor properties of cells from spleen or lymph node from six patients with nodular lymphoma were compared with properties of lymphocytes from histologically normal spleen and node. Properties investigated were C3 receptor, receptor for cytophilic antibody, and spontaneous rosette formation with sheep erythrocytes. These surface receptors are characteristic of B lymphocytes, histiocytes, and T lymphocytes, respectively. Rosette formation was studied in cells from tissue suspension and in frozen tissue sections. Proportions of complement receptor and sheep erythrocyte rosette-forming lymphocytes showed variation in control spleen suspensions and greater variation in suspensions from control lymph nodes. In spleen, T and B cells were more nearly equal, while in nodes, T cells were more numerous than B cells. In general, suspensions of neoplastic cells from nodular lymphomas showed similar cell distribution, but in all cases neoplastic–appearing cells showed evidence of presence of C3 receptor sites. Frozen sections of normal lymph node, spleen, and tonsil subjected to rosette formation challenge showed the expected distribution: follicular presence of C3 receptor and histiocytic localization of cytophilic antibody receptor. The authors demonstrated presence of C3 receptor sites in the neoplastic nodules from the patients with nodular lymphoma. They conclude that a follicular B-cell origin for nodular lymphomas is probable. — P.F.

**HEMOSTASIS**

**Inhibition of Platelet Adherence to Collagen-coated Surface by Non-steroidal Anti-inflammatory Drugs.** Pyrimidol-Pyrimidine and Tricyclic Compounds and Lidocaine. J. P. Cazenave, M. A. Packham, M. A. Guccione, and J. F. Mustard. Department of Biochemistry and Department of Faculty of Medicine, University of Toronto, and Department of Pathology, Faculty of Medicine, McMaster University, Hamilton, Ontario, Canada. J Lab Clin Med 83:797, 1974

The effect of a number of drugs inhibiting the
platelet release reaction was studied on the adherence of platelets to collagen-coated surfaces. Adherence of washed platelets labeled with $^{14}$C-serotonin or $^{51}$Cr was measured in Tyrode solution containing albumin and apyrase. Under these conditions there was no aggregation and very little release or loss of platelet constituents. Acetylsalicylic acid, phenylbutazone, imipramine, promethazine, and lidocaine as well as a number of experimental pyrimido-pyrimidine compounds significantly inhibited adherence of platelets to collagen. Under the conditions used, release of alpha granular content was not significant. It could be shown that acetylsalicylic acid altered the platelets and not the collagen. Nor did the drug remove platelets already adherent to the collagen. The authors speculate that the inhibitory effect of these drugs on collagen-induced platelet release reaction may be secondary to the inhibition of platelet adherence to collagen.—M.S.

**Isolation and Structure of Two Prostaglandin Endoperoxides that Cause Platelet Aggregation.**


Brief incubation of arachidonic acid with the microsomal fraction of a homogenate of vesicular gland of sheep in the presence of 1 mM $p$-mercuribenzoate yielded two prostaglandin endoperoxides. Both of these endoperoxides were precursors of prostaglanden E2. The structures of these compounds, i.e., 15-hydroperoxy-9 $\alpha$, 11 $\alpha$-peroxidoprosta-5, 13-dienoic acid (prostaglanden G2) and 15-hydroxy-9 $\alpha$, 11 $\alpha$-peroxidoprosta-5, 13-dienoic acid (prostaglandin H2) were determined mainly by chemical transformations into previously known prostaglandins. The new prostaglandins were 50–200 times, PGG2 and 100–450 times, PGH2, more active than prostaglandin E2 on the superfused aorta strip. Addition of 10–300 ng/100 ml of these endoperoxides to suspensions of washed human platelets resulted in their rapid aggregation. The authors speculate that the inhibitory effect of these drugs on collagen-induced platelet release reaction may be secondary to the inhibition of platelet adherence to collagen. —M.S.

**A Comparison of Bovine Prothrombin, Factor IX (Christmas Factor) and Factor X (Stuart Factor).**


A comparison was made between electrophoretic behavior, chemical composition, amino terminal sequence, and immunologic properties of bovine prothrombin, factor IX, and factor X. Immunologic cross-reactivity was found between the antibody to prothrombin and factor X, although prothrombin and factor X differed substantially in amino acid and carbohydrate composition. Considerable amino acids sequence homology was found in the amino terminal portion of prothrombin factor IX and the light chain of factor X. These data were interpreted to provide further evidence for support of the hypothesis that at least three of the vitamin K-dependent clotting factors have evolved from a common ancestral gene. —M.S.

**Prevention of DIC (During Schwartzmann Reaction) by a System (Creatine Phosphate-Creatine Phosphokinase) Consuming Plasmatic ADP.**


The creatine phosphate-creatine phosphokinase (CP-CPK) system is able to disaggregate platelets even during the so-called irreversible aggregation. The permanent infusion of CP-CPK during 4 hr in the rabbit reduced the velocity of ADP-induced aggregation by about 50%. A generalized Schwartzmann reaction was obtained in the rabbit by two successive injections at a 24-hr interval of Escherichia coli endotoxin. All the animals, even those treated by CP-CPK, developed shock, but those treated had a platelet count higher than those not treated. In the control group 90% had DIC lesions, namely in the kidney, while in the treated group, lesions were found only in 20% of the animals. —J.C.

**C1 and Platelets.**


C1 was demonstrated in human platelets. The role of this component of complement in
Platelet aggregation and in platelet function in general is discussed.—J.C.


The authors report their experience in rheumatoid purpura in children and discuss 100 cases observed in a nonspecialized pediatric unit. The clinical signs are fully discussed and compared with data in the literature. There was renal involvement in 25% of the series. They then discuss the course of the disease, both in the simple forms and in the forms with renal complications. In the latter group, a certain number of children were followed up clinically and in the laboratory for several years. The authors also discuss the pathogenesis of the disease, noting the information supplied by renal immunofluorescence.—J.C.


A serious clinical syndrome consisting of pulmonary artery thrombosis was seen in a 32-yr-old woman who had been taking a low dosage of an estrogen-progestagen agent for 3 mo. Apart from mild varicose veins without evidence of thrombophlebitis, there existed latent diabetes, a mixed-type hyperlipemia, and hyperuricemia. Reference is made to other similar cases. By virtue of the clinical severity of the condition, urokinase was used in high dosage and with success, using scintigraphy and angiography to monitor the results. The authors’ marked a preference for the use of urokinase. The dose used is defined and discussed.—J.C.


Heparin precipitation of the different classes of human serum lipoproteins can be accomplished either at neutral pH after addition of divalent cations or at acid pH without addition of metal ions. In both cases, the ease of precipitation is different for the various classes of lipoproteins. At neutral pH it depends on the protein/lipid ratio (the precipitation is more easily achieved the lower the protein/lipid ratio), while at acid pH it is related to the nature of the protein moiety.—J.C.

Amidase Activity of Human Macroglobulin. A. Szwczuk, and A. Szcze{z}ek. Department of Biochemistry, Institute of Immunology Experimental Therapy, Polish Academy of Sciences, Wroclaw and Department of Clinical Immunology, Institute of Internal Medicine, School of Medicine, Kraków, Poland. Arch Immunol Ther Exp (Warsz) 21:839-847, 1973

The amidase activity of α-2-macroglobulin purified from human plasma and serum was studied using N-s-carbobenzoxydiglycyl-l-arginine-β-naphthylamide as substrate. Serum α-2-macroglobulin exhibited distinct amidase activity, while weak amidase activity was found in plasma α-2-macroglobulin. When minute amounts of trypsin, plasmin, or thrombin were added to plasma α-2-macroglobulin a marked increase in amidase activity occurred. Trypsin inhibitors from pancreas, lung, and thyroid gland, and also Trasylol as well as ε-aminocaproic acid, strongly inhibited the new amidase activity but did not affect the spontaneous amidase activity of serum α-2-macroglobulin. Neither soybean trypsin inhibitor nor ovomucoid affected the amidase activity of the macroglobulin. The effect of incubation time, pH, and metals on amidase activity was investigated. In clotting plasma, amidase activity appeared in the α-2-macroglobulin simultaneously with the appearance of thrombin. Generation of the amidase activity depends both on thrombin formation and on the presence and concentration of α-2-macroglobulin.—M.K.


Template bleeding times (BT) were performed before and 2 hr after ingestion of analgesics other than ASA, or corticosteroids, by controls and hemophiliacs. No change in BT was observed after prednisone, codeine, propoxyphene, HCl, salicylate choline, or pentazocine HCl. No evaluation of effectiveness of analgesic effect of any of these agents in arthropathy is included.—J.B.S.
IMMUNOHEMATOLOGY

A Severe Hemolytic Transfusion Reaction Without Demonstrable Antibodies in the Serum. R. M. A. Kursjens and V. A. J. M. Kunst. Department of Hematology, University Hospital St. Radboud, Nijmegen, The Netherlands and Bloodbank, Department of Hematology and Department of Immunology, St. Radboud University Hospital, Nijmegen, The Netherlands. Ned Tijdschr Geneesk 11:367-370, 1974

A report is presented of the case of a woman with a hemolytic transfusion reaction in whose serum no blood group antibodies that might have caused the reaction could be demonstrated. Possibly, the transfusion reaction may have been due to cell-mediated immune mechanisms. Familiarity with reactions of this nature is of importance because of the changes in transfusion management necessary in these patients. — K. P.

Immunoresistance in Tumors; An Experimental Study of a Model System. S. Friberg Jr. Department of Tumor Biology, Karolinska Institute, Stockholm, Sweden. Stockholm 1973, Thesis

"Immunoresistant" ascites tumor cells transplantable to many mouse strains (allo-transplantable) have "masked" antigens exposed on cell rupture, and a cell-membrane carbohydrate composition differing from that in immunosensitive, nonallo-transplantable tumor cells. When fused with normal fibroblasts, the allo-transplantable tumor cells lose allo-transplantability, and acquire some cell-membrane characteristics of immunosensitive cells. — P. G. R.


In a youngster with symptomatic milk allergy, typical L.E. and turt cells were seen in slide preparations. These findings coincided with milk administration, and the presence of milk precipitins, hemagglutins against milk proteins, as well as extractable nuclear antigen (ENA) antibodies. — J. B. S.

MISCELLANEOUS


The effect of several estrogenic compounds on the number of colony forming units (CFU) in the marrow and spleen, on the $^{59}$Fe uptake into these organs, and on peripheral blood counts was studied. Diethylstilbestrol, estrone, and estradiol benzoate all caused a rapid decrease in the number of CFU both in the marrow and the spleen. Those in the spleen regenerate rapidly and reached supranormal levels within 4 days after discontinuation of the estrogen treatment; those in the marrow did not increase for at least 2 wk after discontinuing therapy. $^{59}$Fe uptake of the spleen increased to higher than normal levels 3 days after stopping estrogens, while that of the femur decreased and remained low. Profound thrombocytopenia and also leukopenia, i.e., neutropenia, were evident in the estrogen-treated mice, but no anemia was present. A direct damage of CFU by estrogens at all hematopoietic sites was postulated. The discrepant behavior of thrombopoiesis and myelopoiesis on one hand and erythropoiesis on the other hand may be due to a more favorable microenvironment for myelopoiesis and thrombopoiesis in the bone marrow, which was shown to undergo profound osteosclerosis after estrogen treatment. The rapid regeneration of erythropoiesis after estrogen treatment was thought to be due to the more favorable microenvironment for this function in the spleen. Hematopoiesis in the latter apparently regenerates more rapidly after discontinuation of estrogen treatment. — M. S.


Experiments upon dogs showed that massive transfusions of blood (40 ml/kg body weight) caused severe and sustained changes in liver and kidney function by reducing the blood circulation in those organs. Because this effect could be aggravated by the pretransfusion administration of atropine and prevented by pretransfusion ergotamine, it is suggested that reduced blood circulation in liver and kidneys was due to excitation of the sympathetic nervous system. — J. V.

Extensive tests, including tests of cardiac function and tests of blood coagulation, were carried out in 65 patients with cardiac sclerosis and anemia before, 24 hr after, and 72-96 hr after blood transfusion therapy using both regular donor and cadaver blood. Slow (droplet) transfusions produced brief changes in the contractile function of the myocardium, and later improvement of the myocardial function was noted. An increase in coagulation indices showed no tendency to favor the development of thromboembolism. In cases where hypercoagulability had been a clinical problem, transfusions of cadaver blood increased the fibrinolytic activity in a beneficial manner and in such patients, this procedure is to be recommended.—J.C.


Retrospective study of fine needle biopsies of spleens from 17 patients with polycythemia vera, nine of whom had splenomegaly, showed no major incidence of frequent abnormalities except for many platelets.—P.G.R.


Boys and girls between 2 and 17 yr of age who received cyclophosphamide for nonmalignant conditions had normal levels of FSH and LH despite continuing therapy, in some instances, for 1-2 yr.—J.B.S.


The median survival rate of children with disseminated neuroblastoma, treated by protocol with VCR and CPM, during 1966-68 was compared to that of children treated in 1956 and 1962. Although over-all statistics seemed the same, patients who could be classified as "responders" survived twice as long as "non-responders," who made up 40% of the 51 patients evaluated.—J.B.S.

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BOOK REVIEWS


This monograph, true to its title, deals with clinical trials in acute leukemia, but it is also concerned with three more or less separate problems: diagnosis, study design, and the reporting of results. This last section is of the least...