ABSTRACTS
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ERYTHROCYTES


The authors suggest that two forms of the enzyme pyruvate kinase exist in human erythrocytes. At low phosphoenolpyruvate concentrations the rate of the reaction corresponds well to the Michaelis-Menten equation; at higher concentrations the pyruvate kinase kinetics are similar to those of an allosteric enzyme with two possible forms, activated or cooperative. The allosteric enzyme is activated by FDP and inhibited by ATP. The “Michaelian” form is not stable and disappears at low protein concentration, at high ionic concentration and with ageing. Cu++ and PHM13 are inhibitors at low concentrations. Abstractor's comment: The investigations were carried out with hemolysates or with concentrated preparations of human erythrocyte pyruvate kinase. It is possible, however, that FDP (which is present in the hemolysate) binds to the enzyme and influences in this way Michaelis-Menten kinetics.—K.P.

Change in Electrophoretic Mobility of Glucose-6-Phosphate Dehydrogenase with Aging of Erythrocytes. B. Bakav, W.L. Nyhan, and E.S.J. Monkus.

Erythrocyte G6PD isoenzymes of neonates, and from young red cells of adults, migrate in acrylamide gel 3%–4% faster than isoenzymes of older red cells from adult blood. The observation that in all three groups of red cells the G6PD was in the same state of aggregation, and that the differences in migration could be eliminated by equilibrating the hemolysates with NADP, indicate that the difference in charge is mediated by differences between young and old red cells in binding of NADP to G6PD. This may be related to the fact that erythrocyte NADP decreases as the red cell ages, or may indicate that “old” G6PD has less affinity for NADP.—J.B.S.

Erythrocyte Lipids in Childhood. R.C. Neerhout.
Total erythrocyte lipid levels fall during the first 6 mo of life, then rise slightly after the age of 6 yr; these changes reflect changes in cell size. Between 6 mo and 6 yr, phospholipid content is lower and cholesterol is higher than in red cells of older children and adults. While sphingomyelin levels remain constant, phosphatidyl choline progressively increases during early childhood reaching adult levels after 6 yr. Fatty acid partition demonstrates the adult pattern by 1 yr of age. —J.B.S.


Although neonatal RBC have decreased GSH-peroxidase activity, the response to H2O2 stress was similar in neonatal and adult erythrocytes. Thus the increased sensitivity to oxidants seen in neonates’ RBC appears not to be related to deficient GSH-peroxidase. —J.B.S.


In contrast to adult erythrocytes, exposure of red cells of neonates to acetylphenhydrazine (APH) leads to irreversible loss of hexokinase and glyceraldehyde-3-phosphate; exposure to medionine causes a decrease rather than an increase in incorporation of fatty acids into the erythrocyte membrane. When APH exposure took place in the presence of added glucose, the changes noted were different, but again adult and neonatal red cells responded dissimilarly. Impairment of glycolysis by oxidants could be prevented if the neonatal RBCs were pretreated by exposure to CO, suggesting that H2O2 mediated the metabolic injury noted. —J.B.S.


Per mole of heme catabolized, 1 mole of CO and 1 mole of bilirubin is produced. Thus, measurement of CO production can be used to evaluate heme turnover and bilirubin production. Among Rh-erythroblastic infants CO production is markedly increased prior to the first exchange transfusion and may remain so following this and one or more subsequent exchanges. The age at which normal CO production was first noted ranged between 74 and 156 hr. The source(s) of the heme from whose catabolism the postexchange CO is produced remains unclear. Hemolysis of remaining Rh-positive cells, or newly produced antibody susceptible erythrocytes, damaged transfused erythrocytes, and/or “early-labeled” bilirubin are mentioned as possible sources. It may also be that heme which has accumulated in the form of methemalbumin during rapid hemolysis in utero, may be a major source. —J.B.S.


Cultures of normal human marrow exposed to trimethoprim in concentrations similar to those reached in vivo showed no disturbances in folate metabolism, and neither did normal marrow in vivo. Cultures of folate-deficient marrow suffered further deterioration in defective DNA synthesis, not corrected by folate or B12. This was confirmed in vivo. It is concluded that trimethoprim produces its hematologic side effects only in patients with preexisting folate or B12 deficiency. —F.W.G.


When transferrin saturation is above 16%, infants and toddlers usually maintain hematocrits of at least 33%. The authors indicate that in many instances saturation levels below 16% precede appearance of anemia by several months, and that half the children 6–16 mo of age with hematocrits above 33% had evidence of inadequate iron stores, if 16% saturation is taken as the lower limit of normal. —J.B.S.


PH syndrome in conditions with hypoplastic
hemopoiesis is considered as the potential first phase of Strubing-Marchiafava disease, i.e., paroxysmal nocturnal hemoglobinuria (W. Dameshek, 1967, G. Alexeev, 1972). This phase is characterized by the predominance of symptoms of hypoplastic hemopoiesis, namely fatty degeneration of bone marrow with islands of "PH" (PNH) erythroblasts and sideroblasts, hemosiderosis of tissues, positive Desferral test, elevated serum iron, pancytopenia, thrombocytopenic hemorrhagic syndrome, disturbance of $^{59}$Fe utilization in the bone marrow, sequestration of $^{59}$Cr-labeled erythrocytes in the spleen, good tolerance of blood transfusions, favorable result of splenectomy. The symptoms of intravascular hemolysis (hemoglobinemia, hyperbilirubinemia) are insignificant and unstable. Only the Ham and Hartmann-Jenkins tests are constantly positive. On the contrary, the second phase of Strubing-Marchiafava disease is characterized by the predominance of symptoms of intravascular hemolysis, namely erythroblastic transformation of bone marrow with total disappearance of fat and increased utilization of $^{59}$Fe in the bone marrow, significant and stable hemoglobinemia, hyperbilirubinemia, urinary hemolytic syndrome (permanent hemosiderinuria; paroxysmal, mainly nocturnal hemoglobinuria), iron deficiency, negative Desferral test, thrombohemorrhagic syndrome, isolated hemosiderosis of kidneys. Intensification of intravascular hemolysis is observed in connection with blood transfusions and with remedies stimulating blood production. Splenectomy in this phase is not indicated. —G. A.


A boy with steroid-responsive congenital hypoplastic anemia is described. In addition to long thumbs which are triphalangeal, he demonstrates hypertelorism, antimongoloid slant of his eyes as well as retinopathy, delay in closure of the anterior fontanelle, cleft lip and palate, scoliosis, and narrow shoulders. He also has a short webbed neck, abnormal skin pigmentation, and mental and physical growth retardation. Ka rogram was normal. Interestingly the boy's mother has a long triphalangeal left thumb, which may also have been present in her brother and her mother's aunt. —J. B. S.


An infant with plethora developed cyanosis of a finger at 6 hr of age, with progressive blackening of the distal two digits. Despite phlebotomy which decreased the hematocrit from 78% to 65%, loss of part of the finger tip ensued. This is another complication which can arise in neonatal polycythemia. —J. B. S.

LEUKOCYTES


The osmotic release of $\beta$-glucuronidase from polymorphonuclear leukocyte lysosomes was found to be inhibited by catecholamines and cyclic AMP whereas cholinergic agents and cyclic GMP accelerated the release. These actions appeared to be specific for the sympathetic and parasympathetic neurotransmitters and for the two cyclic nucleotides, as phenylephrine and tyramine—both sympathomimetic amines that do not possess a catechol moiety and choline—adenosine-5-monophosphate, and guanosine-5-monophosphate did not modify the lysosomal enzyme release. It is suggested that the antiinflammatory effects of catecholamines seen in various animal models may be due to an alteration of the physical properties of the lysosomal membrane which may influence certain intracellular events such as peripheral migration of lysosomes and subsequent fusion with heterophagic vacuoles leading ultimately to the extracellular release of lysosomal contents. The fact that cholinergic agents elicit an opposing action to that of catecholamines on lysosomes suggests that the autonomic nervous system might play an important role in regulating or controlling the inflammatory process. —M. S.


Polysome OD$_{250}$ profiles of glass column separated normal and leukemic lymphocytes were studied. Contaminating RBCs were hemolysed with NH$_4$Cl prior to culture. Postmitochondrial
supernatants from lymphocytes ruptured by nitrogen cavitation were analyzed in a scaled-down sucrose gradient procedure. Normal lymphocytes responded to phytohemagglutinin stimulation in a few hours with increases in all classes of polysome OD260 absorbances from the low levels found in both normal and leukemic unstimulated lymphocytes. Leukemic lymphocytes which responded to PHA showed delays in both polysome formation and blast-cell production. With leukemic lymphocytes the degree of responsiveness to PHA and the amount of polysome OD260 produced were inversely related to the level of the peripheral WBC counts. Incorporation of (14C)-amino acids at polysomes in both intact cells and in cell-free reactions increased with PHA stimulation and blast-cell production. With both normal and leukemic lymphocytes, increased (14C)-amino acid incorporation was related to increase in polysome OD260. There was no evidence that leukemic polysomes when formed were defective.—J.E.U.


Patients with Hodgkin’s disease treated with total nodal irradiation were evaluated to determine whether splenectomy, performed for clinical staging, influenced their hematologic tolerance to radiotherapy. Eighteen patients underwent splenectomy, while 17 who did not served as controls. Neutropenia and thrombocytopenia severe enough to warrant interruption of therapy were observed in 11 of 17 (64%) controls but in only five of 18 (28%) splenectomized patients. Hematologic depression was generally less severe in postsplenectomy patients, and most severe in controls previously treated with vinblastine and nitrogen mustard. Splenectomized patients with Hodgkin’s disease, even if previously treated with chemotherapy, appear to experience less hematologic depression than nonsplenectomized patients.—J.E.U.


A study of the incidence of Hodgkin’s disease in a small rural township in Ohio since 1960 revealed five diagnosed cases, four since 1968—an attack-rate equivalent to 77 per 100,000, as compared with an expected rate of 4.3 per 100,000. Inquiries among patients, friends, and family members pointed to associations between patients similar to the patterns reported by investigators in Albany, N.Y. This is the first such clustering of Hodgkin’s disease to be reported—i.e., clearly a space-time cluster with the added feature of an association-between-cases clustering. See also letter in Lancet, vol. 1, pages 669-670, 1973.—J.E.U.


Chromosome analyses on multiple tissues from three children with lymphosarcoma revealed a consistent chromosome abnormality for each tumor. In Case 1, direct examination of cells from a supraventricular lymph node revealed a normal cell type and two different main cell types with 47 chromosomes and related cell types with 48 and 49 chromosomes. A normal chromosome complement was found in peripheral blood and bone marrow aspirate. An axillary lymph node, removed at autopsy 5 wk later, was cultured for 11 hr and contained the same five cell types but in different proportions. In Case 2, cells with 46 to 49 chromosomes were found in a tumor involving the kidney. In Case 3, cells with pseudodiploid karyotype were found in a tumor involving ovaries and bone marrow on direct examination and after 24 hr in culture. The karyotype of peripheral blood was normal. The findings in Cases 1 and 3 provided evidence of preservation of the same chromosome abnormalities in different tumor sites within the individual. The chromosomal abnormality was present in short-term cultures in Cases 1 and 3, suggesting that brief periods of culture can be used to increase the number and quality of mitoses. The karyotypes of all three cases were near-diploid or pseudodiploid.—J.E.U.


Radiologic evidence of bone involvement in children with ALL was noted in 21% of 191
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305 patients. There was no prognostic significance whatever to the finding.—J. B. S.


The mother of a male infant with apparent CGD had normal leukocyte bactericidal function, normal leukocyte enzyme levels, normal myeloperoxidase, and normal NBT reduction. The authors speculate that these findings may be due to disproportionate inactivation of CGD-affected X-chromosomes. Because of the probability that different genetic mechanisms are operable among patients with CGD, speculation concerning the significance of the “silent carrier” must remain uncertain.—J. B. S.


The index of activity of alkaline phosphatase of neutrophiles was measured by the score method of Kaplow in 20 untreated patients with early tuberculous infiltrations in the lungs and in 20 healthy subjects. Marked difference was found between the values observed in patients (59.20 ± 25.67) and in controls (25.47 ± 9.27). There was no correlation between alkaline phosphatase scores and neutrophile absolute and differential counts. According to the authors’ opinion increase of the neutrophile alkaline phosphatase score may be considered as an additional index of the active tuberculous process.—M. K.


Alkaline phosphatase activity (AP) of leukocytes was examined in 35 people occupationally exposed to vapors of benzene, toluene, and xylene within or somewhat above the range of acceptable concentrations during 1–20 yr. The results were compared with those obtained in control healthy subjects not exposed to any noxious agents. Significantly decreased mean scores of AP—15.17 ± 3.14 were found (control value 37.61 ± 4.72). Slight decrease of erythrocyte counts and macrocytosis of red blood cells were the only abnormalities in routine examination of the peripheral blood. The author suggests that the determination of AP in leukocytes is a useful test for the prophylactic control of people with occupational exposure to aromatic hydrocarbons.—M. K.


The mean incidence of myelomatosis for North-East Scotland from 1960-1969 was 34 per million per annum. It appears that in this region the incidence and mortality are higher than the national rate and also higher than for comparable Western countries.—J. M. B.


Preliminary evaluation of the usefulness of a new “cytocentrifuge” (Cyto-Centrifuge, Shandon Scientific Co., Sewickley, Pa.) suggests that the instrument may be helpful in preparing better slides of cells from the cerebrospinal fluid, particularly when mononuclear cells are present in small numbers.—J. B. S.

HEMOSTASIS


Von Willebrand’s disease (VWD) is characterized by a family history showing autosomal dominant transmission, a prolonged bleeding time, a reduced plasma Factor VIII concentration which rises slowly after infusion of normal or hemophilic plasma, and various platelet abnormalities including decreased adhesiveness to glass beads and impaired aggregation induced by the
antibiotic ristocetin. In a patient with a lifelong bleeding tendency described in this paper, these findings were present, except that the plasma Factor VIII level was normal. Defective platelet aggregation in response to ristocetin was restored by normal plasma but not by plasma from VWD. The authors postulate that this is a new variant of VWD in which there is a lack of a plasma factor with which ristocetin reacts before it induces platelet aggregation. It may be that Factor VIII has a double activity, one concerned with intrinsic blood coagulation (normal in this case), the other with platelet function (abnormal in this case). *Abstracter’s comment:* The complicated hematologic and genetic problems posed by VWD are further discussed in an Editorial by W.R. Pitney of St. George Hospital, Sydney, Australia in the same journal, 3:305, 1973.—F.W.G.


This study was undertaken to resolve the diversity of opinion concerning the role of cyclic AMP as a possible regulator of platelet aggregation. Cyclic AMP levels in citrated platelet-rich plasma were determined during the course of platelet aggregation induced by ADP, adrenalin, or collagen. The studies were performed in the presence or absence of prostaglandin E, a powerful aggregation inhibitor. Cyclic AMP levels were estimated by a protein-binding assay. Aggregating agents had no consistent effect on basal cycle AMP concentrations. Preincubation of platelet-rich plasma with PGE, resulted in increases in cyclic AMP concentration, and addition of ADP, collagen, or adrenalin to such preparations produced a prompt reduction of cyclic AMP to basal levels. Inhibition of platelet aggregation by PGE, appeared to be mediated by increases in cyclic AMP level. For a given concentration of aggregating agents the strength of this inhibition was related to the size of the increase. The authors found no indication that a lowering of cyclic AMP levels was essential for aggregation to occur. The maximum effect of PGE, on aggregation lagged behind the maximal increase in cyclic AMP and persisted after the cyclic nucleotide levels had declined. Reversal of ADP-induced aggregation by PGE, also appeared to be mediated by increased platelet cyclic AMP. When cyclic AMP levels of platelets deaggregated by PGE, declined reaggregation did not occur.

The authors suggest that cyclic AMP is not a direct inhibitor of the action of aggregating agents on platelets. They speculate that the increases in cyclic AMP may trigger a mechanism which results in a relatively stable inhibitory effect on platelet function.—M.S.

**Collagen Induced Platelet Aggregation: Involvement of an Active Glycopeptide Fragment (α-1-CBS). R.L. Katzman, A.H. Kang, and E.H. Beachey. Veterans Administration Hospital, and Departments of Biochemistry and Medicine, University of Tennessee, Memphis, Tenn. Science 181:670, 1973.**

An examination of the structural features of collagen which might be involved in platelet aggregation was undertaken. Collagen from various phyla and tissues were examined. Contrary to the widely held belief that the tertiary structure of collagen is essential for the induction of platelet aggregation the authors found that purified α-1-chain prepared from denatured sheep-skin collagen aggregates platelets. The activity appears to be confined to a distinct region of the molecule representing less than 4% of the total length of the α-1 chain. Of all the cyanogen bromide peptides of the α-1 chain tested only one, α-1-CBS, was active in platelet aggregation. This glycopeptide devoid of any tertiary structure contains 36 amino acids and one residue of glucopyranosylgalactopyranosyllysine (Glc-Gal-Hyl). Blocking experiments strongly suggest that Glc-Gal-Hyl is one of the structural determinants of collagen-induced platelet aggregation.—M.S.

**Complement Induced Platelet Protein Alterations. T. S. Zimmerman and H. J. Muller-Eberhard. Scripps Clinic and Research Foundation, La Jolla, Calif. Science 180:1183, 1973.**

Activation of complement is known to be able to trigger aggregation of platelets and release of vasoactive amines and has been implicated in the precipitation of intravascular coagulation. The authors report complement-induced alterations in high molecular weight polypeptides residing in the sedimentable portion of human and rabbit platelets. Polypeptides of high molecular weight were deleted from this fraction when intact platelets were subjected to complement action. These polypeptides appear to be distinct from the previously described thrombin-sensitive protein released from platelets after exposure to thrombin.—M.S.

**Influence of Nicotinic Acid on Fibrinolysis in**

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Changes in fibrinogen level and in euglobulin fibrinolysis time induced by a single intravenous injection of 100 mg of nicotinic acid were examined in 20 patients with liver cirrhosis and in two groups, one in which spontaneous fibrinolytic activity exceeding that of controls but failing to rise significantly after injection of nicotinic acid, the second with spontaneous fibrinolytic activity not increased but rose significantly after administration of nicotinic acid. On the basis of limited personal experience the authors suggest that the patients belonging to the first group are candidates for severe bleeding after operations and that the test may be of some prognostic value in this respect.—M.K.


Thrombocytopenia in sarcoidosis may result either from hypersplenism or immunologic destruction. Only in the latter case is there likely to be evidence of significant bleeding. In some patients whose sarcoidosis is not accompanied by portal hypertension, relatively asymptomatic thrombocytopenia may be seen, in others, acute fulminating hemorrhage necessitating steroid and/or surgical therapy is necessary.—J.B.S.


Severe thrombocytopenia with platelet levels of 5000 and less than 5000 per cu mm, respectively, occurred in two young adults with I.M. Each had extensive purpura. Prednisone, 60 mg per day, caused immediate cessation of purpura, and platelets rose to normal levels within 2 wk in both cases.—F.W.G.


Four infants with cystic fibrosis of the pancreas (theretofore unsuspected in three) developed significant hemorrhage into skin, GI tract, and/or CNS. Marked prolongation of the prothrombin time was found, which promptly and permanently responded to vitamin K. The vitamin K deficiency in the infants appeared to be due to administration of vitamin K-deficient artificial formulae, to chronic diarrhea, and/or prolonged broad-spectrum antibiotic therapy.—J.B.S.


Thrombocytopenia was the most common abnormality in coagulation studies performed on 33 patients with cyanotic congenital heart disease. When the hematocrit was above 65%, thrombocytopenia was usually present, and its severity was directly related to the degree of polycythemia. Factor I was normal, and for the most part so were Factors II, VII, VIII, IX, X, XI, and XII. Occasionally mild deficiencies of vitamin K-dependent factors were found. The most consistent coagulation factor deficiency was of Factor V, with the lowest levels present in the most cyanotic patients. Accelerated fibrinolysis was seen in almost half the patients with hematocrit levels over 65%, and in some, increased fibrin degradation products were also present; however the authors...
do not view these changes as evidence of DIC.—J.B.S.

**IMMUNOHEMATOLOGY**


The authors investigated the distribution of blood group factors of the ABO, Rh, MN systems and P factor in 2094 patients with diseases of the blood: 789 patients with leukemia (among them 300 patients with acute leukemia, 257 patients with chronic granulocytic leukemia, and 232 patients with chronic lymphocytic leukemia), 958 patients with different forms of anemia (among them 784 patients with hypochromic anemia, 121 patients with pernicious anemia, and 121 patients with aplastic anemia), 347 patients with hemolytic diseases, 126 patients with hemorrhagic vasculitis, 52 patients with hemophilia. The data obtained were compared with results in healthy persons. All the investigations were carried out among persons of Armenian nationality. The analysis of distribution of blood groups among 44,632 healthy persons gave the following results: blood group O—28%; group A—49.9%; group B—13.3%; group AB—7.9%; Rh(+)—87.8%; Rh(−)—12.2%. Among patients with acute leukemia a considerable percentage (36.4%) were persons with blood group O; in patients with pernicious anemia there was an increase in the percentage of persons with blood groups A and AB (53.7% and 10%, respectively) on account of a decrease in the number of patients with group O (24%); in patients with I.T.P., blood group AB was more frequent (11.9%), and in patients with hemophilia there was a prevalence of group B (17.3%). As to distribution of Rh factor, in acute leukemia and in chronic granulocytic leukemia there was an increase in percentage of Rh negative patients (17% and 18.3%, respectively). The greatest increase in percentage of Rh negative patients was revealed among hypoplastic anemia and hemophilia cases (24.5% and 19.3%, respectively). The mentioned diseases, particularly hypoplastic and aplastic anemia cases (13.2%) were accompanied by a reduction in the agglutination capacity of Rh positive erythrocytes. The incidence of agglutinogens of the MN and P system in hematological patients showed almost no difference from that in healthy persons. 

*Abstractor's comment:* There was a very high frequency of group A that confirms the data obtained by other investigators (A. E. Mourant, 1954). There was a relatively low frequency of Rh(−) persons. The great predominance of blood group A among the patients with pernicious anemia (the same as among the patients with cancer of stomach) also coordinates with data in the literature. The high frequency of Rh(+) persons among the patients with chronic granulocytic leukemia, contrasting with the low frequency of this group among the patients with chronic lymphocytic leukemia, is of great interest.—G.A.


The percentage of peripheral blood lymphocytes forming rosettes with sheep erythrocytes was not significantly different from normal in 207 subjects with rheumatoid arthritis and was not altered by therapy.—J.M.B.


There are at least two in vitro models in which lymphocytes may exert an immunologically specific cytotoxic effect on target cells. The first one involves thymus-derived lymphocytes as the effector cells. Lymphocytes explanted from animals sensitized with allogeneic cells are specifically cytotoxic to cells bearing the sensitizing alloantigens. In the second in vitro model nonimmunized thymus-independent lymphocytes may be cytotoxic to target cells coated with small amounts of antibody to target cells. The effects of colchicine and vinblastine, both plant alkaloids which specifically bind to microtubular protein and cause disaggregation of microtubular subunits, were studied with respect to the two lymphocyte mediated cytotoxicities. In both systems disaggregation of microtubular subunits in effector lymphocytes was found to inhibit their ability to injure target cells. Denteriumoxide, an agent known to stabilize microtubular subunits, was unable to reverse this inhibition.—M.S.
Plasma Therapy in Immunodeficiency Diseases.  

Higher and more prolonged levels of IgG, IgA, and IgM were obtained following plasma infusion into children with immunoglobulin deficiencies than were obtained after serum globulin injections. In several children clinical effectiveness of the plasma therapy appeared to be substantially better than that of the intramuscular gamma globulin. Plasma was obtained by plasmapheresis from one of the parents, and there were no untoward reactions in donors or recipients. There would appear to be several distinct advantages to this therapeutic approach, which I believe merits wider adoption.—J.B.S.


Two antigens that exist in high frequency in tumor tissues of patients with Hodgkin’s disease have been obtained in relatively concentrated form. Extracts of Hodgkin’s spleen tumor tissue, when subjected to chromatography on Sephadex G-200, separate into three major protein peaks of which only the first (peak I) possesses the predominant antigenic activities associated with the disease. Antigenic analysis performed with hyperimmune rabbit antisera obtained after repeated immunizations with peak I proteins demonstrated that this fraction contained both F and S antigens associated with Hodgkin’s disease and small contaminant amounts of an antigen associated with normal lymphocytes. The tissue distribution patterns of the Hodgkin’s disease tumor-associated antigens suggest that they both originate in lymphoid tissues and that the F antigen may represent a product of reactive lymphocytes while the S antigen may be a dedifferentiation antigen expressed in very immature lymphocytes.—J.E.U.


Although pericarditis is common in SLE, massive pericardial effusion as the presenting sign is not. A 15-yr-old girl admitted with complaints of fever, shortness of breath, and nonpleuritic left chest pain, had such pericardial effusion. Diagnosis of SLE was made on the basis of a positive LE prep., and on an ANA titer of 1024. Steroid therapy led to a prompt remission.—J.B.S.


A patient with reticulum cell sarcoma and hypercalcemia had a remission of the hypercalcemia after localized radiation therapy. The tumor and hypercalcemia recurred 2 yr later. Examination of blood and tumor extract from the patient by a sensitive radioimmunoassay for parathyroid hormone failed to detect the hormone. Tumor extract raised serum calcium when injected into parathyroidectomized mice and caused release of $^{45}$Ca from mouse calvaria in vitro. At autopsy the patient had four normal parathyroid glands and no skeletal metastases. The humoral agent responsible for the hypercalcemia in this patient remains to be elucidated.—J.E.U.


The serum transferrin level was determined in 20 donors and 65 patients with viral hepatitis using the method of immunoprecipitation on Partigen-Transferrin plates. Examinations repeated weekly during 9 wk showed in the patients with viral hepatitis a slight increase of serum transferrin level correlating with alanine aminotransferase activity. No correlation could be demonstrated between the severity of the clinical course and the serum transferrin level.—M.K.