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IMMUNE STATUS OF CHILDREN WITH ACUTE LYMPHOBLASTIC LEUKEMIA (ALL) IN PROLONGED REMISSION (CR). N. Dunn, E. Russell, T. Mohanakumar, N. McWilliams and H. Maurer. Med. Col. of Va., Depts. Ped. & Surgery, Richmond, Va.

With the marked improvement in survival in childhood ALL, it is increasingly important to correlate immune status with long-term disease control. 24 ALL patients, 7 in CR for 5-12 yrs. and off therapy for a $\bar{X}$ of 3.5 yrs., and 17 in CR on therapy for a $\bar{X}$ of 2.8 yrs. (3-5 yrs.) were studied.

The mean % of sheep RBC rosettes (T-cell) on therapy (53%) and off therapy (50%) was similar to controls (60%). Complement receptor lymphocytes (B-cell) averaged 25.6 and 15.6% in patients off and on therapy, respectively, a significant difference ($p < .05$), but still WNL. Mouse RBC receptors, detecting a B-cell subgroup, were deficient in 93% of patients on and 40% of patients off therapy ($p < .01$). Stimulation by T (PHA, CON-A) and B (S aureus) cell mitogens was comparable (all $p > .05$) in the 3 groups of children after 3 or 6 days of culture. In 2 patients who relapsed, a marked reduction in all mitogen stimulation was noted at a time when there was no clinical evidence of relapse. None of the patients had cells reactive with either the antisera detecting Ia like or leukemia associated antigens (LAA), including the 2 in the pre-relapse state.

We conclude that these patients in prolonged CR have normal T-cell % and function. Maintenance therapy, however, does reduce a B-cell marker while mitogen response remains intact. Preliminary data suggest that mitogen response may be more sensitive than the reappearance of LAA in detecting impending relapse.

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HYPERKALEMIA IN A CHILD WITH SICKLE CELL ANEMIA AND DIABETES MELLITUS. Bertrand Duval-Arnould, Beatrice C. Lampkin, Paul T. McEnery. Hematology-Oncology and Nephrology Divisions. Department of Pediatrics, University of Cincinnati College of Medicine, Cincinnati, Ohio.

An 18 year old black girl with sickle cell anemia (SCA) who has diabetes mellitus, a high renal threshold for glucose, and unexplained hyperkalemia is reported. The patient was diagnosed to have SCA at 2-8/12 years of age and diabetes mellitus at 11 years of age. During her recent admissions for painful crises her renal threshold for glucose was found to be increased to 350 mg/dl and on several occasions her serum potassium levels were elevated (5.5-8.5 mEq/liter). Ketones were not found in the blood or urine and her blood gases were normal. In an attempt to explain the hyperkalemia and high renal threshold for glucose, sodium and potassium balance and renal function studies were done, as well as an evaluation of her renin aldosterone axis (RAA). The following results were found: (a) normal plasma renin activity and aldosterone at the time of hyperkalemia and under controlled circumstances, (b) slightly positive sodium balance, (c) positive potassium balance, as long as the hyperglycemia was not well controlled, and (d) glomerular, proximal and distal tubular dysfunction. These results can best be explained by lack of insulin and a mild degree of renal insufficiency. This renal insufficiency is mainly secondary to SCA but is aggravated by her diabetes as shown by renal biopsy.

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ACQUIRED APLASTIC ANEMIA: ANTIBODY-MEDIATED ERYTHROPOIETIC FAILURE. Melvin H. Freedman, Erwin W. Gelfand, and E. Fred Saunders. Univ. of Toronto, Hosp. for Sick Children, Dept. of Pediatrics, Toronto, Canada.

Now that marrow transplantation is used for acquired aplastic anemia (AAA), study of mechanisms of marrow aplasia may provide a rational selection of suitable candidates. The red cell failure in AAA was studied in 6 pts by assessing marrow erythroid stem cell colony growth (CFU-E). CFU-E were absent in 5 pts compared to control values (mean $197 \pm 22/10^5$ cells plated). Pts' sera cultured with control marrow to detect inhibitors produced a mean increase in CFU-E of 14% (range 6-24%) compared to normal serum. In contrast, marrow from 1 pt yielded 80 CFU-E/ 10^5 . His serum decreased CFU-E from his own marrow (80 to 37/ 10^5) and from control marrow (122 to 9/ 10^5), an inhibitory effect which was shown to be dose dependent. Peripheral blood lymphs (PBL) separated by Ficoll-Hypaque and co-cultured with autologous or control marrow similarly decreased CFU-E from his marrow (93 to 33/ 10^5) and from control marrow (278 to 198/ 10^5). The inhibition was more marked in co-cultures using a B-cell enriched PBL fraction obtained on an albumin density gradient. Pt's purified IgG demonstrated a dose-responsive suppression of CFU-E from control marrow (102 to 8/ 10^5). We conclude that 1 pt demonstrated immune-mediated red cell failure in contrast to the others. In vitro testing can detect such a pt in whom immunosuppression $\pm$ plasmapheresis may be more appropriate therapy than transplantation.

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INHIBITORY ACTIVITY OF TYPE I & TYPE II INTERFERON AGAINST TRANSPLANTABLE OSTEOGENIC SARCOMA IN MICE. Lowell A. Glasgow, John L. Crane, Jr., Earl R. Kern, and Julius S. Youngner. Univ. of Utah Col. of Med., Salt Lake City, Ut. and Univ. of Pittsburgh School of Med., Pittsburgh, Pa.

Interferon (IF) has been recognized to have antitumor as well as antiviral activity. Strander et al. have reported possible therapeutic effects of human IF in patients with osteosarcomas. To study the antitumor activity of IF under more controlled conditions we have utilized a 239Pu-induced osteogenic sarcoma (OGS) in mice as a model system. We previously reported that growth of murine OGS cells in tissue culture was inhibited by IF. In the present study the effect of IF on the growth of transplantable OGS in mice was determined. Two types of IF were utilized: type I (viral-induced) IF and type II (immune) IF. Mice inoculated sc with 1×10^5 OGS cells were treated daily with 32,000 U type I IF at the tumor site for 7 days. The time of appearance of tumors was increased by approximately 40 days. Utilizing a similar treatment schedule the antitumor effects of type I and type II IF were compared using groups of 10 animals:

Control	100%	mice had tumors by day 16
600 U type I IF	100%	" " " " " day 26
60,000 U type I IF	20%	" " " " " day 80
600 U type II IF	10%	" " " " " day 80

These data extend in vitro studies which demonstrated that OGS cells were inhibited by IF and indicate that: (1) transplantable murine osteosarcomas can be inhibited by local treatment with IF and (2) type II IF was more effective than type I IF in this experimental model.

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FETO-MATERNAL ABO INCOMPATIBILITY. A DISCRIMINANT ANALYSIS TO PREDICT SERUM BILIRUBIN LEVELS IN THE NEONATAL PERIOD. F. Gloria-Bottini, A. Polzonetti, N. Lucarini, R. Palmarino and E. Bottini (Spon. by C.D.Cook). Univ. of Camerino, Dept. of Genetics and Computer Center and Univ. of Rome, Dept. of Pediatrics.

Two series of newborn infants (432 white (W) and 99 Negro (N)), collected at Yale-New Haven Hospital from 1968 to 1971, were studied. Each series, which included only infants ABO incompatible-Rh compatible, was subdivided into 2 groups according to the maximum serum bilirubin level (SBL < 10 mg/dl or > 10 mg/dl) recorded during the neonatal period. A discriminant analysis was performed in males (m) and females (f) separately on the following 8 variables: gestational age, birth weight, birth order, ABO maternal phenotype, type of feto-maternal ABO incompatibility, Coombs test, presence of I^B (ABO) and PI^f (placental alkaline phosphatase) factors, dose of PI^f - PI^f factors. The percent of correct classifications was 83% in Wf, 73% in Wm, 77% in Nf and 75% in Nm. By the present method of discrimination an early discharge of more than 50% of ABO incompatible infants with a very low risk of hyperbilirubinemia might be allowed. The analysis showed a remarkable influence of sex and race on the pattern of relationships between the SBL and the set of variables examined.

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ABNORMAL MEMBRANE FUNCTION OF PHAGOCYTES FROM VITAMIN E DEFICIENT RATS. Richard E. Harris, Laurence A. Boxer, and Robert L. Baehner. Indiana University School of Medicine, J. W. Riley Hospital for Children, Department of Pediatrics, Indianapolis.

Human polymorphonuclear leukocytes (PMN) rich in Vitamin E have enhanced rates of chemotaxis and phagocytosis. To determine the effect of Vitamin E deficiency, weanling rats were nutritionally deprived of Vitamin E for two months. Both PMN and peritoneal macrophages (PM) were purified from Ficoll-hypaque gradients. Serum Vitamin E levels were 5.1% of control values. Chemotaxis was markedly impaired by both PMN and PM with values being 68% and 44% of control respectively. Ingestion of opsonized paraffin oil droplets was 44% and 43% of control by PMN and PM respectively. In contrast, those functions and metabolites reflecting an intact microtubule system were normal; e.g. degranulation, as quantitated by the release of the lysosomal enzyme β -glucuronidase into the extracellular media, capping as determined by the lateral migration of surface glycoprotein receptors into polar caps, and glutathione levels. On the other hand, the quantity of lipid membrane polyunsaturated fatty acids that were oxidized as assayed by the formation of malonyldialdehyde was increased by 1.5 and 2.3 fold in PMN and PM compared to control respectively. These studies indicate that a deficiency in Vitamin E leads to altered perturbation of membrane function and lipid chemistry without altering cytoplasmic microtubule function and glutathione content.