basis of these results it is proposed that the impaired erythropoiesis in marasmus is not caused by a specific nutritional deficiency. The 'anemia' that is present represents an adaptation to the reduced metabollic demands of these undernourished subjects. The proliferative activity of the erythropoietic tissue may provide a sensitive measure of response to nutritional rehabilitation. (APS)

57 *Autoimmune Hemolytic Anemia in a Patient with Congenital Hypogammaglobulinemia.* HOWARD A. PEARSON, JOHN B. ROBBINS and RICHARD G. SKINNER*, Univ. of Florida, College of Medicine, Gainesville, Fla., and Children's Medical Group, Jacksonville, Fla.

Since the clinical findings of hypogammaglobulinemia (HGG) are explained by a deficient synthesis of circulating antibody, the development in such a patient of autoimmune hemolytic anemia (AHA), due to excessive production of a specific abnormal antibody is of interest. A six-year-old boy with HGG, of the sex linked variety, developed Coombs' positive AHA despite profound deficiencies of immunoglobulins (IgG 100, IgM 18, IgA 0 mg %). He had never received transfusion and penicillin had not been given for 3 months. Lymph node biopsy did not show malignant changes and cytomegalic virus could not be cultured. Direct and indirect Coombs' tests, using gost anti-human IgG and IgM sera, were positive. Eluted antibody was an IgG globulin. The serum reacted with all cells of a large panel but tests by Ortho Labs showed specificity against the public Rh antigen designated LW. The commercial gamma globulin used for therapy had no anti RBC activity. He has responded well to corticosteroids for 6 months, but has relapsed twice when this was discontinued. Although three adults with acquired HGG have developed AHA, this case represents the first instance in a patient with congenital HGG. All of the adults have had lymphoma or leukemia. Possibilities to explain this combination include graft vs host reaction with secondary 'runt' disease. Although unlikely in view of the patients age and lack of blood transfusions, investigations to examine this possibility are in process. Alternatively lymphoma may be present, and, if this proves true, speculations both on the nature of lymphoma and hypogammaglobulinemia are possible. (SPR)

58 *Inappropriate Glucose Consumption by the Erythrocytes of the Premature Infant.* FRANK A. OSKI, ERNESTINE P. BRIGANDI* and CHARLES F. SMITH*, Dept. of Pediatrics, Hosp. of Univ. of Pa. and Univ. of Pa. Sch. of Med., Philadelphia, Pa.

Although it is recognized that the red cells from premature infants consume more glucose than do the cells from adults no attempt has been made to determine if this increased glucose consumption is proportional to the younger mean age of the red cell population. Specific gravity separation revealed that 50 % of the red cells from the premature infant are as young as the youngest 10 % from adults. Red cell glucose consumption and lactic acid production was measured in 15 premature and 23 term infants, 23 adults and 12 subjects with reticulocytosis (mean 6.8 %). Glucose consumption was related to cell age expressed as a function of red cell G-6-PD and glutamic-oxaloacetate transaminase (GOT) levels, both enzymes particularly GOT showing marked variation in red cell populations of differing ages. Red cell glucose consumption expressed as μM/ml RBC's/h/100 GOT u. averaged 0.121 in the adults, 0.094 in the group with reticulocytosis, 0.086 in the term infants and only 0.068 in the prematures. When glucose consumption was expressed as a function of 100 G-6-PD units values averaged 1.01 in the reticulocytosis group, 0.86 in the adults, 0.80 in the term infants, and 0.64 in the prematures. Both GOT/G-6-PD and lactate/glucose ratios were similar in the 4 groups. GOT averaged 2928 u/10^{10} RBC's in the reticulocytosis group, 2904 in the prematures, 2499 in the term infants, and 1196 in the adults. The red cells of the premature infant appear to consume less glucose than would be expected from their young cell age. This finding suggests that 1 or more regulatory steps in glycolysis may be operating at different K_m's or that these cells utilize other substrates as a source of energy. (SPR)

59 *Prevention of Hyperbilirubinemia of Prematurity by Phototherapy.* JEROLD LUCEY, MARIO FERREIRO* and JEAN HEWITT*, Department of Pediatrics, University of Vermont College of Medicine, Burlington, Vt.

The ideal treatment for hyperbilirubinemia of prematurity would be a safe and simple method for preventing its occurrence. CREMER *et al.* (1958) first demonstrated that serum bilirubin concentrations of newborn infants can be reduced by exposure to light. This treatment has not been widely used because of doubts as to its effectiveness and concern for the possible toxicity of the photochemical decomposition products of bilirubin. Recent experimental evidence indicated that these products are non-toxic. A controlled clinical trial has been carried out among 59 premature infants to test the effectiveness of artificial blue light in *preventing* hyperbilirubinemia of prematurity. Treated infants were placed in light from 12 to 144 hours of age and serial bilirubin determinations were carried out. The control and treated groups were comparable with respect to birth weight, gestational age, fluid intake and weight loss. The results are summarized below and indicate a statistically significant difference between the groups. Additional observations on the effect of phototherapy on serum albumin. H.A.B.A. dye binding capacity, free fatty acids and uric acid will be presented. (APS)

	Control	Light Treatment
Number of infants	31	28
Serum bilirubin—Mg %		
1st day	4.0±0.5	3.7±1.4
2nd day	7.5±3.3	5.3±1.4
4th day	9.7±3.5	5.4±1.9
6th day	8.2±5.6	4.9±2.2

60 *Indications for and Consequences of Intrauterine Transfusions.* PAUL JOHNSON*, ALAN MARGOLIS*, SUSIE FONG*, RODERIC H. PHIBBS* and WILLIAM H. TOOLEY*, Department of Pediatrics, Obstetrics, and the Cardiovascular Research Institute, Univ.-California-S.F. Medical Center, San Francisco, California (introduced by M.M. Grumbach).

We performed spectrophotometric analyses (technique of Liley) on 595 amniotic fluid specimens from 310 Rh-sensitized women; 1 or more specimens from 84 women had concentrations of pigment above levels which are generally considered to predict, with 95 %

confidence, intrauterine death before 34 weeks gestation; 30 of these 84 fetuses were not transfused and 18 were liveborn (gest. age 34 weeks, SD 1.9,) of whom 9 (30 %) survived. We transfused 54 infants in utero (IUT) on 96 occasions. Of these, 25 had marked ascites at the time of transfusion: 13 died in utero, within 48 h of the procedure; 11 were born alive (gest. age 32.2 weeks, SD 2.1), were hydropic and died of the respiratory distress syndrome (RDS) in the neonatal period; 1 (4 %) was not hydropic at birth and survived. 29 did not have ascites at the time of transfusion: 13 died in utero, 12 within 48 h of the procedure; 6 were borne alive (gest. age 33 weeks; SD 1.3) and were hydropic, of whom 4 survived and 2 died with RDS; 10 were born alive (gest. age 34.6 weeks, SD 1.6) without hydrops, 1 died with RDS and 9 survived (45 %). We conclude that serial spectrophotometric analysis of amniotic fluid is required to adequately establish the need for IUT since single or paired analyses may be misleading, and that an intrauterine transfusion, particularly in an infant with ascites or performed earlier than 26 weeks gestation carriers a high mortality. (Supported by USPHS Grant HE-06285) (APS)

61 *Quantitative Aspects of Sensitivity in Allergic Children.* CHARLES D. MAY, JANE CHENG* and MARGARET LYMAN*, New York Univ. School of Medicine, New York, N.Y.

Procedures were devised for quantitative chemical assay of histamine released by antigens from leukocytes separated from a 10-ml sample of blood. Nine concentrations of antigen can be utilized in each examination to find the amount required for maximum release of histamine or dose response. Studies have been conducted with a variety of antigens for comparison with clinical manifestations and wheal and flare dermal reactions to the antigens. Also the procedures have been employed to measure the capacity of the sera of sensitive persons to inhibit histamine release with specific antigens (presumably by antibodies) and to follow fluctuations in this capacity and in the sensitivity of leukocytes during injection therapy with antigenic extracts. Data have been accumulated from study of over 100 children, including 30 receiving injection therapy. Histamine is released from leukocytes of sensitive subjects by antigens specifically, in agreement with wheal and flare dermal reactions, and the leukocytes of normal non-allergic individuals are unaffected. Sera of normal persons enhance histamine release but the sera of allergic children inhibit histamine release by the antigens specifically involved. During injection therapy the sensitivity of leukocytes to release of histamine by antigen and the capacity of the patient's serum to inhibit histamine release may vary independently. The net effects are ascertained by determining the amounts of antigen required for release of 50 % of the total histamine in the cells in the presence of the subject's serum in contrast to normal serum. This comparison affords an objective index of any influence of injection therapy on sensitivity, and an objective means of grouping patients by immunochemical response before undertaking clinical appraisals. (APS)

62 *Hereditary Splenic Hypoplasia.* SHERWIN V. KEVY*, MELVIN TEFFT*, GORDON VAWTER* and FRED S. ROSEN, Children's Hosp. Med. Ctr. and Harvard Med. Sch., Boston, Mass.

The one boy and two of three girls in a consanguineous kindred have exhibited undue susceptibility to invasive infections with *Hemophilus influenzae* and pneumococci. One of the affected siblings died of overwhelming *H. influenzae* type B sepsis and was found at autopsy to have a minute spleen. No other anatomic abnormalities were present. The two affected live siblings were each shown to have no demonstrable splenic tissue by scintillation scanning of the abdomen following intravenous injection of colloidal Au^{198}. Normal splenic tissue was demonstrable in both parents by this technique. Examination of the peripheral blood of affected offspring revealed the presence of Heinz and Howell-Jolly bodies. The antibody response to subcutaneous injection of tetanus and diphtheria toxoids and typhoid bacilli was normal. Their red cell survival and response to intravenous particulate antigens are under investigation and the results will be reported. (SPR)

63 *Sex Linked Recessive Hereditary Thrombocytopenia with Immune Globulin Abnormalities. A Form of Wiskott-Aldrich Syndrome?* LUIS CANALES and ALVIN M. MAUER, Dept. of Pediat., Univ. of Cincinnati, Ohio

A family was studied in whom hereditary sex-linked recessive thrombocytopenia was associated with immunologic abnormalities suggestive of relationship to Wiskott-Aldrich syndrome. Twenty-one male and 10 female members of 4 generations were included and 7 thrombocytopenic males found in a sex-linked recessive pattern of inheritance. Platelet counts in affected males ranged from 8,000 to 57,000/mm^3. Bleeding symptoms were mild except in one where recurrent epistaxis led to splenectomy at age 17 years. There was no history of eczema or increased susceptibility to infection. In 5 affected members studied isohemagglutinins were either absent or significantly decreased in titer. On immunoglobulin quantitation, increased levels of IgA were found in 4 of 5. IgM and IgG levels were normal in all. In one affected male, lymphocyte response to phytohemagglutinin was tested and found normal. All unaffected members, including carrier females, had normal platelet counts, isohemagglutinins and immunoglobulins. The absence of significant clinical history of infection or eczema may not preclude the diagnosis of Wiskott-Aldrich syndrome. The finding of detectable isohemagglutinin levels in 2 and normal IgA levels in one affected males indicates variability of severity within the family. All patients suspected of having sex-linked recessive thrombocytopenia should be studied for coexistent immunoglobulin defect. (SPR)

64 *Serum α-Fetoprotein Synthesis in the Human and Rat Fetus and its Inhibition in the Rat.* DAVID GITLIN and MARY BOESMAN*, Univ. of Pittsburgh Sch. of Med., Pittsburgh, Pa.

The serum of the human conceptus contains α-fetoprotein, a protein not found in the serum of the pregnant woman. Concentrations of α-fetoprotein may be low in infants born after premature spontaeous labor, suggesting that fetal synthesis of the protein in these instances is inhibited some days or weeks prior to the actual onset of labor. In the present study, selected tissues from human embryos of 6 to 9 weeks' gestation and from rat fetuses of 15 days' gestation were incubated with C^{14}-amino acids. Immunoelectrophoresis of the culture fluid followed by autoradiography revealed that α-fotoprotein was synthesized in human liver, rat liver and rat yolk sac, but not in any of the other tissues examined; human yolk sac was not studied. Serum