860 DIAGNOSIS OF IRON DEFICIENCY IN ANEMIC ONE-YEAR-OLD INFANTS: THE LIMITATIONS OF LABORATORY TESTS IN PREDICTING RESPONSE TO IRON TREATMENT. Jerry D. Reeves, David A. Driggers, Edward Y.T. Lo, Peter R. Dallman. David Grant Med Ctr., Dept. of Peds., Travis AFB, CA., and Univ. of California San Francisco, Dept. of Peds., San Francisco.

The purpose of this study was to determine the value of combining laboratory tests of iron status in predicting a rise in hemoglobin (Hb) in infants undergoing a therapeutic trial of iron. Screening for anemia was performed on capillary blood of 1128 healthy one-year-old infants of air force personnel. The 25% who had Hb < 11.5 g/dl were asked to return for tests on venous blood before and again after 3 mo of therapy. Of the total of 188 infants completing therapy, 66 (35%) had a rise in Hb $\geq$ 1.0 g/dl and were designated responders. Each additional laboratory test on venous blood showed a marked degree of overlap in results on responders vs non-responders. We used MCV < 70 fl, erythrocyte protoporphyrin > 3 µg/g Hb, transferrin saturation < 10%, and plasma ferritin < 10 µg/l as cutoff values. For each individual test, about half of those with abnormal values had a response. However, it was disturbing to find that for any given test, half to two-thirds of the responders had a normal value. Because of the difficulty in distinguishing responders from non-responders and the simplicity, low cost, and low prevalence of side effects, we favor the use of a therapeutic trial based on only a low Hb in similar high-risk populations. Further costly workup could then be reserved for the small number of infants who have unexplained anemia (Hb < 11.0 g/dl) after a therapeutic trial.

861 PNEUMOCOCCAL IMMUNIZATION OF PATIENTS WITH SICKLE CELL DISEASE (SCD): REACTIONS AND ANTIBODY (Ab) RESPONSE. Jose G. Rigau Perez, Gary D. Overturf, Darleen Powars, and the SCD Pneumococcal Vaccine (PV) Collaborative Study Group. Univ. of So. Calif. Sch. of Med., LAC-USC Med. Ctr., Dept. of Ped., Los Angeles, Ca.

Patients with SCD (n=174) immunized with PV had a high rate (70%) of mild reactions, primarily at the site of injection and directly associated ($p < 0.01$) with the level of preimmunization pneumococcal IHA Ab titer. Ab response to different antigens followed 3 patterns: poor or good response regardless of age at immunization and improving response with advancing age at immunization; increase in Ab was strongly correlated ($p < 0.0005$) with increasing level of preimmunization Ab.

In 24 months of surveillance 3 episodes of type 23 pneumococcal sepsis were documented in study patients: 2 children under age 30 months (incidence, 4.40/100 patient years in children age 1-4 years) and a child age 5.6 years (0.66/100 patient years in children $\geq$5 years). The incidence of pneumococcal sepsis among nonimmunized children age 1-4 years followed at Los Angeles County-University of Southern California SCD Center was 6.20-9.03/100 patient years.

Therefore, anamnestic response seems to contribute strongly to the enhanced Ab response observed in older children. Only modest vaccine protection may be expected among children with SCD who receive a single dose of PV.

● **862** IN VITRO STUDIES OF THE PATHOGENESIS OF TRANSIENT ERYTHROBLASTOPENIA OF CHILDHOOD (TEC). A.Kim Ritchey, Nicholas Daniak, Ronald Hoffman (Spon. by Diane Komp), Yale U. School of Med., Depts. of Ped. and Med., New Haven, CT.

To explore the nature of the defect in TEC, we utilized the plasma clot culture technique to study the effect of patients' serum on the proliferation of erythroid stem cells in vitro. Six patients with TEC, ages 1-4 yr., presented with an average hemoglobin of 6.2gm/dl and reticulocytopenia. Bone marrow (BM) aspiration revealed decreased or absent erythroblasts. Recovery was evident by 2 weeks in all patients. When $6x10^4$ BM mononuclear cells obtained from 4 patients at the time of diagnosis were plated with 2 I.U. of erythropoietin, 1 patient had increased numbers (159) of CFU-E-derived colonies, 2 had decreased numbers (2;18), and in 1 patient there was none (N1:58±6). Addition of 10% patient's serum to this syngeneic system resulted in 100% inhibition of erythroid colony formation in the patient with increased numbers of CFU-E, no effect on those with decreased colonies, and increased colony formation in the patient with absent CFU-E's compared to controls. When serum from the 2 other patients was added to allogeneic BM cells in culture, there was 58 and 81% inhibition of CFU-E-derived colony formation compared to controls. Study of recovery serum from the 3 patients with inhibitors revealed loss of inhibitory activity. We conclude that while TEC has a uniform clinical presentation, there are at least two pathogenetic mechanisms:(1)a serum inhibitor directed against erythroid stem cells and (2)an abnormality of erythroid stem cells either in number or in responsiveness to erythropoietin.

● **863** DIFFERENTIAL EFFECTS OF FOLINIC ACID (FA) AND GLYCINE, ADENOSINE, THYMIDINE (GAT) ON METHOTREXATE (MTX) TOXICITY IN CULTURED HUMAN CELLS. David S. Rosenblatt, V. Michael Whitehead, Nora V. Matiaszuk, Angela Pottier, Mary-Jane Vuchich and Denise Beaulieu. McGill University-Montreal Children's Hospital Research Institute, Department of Pediatrics, Montreal, Quebec.

Culture medium containing GAT protects human fibroblasts from MTX toxicity but does not prevent the accumulation of MTX polyglutamates. FA competes with MTX for transport into cells and when added to the medium along with MTX prevents the accumulation of MTX polyglutamates. Confluent fibroblasts were co-incubated in MTX (10 µM) plus either GAT (glycine 0.67 mM, adenosine 37.5 µM, thymidine 41.3 µM) or FA (10 µM) or in MTX alone for 16 h. The cells were then re-fed MTX-free medium lacking all of the additions but containing 2.1 µM [^{3}H] deoxyuridine and the incorporation of label into a trichloroacetic acid precipitate was determined. Untreated cells incorporated 122.9±23.1 nmoles dU/g protein/24 h (nm/g/24 h, n=3, $\bar{x}$±S.D.). Cells preincubated in MTX (10 µM) alone incorporated 0.82±0.21 nm/g/24 h (n=3). Cells preincubated in MTX (10 µM) + GAT incorporated 1.11±0.18 nm/g/24h (n=3), whereas cells preincubated in MTX (10 µM) + FA (10 µM) incorporated 210.7±11.6 nm/g/24 h (n=3). Thus folinic acid reverses the effect of MTX when present along with MTX in the preincubation medium. GAT allows normal cell growth when present along with MTX in the preincubation medium. However, upon removal of GAT and MTX from these cells after a preincubation in both, cell growth and DNA synthesis remain suppressed.

864 CHILDHOOD ACUTE LYMPHOBLASTIC LEUKEMIA WITH B-CELL PHENOTYPE (B-ALL). E.C. Russell, T. Mohanakumar, N.B. McWilliams, N.L. Dunn, H.M. Maurer, Medical College of Virginia, Departments of Pediatrics and Surgery, Richmond, VA

B-ALL is uncommon in children and is associated with a poor prognosis. No distinctive clinical features have been identified. At present B-ALL is defined immunologically by the presence of surface immunoglobulins (sIg+) on leukemic lymphoblasts. Since 1976 6/56 (11%) of our newly diagnosed ALL patients have been sIg+ and their data are presented below.

Pt.	Age/Race/Sex	Initial WBC	sIg+ blasts	PNA	Status
1	4 mo WM	246 x 10^3/mm^3	55%	-	NED at 36+ mo
2	2 yo WF	101	80%	-	NED at 29+ mo
3	16 yo WM	42.1	38%	+	Relapsed 19 mo
4	9 yo WF	410	100%	+	Died 3 d
5	7 yo WM	11.2	41%	+	Relapsed 19 mo
6	11 yo BF	89	73%	N.D.	Relapsed 17 mo

Blasts from all six patients expressed Ia-like antigens, a normal B cell alloantigen; 2/6 reacted with rabbit antiserum specific for human thymic lymphocytes; and 1/6 had a common ALL antigen characteristic of null cell ALL. Receptors for the lectin peanut agglutinin (PNA) were present on 3/5 tested and these 3 patients have died. 2/5 did not express PNA receptors and remain in remission at 36+ and 29+ months.

We conclude that: (1) B-ALL is immunologically heterogenous with some patients simultaneously expressing a common ALL antigen or certain T cell characteristics and (2) the presence of receptors for PNA appears to have prognostic significance in B-ALL.

865 NUTRITION AND POOR GROWTH IN PRE-ADOLESCENT CHILDREN WITH SICKLING DISORDERS. Marie O. Russell, Amelia Finan, Sharon R. Moskowitz, Melanie A. Elmer and Frances M. Gill, (Spon. by Elias Schwartz). University of Pennsylvania School of Medicine and The Children's Hospital of Philadelphia, Department of Pediatrics, and Drexel University, Department of Nutrition and Food Sciences, Philadelphia.

Of 170 sickle cell patients 2-12 years old, 22% had height and /or weight <5%ile. Growth and nutrition studies were done on 20 growth-retarded patients (GR) and 20 growth-normal patients (GN) matched for age, sex, hemoglobin disorder, and hematologic values. Selected tests were done on 10 control patients (C). Bone age was delayed in 5/16 GR and 5/12 GN. Somatomedin C (Som C) levels were below age-normals in 7/18 GR and 9/18 GN. Serum zinc levels were <70µg/dl in 32% of GR, 63% GN, and 10% C. Prealbumin levels were normal in all, suggesting that protein malnutrition was not present. Vitamin A (Vit A) levels <20µg/dl were found in 70% of GR, 47% GN, and 20% C. The mean Vit A level in C was higher than in GR ($P = .004$) or GN ($P = .017$). Retinol binding protein (RBP) was normal in all but <2.0mg/dl in 20% of GR and 5% of GN. The mean RBP was lower ($P = .004$) in GR than in GN; both means were significantly lower than in C. RBP and Vit A levels correlated well in C ($r = .81$) but poorly in sickle cell patients. Zinc, Som C, and bone age were frequently abnormal in sickle cell patients but did not correlate with growth status. Vit A and RBP were more often abnormal in GR than in GN. While zinc and Som C do not seem to cause poor growth, other nutritional factors, such as Vit A, may be important.