A COMPARATIVE ANALYSIS OF SEROLOGIC ASSAYS FOR PERINATAL CYTOMEGALOVIRUS (CMV) INFECTIONS. Alex Tsiantos, Sergio Stagno, David W. Reynolds, Walter K. Long, David A. Fuccillo, and Charles A. Alford, Univ. of Ala. in Birmingham, Sch. of Med., Dept. of Ped. and NIH, Bethesda, Md.

Complement fixation (C-F) antibody test has been commonly employed to assess the role of CMV in human disease. But results have been controversial for unknown reasons. To determine the adequacy of this approach, C-F, indirect IgG fluorescent (FA), and hemagglutination (IHA) antibody responses were determined serially in 33 congenitally and 17 natally infected infants between birth and 5 years (mean ages 31 and 15 months, respectively). Both FA and IHA were far more sensitive for detection of CMV antibody throughout the study interval with AD-169 as antigen. The FA and IHA levels were high and remained significantly elevated from birth onward in spite of slight waning. In contrast, C-F levels waned unpredictably and rapidly in the first year; low levels which fluctuated between + and - were observed in the latter half. Thus only FA and IHA consistently monitored the continuing active viral infection in both groups. In natal infections, IHA proved the better monitor for infant antibody production in the presence of maternal antibody, probably due to its increased responsiveness to IgM antibody moieties. The data indicate that both FA and IHA are more appropriate diagnostic and sero-epidemiologic tools for study of perinatal CMV infections. In fact, validity of the results of prior sero-epidemiologic studies using C-F tests should be reexamined.

MYCOPLASMA PNEUMONIAE (M. Pn.) IN A PEDIATRIC TUMOR CLINIC POPULATION. Leonard B. Weiner and Gerald M. Vladimer (Intro. by Frank A. Oski). State University of New York, Upstate Medical Center, Syracuse, New York.

M. Pn. screening was undertaken in a tumor clinic population because of the high incidence of symptomatic respiratory illness. Forty-five patients (Gp. I), ages 18 mos.-22 yrs., with neoplastic disease on immunosuppressive therapy, were cultured over a 6 mo. period and compared to a general pediatric out-patient population (Gp. II). The positive isolation rate from throat swabs was 33/45 (73%) in Gp. I and 23/55 (42%) in Gp. II. In Gp. I during this period, 13/33 remained free of respiratory symptoms and had negative chest x-rays; 15/33 had pharyngitis, rhinitis or cough; 15/33 had fever; 8/33 had x-rays positive for pneumonia; 7/33 had acute otitis media and 2/33 had conjunctivitis. Of the 12 culture negative patients in Gp. I (2-5 cultures/patient), 7/12 were well; 3/12 had URI; 1/12 had fever; and 1/12 had a positive x-ray for pneumonia. There were 8/33 patients in Gp. I who remained positive for at least 3 mos. with intermittent symptoms and mean H.I. titer rises of 2.5 fold compared to 1.3 fold in the remaining 25/33 short-term positive patients ($p = 0.04$). Absolute white counts and specific chemotherapeutic agents did not appear related to incidence or course of infection. Erythromycin therapy produced definite clinical improvement in 4/14 patients with M. Pn. M. Pn. appears to be an agent of high prevalence in patients with malignancies and is difficult to eradicate.

PERTUSSIS SYNDROME AND ECHOVIRUS 6(E6) INFECTION IN A FAMILY. Catherine M. Wilfert, Ziad H. Idriss, Robert M. Fineman, Dale L. Kessler, and Joan Z. Smith. Intr. by Samuel L. Katz, Dept. Ped., Duke Univ. Med. Ctr., Durham, N. C.

Isolation of E6 from the nasopharynx (N-P) of a 5 month old unimmunized child (patient #1) hospitalized with clinical pertussis was accompanied by a fourfold rise in neutralizing (N) antibody to this virus. (Table) The illness occurred in Aug. and Sept., produced significant symptoms for 4 weeks, and was accompanied by an absolute lymphocytosis. B. pertussis was not isolated from her N-P and no pertussis agglutinins were demonstrated.

13 of 15 family members ranging in age from 1-48 yrs reported clinical illness with prominent respiratory symptoms during the 2 months preceding hospitalization of the index case. A 4 yr old immunized cousin (patient 2) with paroxysmal cough and cyanosis also had E6 isolated from her N-P. 8 of 9 family members demonstrated serological evidence of infection with E6 but not with B. pertussis. Isolation of E6 from 2 patients and antibody titers in 10 persons associate E6 with the clinical illness in this family.

Table. Virus isolation and N antibody in 2 patients

Patient	E6	Serum (9/18/73)	Serum (10/16/73)	Serum (1/10/74)
1	N-P	1:128	1:128	1:512
2	N-P	1:128	1:128	1:256

EVALUATION OF CORTICOSTEROID THERAPY IN EXPERIMENTAL DIPHTHERIA. Ronald G. Williams, Clarence Wilson, James W. Bass, and Albert I. Oda, Dept. of Pediatrics, Tripler Army Medical Center, Honolulu, Hawaii.

Corticosteroids have been used in the treatment of diphtheria but their effectiveness is questionable. A study was performed to evaluate the effects of corticosteroid therapy of diphtheria toxicity in an experimental animal model. 126 pure bred Walter Reed strain guinea pigs were divided into six groups, randomized according to weight and sex. Each group was then inoculated (I.D.) with 0.1 ml of two-fold dilutions of purified diphtheria toxin, ranging from 1/40 to 1/1280. Half of the animals in each group were then inoculated (I.M.) in the opposite extremity with 5 mg of cortisone acetate and daily thereafter for 7 days. All were observed daily for 14 days for signs of illness, weight loss, reaction at the site of injection, and survival. The LD_{50} and untreated animals was a 1/227 (±205-251) dilution of toxin; it was 1/170 (±154-189) in treated animals. Treated animals appeared less ill, lost less weight, and there was less tissue injury at the site of toxin injection compared to controls. Corticosteroid therapy favorably modified the course of illness to some degree in this experimental animal model. A similar study evaluating the effects of corticosteroids in modifying diphtheria cytotoxicity in tissue culture showed no difference between treated and untreated controls indicating no benefit at the cellular level.

AMANTADINE FOR THE PROPHYLAXIS OF INFLUENZA IN PATIENTS WITH CYSTIC FIBROSIS. Peter F. Wright, Kon Taik Khaw, Michael N. Oxman and Harry Shwachman. Children's Hosp. Med. Ctr. and Harvard Med. Sch., Dept. of Pediatrics, Boston.

From January to March 1973, amantadine or placebo was administered in a double-blind fashion to 153 patients with cystic fibrosis, to evaluate its prophylactic efficacy against the A2/England/72 strain of influenza and its toxicity in this group of patients. Influenza vaccine containing A2 (Hong Kong) antigens had been administered to 59 patients during the previous 4 months. Side effects were slightly less common in the control (23) than in the treated (32) group, and only two patients withdrew from the study because of them. Of 38 episodes of acute respiratory illness occurring during the 2 month study period, 21 were in patients receiving amantadine. Their severity was not significantly different in treated and control children. Sera from all patients were tested for antibodies to 5 influenza strains. Only 9 patients had evidence of A2/England/72 infection. Three of the 9 had received amantadine and 4 had received influenza vaccine. All 3 asymptomatic patients had received influenza vaccine. While the low incidence of influenza precluded an evaluation of the efficacy of amantadine, vaccination with A2 (Hong Kong) vaccine appeared to offer protection against illness caused by the A2/England/72 strain. Further serologic studies of the 38 patients with acute respiratory illnesses suggest that viral infections may be important causes of exacerbation of respiratory disease in patients with cystic fibrosis.

METABOLISM

BIOCHEMICAL AND MORPHOLOGIC OBSERVATIONS IN SEVERE INFANTILE FRUCTOSE INTOLERANCE. Philip Bagnell, George Hug, Linda Walling and William K. Schubert. The Children's Hospital Research Foundation, Cincinnati, Ohio.

A white boy was healthy until age 6 wks. when CHO in his diet was changed from lactose to sucrose. Abruptly, he developed vomiting, jaundice, hypoglycemia and SGOT > 2000 U. The fulminant course subsided with elimination of dietary fructose. Liver biopsy specimens failed to decarboxylate fructose-^{14}C, and the diagnosis of fructose intolerance was confirmed by demonstrating less than 10% of normal hepatic aldolase activity (with fructose-1-phosphate or fructose-1,6-diphosphate as substrate). Glucose-6-phosphatase was 6x normal and phosphorylase 2x normal. Light microscopy revealed portal fibrosis, ductular proliferation, pseudoacinous formation and mild inflammatory changes. Mildly fibrotic changes persisted after 5 months. Electronmicroscopy showed hypertrophic endoplasmic reticulum, marked focal cytoplasmic degeneration, stacks of abnormal membranes, many unusual lipofuscin bodies and small α-glycogen particles. We found fructose-1-phosphate (but not galactose, fructose, galactose-1-phosphate or fructose-1,6-diphosphate) to be a competitive inhibitor of rabbit muscle phosphorylase b ($K_i = 3 \times 10^{-3}$M) and human liver phosphorylase ($K_i = 7 \times 10^{-3}$M); K_m of both enzymes for glucose-1-phosphate was of the same magnitude. Thus, glycolysis may be inhibited and hypoglycemia may result after fructose ingestion when intracellular fructose-1-phosphate would be elevated. (Supported by NIH grant RR 00123 and RR 05535)