823 BACITRACIN DISC RESISTANT BETA HEMOLYTIC ORGANISMS FROM PATIENTS WITH PHARYNGITIS. Dorothea Vail, Gilbert Mellin, Katherine Sprunt, Columbia Univ. Coll of Phys. and Surg., Dept. of Pediatrics, Div. of Infec.Dis. NYC

Many patients are known to be treated unnecessarily for Group A streptococcal (strep) pharyngitis by pediatricians who fail to include bacitracin discs on culture plates. In our experience an average of 44% of beta hemolytic (hemol) colonies (col) from throat cultures plated on sheep blood agar and incubated anaerobically are bacitracin resistant. To determine what the resistant organisms are that may trigger unnecessary therapy and the role of Group A strep in this population, beta hemol col from cultures in which disc resistant col predominated or were present in high proportions were isolated and identified. In the 9 month interval studied there were 9075 cultures. The 18% containing disc sensitive populations were considered Group A. 138 cultures with disc resistant populations predominant or in high proportion were studied. In the first group of 41 cultures 15% were enteric bacilli. There were eliminated subsequently because enteric bacilli in high proportion can be identified by odor. Considering all other organisms, 82% were strep, 14% were alpha hemol on replate, 3% were staphylococci and one culture yielded B. subtilis. The strep were Groups G(33%), C(28%), B(26%), A(20%) and F(3%). The disc "resistant" Group A strep made up only 0.11% of the total cultures and an estimated 0.3% of the hemol col. All 10 "resistant" Group A isolates were bacitracin sensitive on replate. We conclude that 44% of the patients are treated unnecessarily to include the 0.3% that may be missed by faulty disc identification

824 RAPID DIAGNOSIS OF H. INFLUENZAE TYPE B (HIB) INFECTIONS BY LATEX PARTICLE AGGLUTINATION (LPA) AND COUNTER IMMUNOELECTROPHORESIS (CIE), Joel I. Ward, George R. Siber, David W. Scheifele, David H. Smith, Children's Hospital Medical Center, Department of Medicine, Boston, Ma.

LPA, a simplified LPA (SLPA) performed at the bedside by house officers, and CIE assays which were developed in this laboratory are sensitive in vitro to 0.2 ng/ml, 0.5 ng/ml and 1 ng/ml of HIB capsular antigen respectively. The sensitivity and specificity of these assays were compared prospectively in 106 episodes of suspected HIB infections.

	LPA+	SLPA	CIE
HIB Meningitis (N=26)			
CSF	25/25	17/17	20/24
Serum	23/24	9/9	20/24
Other HIB infections (5 epiglottitis, 4 cellulitis, 4 pneumonia)			
Serum	13/13	9/9	6/12
Non-HIB infections			
CSF	0/34	0/13	0/33
Serum	0/60	0/11	7/58*

\+ No-positive/No-tested

* Weak positive reactions removable by phenol extraction.

LPA AND SLPA are more sensitive than CIE in the diagnosis of HIB infections, especially in non-meningitic disease ($p = 0.005$). The LPA assay is simple enough for bedside use, specific, and will detect almost all invasive HIB infections.

825 ADENINE ARABINOSIDE THERAPY OF HERPES SIMPLEX ENCEPHALITIS: NIAID COLLABORATIVE ANTIVIRAL STUDY GROUP. Richard J. Whitley, Raphael Dolin, George J. Galasso, Lawrence T. Ch'ien, Charles A. Alford and the Collaborative Study Group. Department of Pediatrics, University of Alabama in Birmingham, Birmingham, Alabama and the NIH, Bethesda, Maryland.

Adenine arabinoside was evaluated for the treatment of herpes simplex encephalitis in a randomized controlled study. Of 50 patients with a presumptive clinical diagnosis and undergoing brain biopsy, 25 cases were proved by isolation of virus from the brain specimen. Adenine arabinoside treatment reduced mortality from 75 percent, 6 of 8, in placebo recipients to 29 percent, 5 of 17, in drug recipients ($P = 0.027$). Both groups were comparable for age, sex, race and concomitant therapy as well as the presenting signs and symptoms of the disease. Fifty percent of treated survivors were left with minor to moderate sequelae, returning home and to gainful activity. The level of consciousness at the time of biopsy and institution of therapy was the major determinant of outcome. At biopsy, the mortality rate of comatose patients was 66 percent while those lethargic or semicomatose was 25 percent. Prognosis was not influenced by age or sex. These beneficial effects were achieved without evidence of acute drug toxicity. Thus, adenine arabinoside has a good therapeutic index (efficacy/toxicity) for the treatment of herpes simplex encephalitis. Drug must be given early in the course of infection before the advent of coma and seizures in order to attain a satisfactory outcome. Moreover, it should be coupled with brain biopsy for specific diagnosis to avoid unnecessary therapy of encephalitides which mimick herpes.

826 ADENINE ARABINOSIDE THERAPY OF VARICELLA ZOSTER IN IMMUNOCOMPROMISED PATIENTS. Richard J. Whitley, Raphael Dolin, George J. Galasso, Charles A. Alford and the Collaborative Study Group. Department of Pediatrics, University of Alabama in Birmingham, Birmingham, Alabama and the NIH, Bethesda, Maryland.

Nineteen immunosuppressed patients with varicella zoster were enrolled in a randomized crossover 10 day trial of adenine arabinoside. Of 14 with reticuloendothelial malignancy, 7 were receiving active chemotherapy. Age, sex, underlying disease, and preceding chemotherapy were comparable for both randomization groups. Eight patients received drug over the first 5 days and placebo the next 5; while 11 received the exact opposite regimen. In spite of natural healing in the placebo group, adenine arabinoside significantly accelerated cutaneous healing for the elimination of virus from vesicles ($P = 0.015$) and cessation of new vesicle formation ($P = 0.026$). Although pustulation was not statistically accelerated, all treated patients had completely pustulated by the time of crossover. Five patients, all with reticuloendothelial cancer, developed pneumonitis and, because of the crossover received adenine arabinoside during their disease course. The mean duration of pneumonitis was 5.6 days $\pm$ 2.1 (range: 3 to 8). Resolution was temporally associated with therapy. There were no study deaths. Clinical and laboratory evidence of toxicity was insignificant at the dose employed. These data for therapy of primary varicella zoster infection support the larger study demonstrating accelerated healing and satisfactory therapeutic index in herpes zoster with adenine arabinoside.

827 TEN YEAR EXPERIENCE WITH FURTHER ATTENUATED RUBEOLA VACCINES IN INFANTS AND CHILDREN. J. Wilkins, P.F. Wehrle. Los Angeles County/University of Southern California Medical Center, Department of Pediatrics, Los Angeles, California.

From July 27, 1965 to September 3, 1975, 1030 infants enrolled in the growth and development clinics at LAC/USC MC were inoculated with live further-attenuated rubeola vaccine singly, simultaneously or in combination with other live virus vaccines. Ages at the time of inoculation ranged from 167 days to 4½ years. The hemagglutination-inhibition (HAI) antibody responses of rubeola-susceptible (HAI $<1{:}8$) infants to rubeola vaccine was independent of other administered antigens. Seroconversion rates by age after 167 days at 23 day intervals (t½ human gamma globulin) were evaluated. After 335 days of age 90 to 96.6% of the inoculated infants developed HAI titers $\geq 1{:}8$. Seroconversion rate to rubeola antigen in infants inoculated at 335-358 days of age was 91.4%; at 359-382 days, 92.6%; at 383-406 days, 96.6%; at 407-430 days, 94.6%; at 431-454 days, 93.3%; at 455-478 days, 90.9%; at 479-502 days, 92.3%; at 503-525 days, 90.0%; and at $\geq$ 530 days, 90.9%. The seroconversion rate in children $\geq$ 15 months of age was similar to that seen in infants 12-14 months of age. Our findings also suggest factors other than age which may be important in the occurrence of rubeola vaccine failures.

828 LIVE PARAINFLUENZA TYPE 3 VACCINE IN CHILDREN Peter F. Wright, Hidenori Meguro, Juliette Thompson, Anne E. Torrence, David T. Karzon, Vanderbilt Univ., Sch. of Med., Dept. of Ped., Nashville, Tennessee

Clinical trials in young children of viral respiratory vaccines provide a model for determining viral and host factors in the control of primary infection. An attenuated parainfluenza 3 (para 3) vaccine was given intranasally (10^{4-5} $TCID_{50}$/dose) to 16 children ages 13 to 35 months-with 5 additional placebo inoculated controls studied for transmission and intercurrent illness. Prior natural para 3, as judged by prevaccination serum antibody, completely inhibited vaccine virus shedding and only 1/7 seropositive children had an antibody rise. In contrast, 7/9 seronegative children shed virus for up to 11 days after inoculation. All seronegatives had serum antibody responses by CF, HAI or plaque-neutralization tests. No nasal antibody responses were observed in 9 children (4 of whom had a serum antibody rise)tested. Mild afebrile respiratory illness was observed at the time of peak virus shedding,8-9 days post inoculation, suggesting illness was a function of virus replication. Thus, multiple laboratory passages (4 in monkey kidney, 75 in eggs) did not sufficiently attenuate para 3 for seronegative children. In spite of symptomatic shedding in vaccinees no transmission to controls was observed. The duration of para 3 vaccine shedding in seronegatives was uniform with a mean of 9.4 days. This is similiar to shedding by seronegatives of attenuated respiratory syncytial virus, 10 days, and influenza, 8.6 days; and suggests a common host mechanism for terminating primary respiratory viral infection.