PNEUMOCYSTIS CARINII PNEUMONITIS IN VIETNAMESE INFANT IN U.S. Arthur Eidelman, Andrew Nkongo and Rachel Morecki. (int. by Lawrence Gartner), Dept. of Ped., A. Einstein Coll. Med.,N.Y.

An infectious disease sequela of the Vietnamese war of epidemiological significance is reported.

A Vietnamese premature female infant, cared for in an orphanage in Saigon, was adopted by an American family at age 2 1/2 mos. Physical exam and chest x-ray on arrival in N.Y. were normal. At 3 1/2 mos. respiratory distress developed, treated for 3 wks. unsuccessfully with antibiotics. X-ray revealed interstitial pneumonitis. Pneumocystis carinii pneumonitis (PCP) was diagnosed by silver methionine stain of fluid obtained by bronchial lavage. Cytomegalic virus (CMV) was cultured from same fluid. Bacterial, mycobacterial, and fungal cultures were negative. Studies of immunological competence were normal. IgG to Pneumocytis carinii (PC) was positive. Family members remained asymptomatic but became serologically positive for PC. Infant died despite 11 days of pentamidine therapy. Autopsy revealed PCP with plasma cell infiltration. Thymus was normal. Brain and liver were free of CMV.

PCP in the U.S. has been diagnosed almost exclusively in children with immunological disorders or with lymphoreticular malignancies, whereas PCP is endemic in underdeveloped areas. With increasing adoptions of Vietnamese infants by Americans, epidemiological surveillance, including serological screening, is advised to detect PCP in these immunologically normal infants.

NEW INSIGHT ON *MYCOPLASMA PNEUMONIAE* INFECTIONS IN EARLY CHILDHOOD. G.W. Fernald, A.M. Collier and W.A. Clyde, Jr. Univ. of N.C., Sch. of Med., Dept. of Ped., Chapel Hill.

Respiratory illness due to *M. pneumoniae* is common in school age children (peak age 9 yrs.) but rare in infants, a consistent epidemiologic finding which never has been explained adequately. Monitoring of respiratory tract microflora in 50 infants and young children attending the Frank Porter Graham Day Care Center (DCC) yielded 15 isolations of *M. pneumoniae* from 1968-1973. These infections occurred in the fall and early winter of each year and coincided with identification of this agent in children with lower respiratory disease seen in a local pediatric practice. Frequent medical evaluation revealed only mild rhinitis and cough in half the DCC subjects. Infected children ranged in age from 2 months to 8 years. Diagnostic serologic rises (complement fixing and growth inhibiting antibody), occurred in all but one culture-positive child, as well as in several with negative cultures. Titers tended to wane rapidly and 3 children became reinfected after 3 yrs. In vitro stimulation of peripheral lymphocytes with *M. pneumoniae* was performed on all infected children during the last year of the study. Antigen reactive cells were detected frequently only in children over 6 yrs. of age. These findings suggest that recurrent unsuspected infection with *M. pneumoniae* occurs during infancy and early childhood and that pneumonic disease, common in school-aged children, is an expression of increasing host immune response to the organism.

NURSERY OUTBREAK OF SCALDED SKIN SYNDROME DUE TO GROUP I STAPHYLOCOCCUS AUREUS. Howard S. Faden, John P. Burke, James R. Everett, Lowell A. Glasgow, Univ. of Utah Med. Ctr., Dept. of Med., Salt Lake City.

Scalded skin syndrome (SSS) has been associated with group II staphylococci. We recently studied a nursery outbreak of this syndrome caused by group I S. aureus.

The epidemic occurred from 8/1-14/73 in the newborn nursery of a Salt Lake City hospital. Four female infants developed a scarlatinaform eruption within the first week of life. The eruption was characterized by a generalized, finely papular erythema; one infant experienced mild epidermal peeling. Two of the infants were febrile. Cultures of the umbilical cords and/or the anterior nares grew S. aureus, phage type 29/52/79/86/D11/81. The eruption rapidly cleared after treatment with Nafcillin.

Bacteriologic surveillance of the nursery revealed one admitting nurse as a nasal carrier of the epidemic phage type; no infants other than the original cases were colonized with the epidemic strain, and no additional cases were uncovered by a telephone survey.

Staphylococci which cause SSS are characterized by the production of a toxin which is capable of producing exfoliation in newborn mice. The epidemic strain, when tested in the suckling-mouse, produced epidermal necrolysis. The demonstration that a group I staphylococcus can produce an exfoliative toxin suggests that a common mechanism for toxin production may exist for groups I and II staphylococci.

ELIMINATION AND REAPPEARANCE OF STREPTOCOCCI IN SKIN LESIONS AND ON NORMAL SKIN FOLLOWING PENICILLIN TREATMENT. Patricia Ferrieri and Lewis W. Wannamaker. Univ. of Minn. Med. School, Dept. of Ped., Minneapolis, Minn.

The rapidity of disappearance of group A streptococci from impetiginous lesions and normal skin was examined in 49 children treated with 10 days of oral penicillin (34) or long-acting benzathine penicillin (15). Before treatment (Rx) 92% of children had streptococci recovered from normal skin. One week later 20 of 23 children (87%) on oral penicillin were free of streptococcal skin lesions; 6 (26%) had streptococci present on normal skin, 2 of whom had positive lesions. Following benzathine penicillin none of 12 children had lesions 1 week later; 2 had positive normal skin sites.

In further studies, 13 children were examined and cultured on presentation and 1,2,3,6 and 7 days after Rx was begun. In 7 children on oral penicillin the mean interval for streptococcal eradication from lesions was 3 days (range 2-4) and from normal skin 2 days; streptococci reappeared on normal skin in 5 children by 4 days. In 6 children, following benzathine penicillin, lesions and normal skin yielded no streptococci by a mean period of 2.5 days (range 2-4); in 1 child organisms reappeared on the normal skin.

Persistence of streptococci in skin lesions for a few days after either oral or parenteral Rx has practical and theoretical implications. Streptococcal recovery from normal skin in these circumstances may reflect survival or new acquisition, and further study is needed to clarify this point.

HOME CULTURE PROGRAM FOR CHILDREN WITH RECURRENT BACTERIURIA. Robert S. Fennell III, Eduardo H. Garin, Sandy R. Austin, R. Dixon Walker and George A. Richard. Univ. of Fla. Col. of Med., Dept. of Ped., Gainesville (Intr. by Martin L. Schulkind)

One hundred sixty-five children with recurrent bacteriuria were placed on a continuing home culture program with 87% compliance. Parents were taught to obtain clean-catch urines on the first urine of the morning using the urine culture tube technique (Bactercult). They were taught to read the cultures following incubation both at room temperature and in an incubator. Positive cultures (>10 cols./unit area) were repeated. If urines were negative, patients were seen on alternate months; if 2 positive urines were obtained, the patient was returned to clinic for confirmation. Colony counts between 10 and 50 were rarely positive in clinic. When 2 positive urines (>50 cols./unit area) were obtained, confirmation in clinic for significant bacteriuria was 65%. The false positive rate was, therefore, significant. The false negative rate was very low at <4%. There was good agreement between incubator and room temperature for colony growth.

A home culture program can significantly reduce office visits; more than 200 visits were avoided for negative cultures. Cultures can be obtained frequently, conveniently, on the first voided urine of the morning and at low cost to parents. Early diagnosis of an asymptomatic recurrence can be made. The epidemiology of bacteriuria can be studied in the family, and a significant advance in the extension of health care to the community can be made.

GENES FOR SUSCEPTIBILITY TO VIRUSES. Park S. Gerald, Virginia Monedjikova, and John F. Enders. Harvard Medical School, Children's Hospital Medical Center, Department of Medicine, Boston and Wrentham State School, Wrentham, Massachusetts.

Polio virus is capable of growing within human cells, and not mouse cells, because human cells possess certain genes required by the polio virus for multiplication. Miller et al. have demonstrated that these polio "susceptibility genes" are probably on human chromosome 19, since human-mouse hybrid cells are susceptible to polio only as long as they possess chromosome 19. We have hybridized cells from a patient carrying an X/19 translocation with mouse cells deficient in the X-linked enzyme HGPRT. These hybrids selectively retain that portion of chromosome 19 which is attached to the piece of the X with the HGPRT locus. These hybrids are susceptible to polio virus Types 1, 2 and 3, even though they lack a portion (the distal part of the long arm) of chromosome 19. This is consistent with assignment of the polio susceptibility genes to this chromosome and suggests they may be confined to a limited region of the chromosome. The hybrid cell approach is generally applicable to the analysis of susceptibility genes for viruses of limited host range, some of which are currently under study.