1044 DIARRHEA-ASSOCIATED ADENOVIRUSES: RAPID PRESUMPTIVE DIAGNOSIS BY ELECTRON MICROSCOPY. Carl D. Brandt, William J. Rodriguez, Hyun W. Kim, Julita O. Arrobio, Barbara C. Jeffries, Mary K. Gardner, Robert H. Parrott. Children's Hospital and the George Washington University School of Medicine and Health Sciences, Washington, D.C.

School of Medicine and Health Sciences, Washington, D.C.
There is a need for the rapid recognition of infection by
the newly-described enteral or diarrhea-associated adenoviruses (adenoviruses that are of proposed neutralizing serotypes 40 and 41, or that have restriction endonuclease electrophoretic profiles of Wadell groups F or G). These viruses
especially infect infants, and they tend to be fastidious in
their growth in conventional cell cultures.

During a 6 minute viewing per specimen, adenoviruses were
visualized by direct electron microscopy (EM) or immune EM in
the stools of 52 pediatric inpatients with acute gastroenteritis. Forty (77%) of these viruses proved to be enteral
adenoviruses on the basis of neutralization test in 293 cells
and/or electrophoretic profile. However, only 5 (36%) of 14

During a 6 minute viewing per specimen, adenoviruses were visualized by direct electron microscopy (EM) or immune EM in the stools of 52 pediatric inpatients with acute gastroenteritis. Forty (77%) of these viruses proved to be enteral adenoviruses on the basis of neutralization test in 293 cells and/or electrophoretic profile. However, only 5 (36%) of 14 patients with less than 1 adenovirus particle per minute of direct EM viewing of the original diarrhea stools were found to have an enteral adenovirus, as compared to 35 (92%) of 38 patients with 1 or more such particles per minute of viewing (X² = 18.34, P = <.01). Thus the quantity of adenoviruses found in a diarrhea stool would appear to provide a strong presumptive indication of the presence or absence of an enteral adenovirus. This determination can be made within 15 minutes of specimen collection.

TRANSPLACENTAL PASSAGE OF ANTI-CMV ANTIBODIES DURING IN UTERO INFECTION. W.J. Britt, R.F. Pass, S. Stagno and C.A. Alford, University of Alabama in Birmingham, Approximately 5-10% of infants infected in utero with cytomegalovirus (CMV) exhibit clinically apparent disease at birth with the remainder having subclinical infection.

Approximately 5-10% of infants infected in utero with cytomegalovirus (CMV) exhibit clinically apparent disease at birth with the remainder having subclinical infection. Although the maternal immune response has been implicated as a virulence factor in congenital infection, conventional serologic techniques have failed to clarify the role of transplacentally acquired maternal anti-CMV antibodies in the prevention of symptomatic congenital infection. To further elucidate the effect of maternal antibody on the development of symptomatic infection, we have utilized the Western immunoblot procedure to investigate both the quantity and the antigen-specificity of anti-CMV antibodies in the cord sera of 6 infants with symptomatic disease and 10 asymptomatic, congenitally infected infants. Both groups acquired CMV as a result of primary maternal infection. Antibodies directed against virion glycoproteins and capsid proteins were detected in both groups; however as a group, a stronger response was noted in sera from asymptomatic infants. Unexpectedly, maternal sera obtained at delivery from 12/16 of the infants contained a greater quantity of anti-CMV antibodies as well as reacting with additional virion proteins not detected by cord sera. These results indicated that transplacental passage of maternal anti-CMV antibodies was incomplete in the majority of infants studied. Furthermore, our findings suggested that maternal antibodies may play a role in the prevention of symptomatic congenital CMV infection which was not previously appreciated.

ACQUIRED IMMUNE DEFICIENCY SYNDROME IN FAMILIES. Kenneth Bromberg, Senih
Fikrig, Edward Kong, Hermann Mendez, Margaret Hammerschlag. SUNY
Downstate Medical Center/Kings County Hospital, Dept. of Peds., Eklyn.

The acquired immune deficiency syndrome (AIDS) is seen in children born to parents with AIDS. In 2 families, AIDS or presumed AIDS occurred in a child prior to the development of AIDS in a parent.

In the first family, a child developed weight loss and diarrhea shortly after birth. At 5 months of age the child was hospitalized because of fever. A lung biopsy done because of a persistent pulmonary infiltrate revealed P. carini; C. neoformans was isolated from the tissue. Immunoglobulins were elevated but mitogen stimulation studies and the numbers of B and T cells were normal. Disseminated CRV disease and T cell depletion were seen at autopsy. Thirty months later the patient's father, after 3 months of weight loss, developed C. neoformans meningitis. Immunologic studies were consistent with AIDS. The mother and a second child born one wear after the jirth are both normal.

wother and a second child born one year after the first are both normal.

In the second family, a child developed a fatal pneumonia over a 2 week period.

In the second family, a child developed a fatal pneumonia over a 2 week period.

Immunologic studies were not obtained and an autopsy was not done. A second child born 1 year later developed weight loss and oral candidiasis at 5 months of age. At 7 months of age this child was admitted because of persistent candidiasis and diarrhea. Immunologic studies revealed a reversed T4/TB ratio, increased immunoglobulins, decreased natural killer activity, and normal mitogen stimulation studies. N. avium was isolated from a lung biososy. Findings at autopsy were consistent with AIDS. One month after the child was hospitalized, his father was noted to be losma weight; immunologic studies were consistent with AIDS but no opportunistic infection has been documented.

In these 2 families, infants developed AIDS or presumed AIDS well in advance of their affected fathers. Just as AIDS in parents is a risk factor for the development of AIDS in their unborn children, AIDS in infants identifies parents at high risk as well. Both attuations should be used to identify individuals who may denefit from interventional strategies to prevent the progression of AIDS.

TREATMENT OF PATIENTS WITH RECURRENT TONSILLITIS

1047 DUE TO GROUP A BETA HEMOLYTIC STREPTOCOCCI: A PROSPECTIVE RANDOMIZED STUDY COMPARING PENICILLIN,
ERYTHROMYCIN AND CLINDAMYCIN. Itzhak Brook and Ronald Hierkawa.
Depts. Pediatrics and Otolaryngology Uniformed Services University of the Health Sciences and the Naval Medical Research Institute. Bethesda, Maryland 20814
Forty-five patients who suffered from recurrent tonsillitis

Forty-five patients who suffered from recurrent tonsillitis due to Group A beta haemolytic streptococci (GABHS) participated in a prospective randomized study comparing penicillin, erythromycin or clindamycin therapy. Surface tonsillar cultures were obtained prior to therapy, 10 days after termination of therapy and once a month for a period of 12-18 months. They were processed for aerobic and anaerobic microorganisms. Mixed aerobic and anaerobic flora were obtained from all cultures. Beta lactamase-producing aerobic and anaerobic bacteria were present in 43 of the 45 (93%) tonsillar cultures. Administration of penicillin eradicated GABHS in 2 of 15 patients, erythromycin in 6 of 15 and clindamycin in 14 of the 15. Of the patients that were followed up, 12 of 14 patients treated with penicillin and 6 of 14 patients treated with erythromycin, and 1 of 15 treated with clindamycin continued to suffer from recurrent tonsillitis. Four patients who were treated with penicillin and two treated with erythromycin had their tonsils removed. This study demonstrates the efficacy of clindamycin, an antimicrobial effective against GABHS, and both aerobic and anaerobic beta lactamase-producing organisms, in prevention of recurrent tonsillitis.

DIFFERENTIATION OF VARICELLA VACCINE FROM WILDSTRAINS. Philip A. Brunell, and Elaine K. Cobb. U.T. Health Science Center, Department of Pediatrics, San Antonio, Texas.

Varicella vaccine (VV) recipients have occasionally developed significant illness following immunization. It is necessary to account in whether these illnesses are produced by Wilden by Wilden by

Varicella vaccine (VV) recipients have occasionally developed significant illness following immunization. It is necessary to ascertain whether these illnesses are produced by VV or by natural infection. Virus was isolated from one vaccine recipient 26 and 28 days post immunization. These isolates, (Lo26 and Lo28) were tested together with early and late passage vaccine, (Oka) strain; 3 wild isolates; and a laboratory, (Ellen) strain. VZ infected cells were lysed and then treated to remove normal cell components. The virus was then treated with SDS to release viral DNA which was purified and digested with a variety of restriction enzymes (RE). The digests were electrophoresed in agarose gels containing ethidium bromide. Two types of patterns were observed. One, found with several enzymes, revealed minor changes reflecting previously described variable regions. The second, seen only with Pst 1, clearly differentiated vaccine and Lo26 and Lo28 from the others. Differences observed appear to result from the deletion of a Pst 1 RE site in the long segment of vaccine VZ DNA. This change was already present in early passage of the Oka strain while the Ellen strain retained this site despite over 150 tissue culture passages. Pst 1 characterization of VZ DNA obtained from isolates will be useful in determining whether rashes in vaccinees or their contacts are due to vaccine or result from unrecognized wild VZ infection. It will also help to characterize isolates from vaccinees who develop zoster.

ANTIGEN-SPECIFIC SECRETORY ANTIBODY (Ab) RESPONSE FOLLOWING RESPIRATORY SYNCYTIAL VIRUS (RSV) INFECTION IN INFANTS. J.C. Burns, R.M. Hendry, J.C. Ho, B. Fernie, and K. McIntosh. Children's Hospital, Div. Inf. Dis., Boston, MA., and Georgetown University, Washington, D.C. We examined nasal secretions (NS) of 13 patients (pts) age 1-8 months (mos) hospitalized with acute RSV infection for IgA Ab to the surface glycoproteins (66K and 84K) and nucleocapsid pro-

8 months (mos) hospitalized with acute RSV infection for IgA Ab to the surface glycoproteins (66K and 84K) and nucleocapsid protein (NCP) of RSV. Acute and convalescent (conv) NS (obtained 3-17 days (d) after initial NS) were examined by enzyme-linked immunosorbent assay (ELISA) using individual mouse monoclonal Abs (RSV Long strain) to 66K, 84K, and NCP and RSV hyperimmune horse serum (HARS) as capture Abs. Competition assays demonstrated that the antigens captured in this system were specific for each monoclonal Ab used. Total IgA Ab to RSV measured by HARS showed a  $\geq$  3-fold rise in Ab titer in 9/13 pts. Ab rise (2 3-fold) to 66K (fusion) glycoprotein occurred in 5/13 pts; 84K, 3/13 pts; and NCP, 6/13 pts. Ab rise was most striking in the 7 pts with later conv NS ( $\geq$  6 d after acute NS). All but the youngest (1 mo) of these 7 pts had a  $\geq$  3-fold rise in total RSV Ab measured by HARS capture. Three, 2, and 4 of these pts had Ab rises to the 66K, 84K, and NCP respectively. Only 2/13 pts, both aged 1 mo, had no detectable RSV-specific Ab in NS. Ab rise in the remaining pts could not be explained by differences in IgA content between acute and conv NS. We conclude that monoclonal Ab capture ELISA can be used to detect class-specific Ab in infant NS and that infants recovering from RSV make IgA Ab to NCP and both surface glycoproteins.