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ACCURATE DIAGNOSIS OF ACQUIRED OR CONGENITAL VIRAL INFECTIONS BY THE USE OF A SINGLE SERUM SAMPLE. Pauli O. Leinikki, Isabel C. Shekarchi, Preston H. Dorsett, John L. Sever, NIH, NINCDS, IDB, Bethesda, Md.

Assay of virus-specific IgM antibody levels is useful in the laboratory diagnosis of recent acquired or congenital infections from single acute or early convalescent serum specimens. Unfortunately, rheumatoid factor (RF) activity causes false positive results even when the levels of RF are below the detection of available assay methods and clinical features do not suggest the presence of RF. Elimination of this false reactivity by sucrose density gradient or chromatographic separation of IgM is laborious and lowers the sensitivity of the assay.

A new method employing adsorption of sera with immobilized protein-A followed by class-specific antibody determination using ELISA has solved this problem. Small aliquots of serum were centrifuged after mixing with protein A sepharose. Virus-specific IgM and IgG antibodies were then assayed from the supernatant by ELISA using microcuvettes sensitized with gradient purified rubella or measles virus antigens and heavy-chain specific conjugates. By removing IgG, the absorption eliminated false IgM positivity due to RF as shown by tests with both rubella and measles antigens. The adsorption did not significantly reduce the specific IgM antibody titers.

The assay of specific antiviral IgM antibodies after adsorption with protein A by ELISA provides a sensitive and specific method for the serodiagnosis of recent or congenital viral infections.

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FEBRILE SEIZURES: NOT A BENIGN EVENT. George A. Lewin, Gary D. Overturf, Department of Pediatrics, University of Southern California School of Medicine, Los Angeles, California.

While pediatricians have considered febrile seizures (FS) benign, recent literature shows a correlation between FS, outpatient bacteremia, and late meningitis. To study the overall morbidity of FS, 161 outpatient children from 2 months - 5 years were analyzed by complete blood count (CBC), blood culture (BC), and complete cerebrospinal fluid analysis (CSFA). Results: 9/161 positive BC (*S. pneumoniae*), 1 clinically unsuspected meningitis, 5 aseptic meningitides, and 1 late meningitis. 16/161 children febrile without source on initial exam had significant disease on re-evaluation done because of a positive CBC (> 20,000 or > 20% band forms): 2 urinary tract infections, 9 pneumonias, 3 positive BC, 1 otitis media, and 1 adenitis. Diarrhea in 5 children with increased band forms indicated the "toxic shigella syndrome." In 3 children with otitis media on initial exam, markedly abnormal CBC's correlated with a positive BC.

FS are not benign. 5.5% of children are bacteremic and 15-20% have clinically inapparent disease. A FS necessitates CSFA, CBC, and BC. Positive CBC's require re-evaluation, with possible chest X-ray and urinalysis. A minimum of 48 hours of intravenous therapy is required for children with a positive BC.

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PREVENTION OF GROUP B STREPTOCOCCAL (GBS) INFECTION IN LOW BIRTH WEIGHT (LBW) INFANTS BY PENICILLIN (P) ADMINISTRATION FROM BIRTH. David J. Lloyd, T.K.

Belgaumkar, Kenneth E. Scott, A. John Wort, Kurt Aterman and Vernon W. Krause. (Spon. by Alexander C. Allen). Depts. of Ped., Obs., Microbiol. & Path., Grace Maternity Hospital and Dalhousie University, Halifax, Nova Scotia, Canada.

From Jan '69 to May '74, 10 of 11 LBW infants < 35 wk gestation died from GBS septicemia. A study was designed to test the hypothesis that mortality from GBS in these infants could be prevented. From Jun '74 to Dec '76, systemic P was started within 2 hr of birth in all infants < 35 wk gestation (changed to < 2500 g from Jan '77) following throat, ear, rectal, umbilical and blood cultures. P was stopped within 48 hours if GBS was not isolated; otherwise P was continued for 10 days. From Jun '74 to Nov '77 when P was administered, no positive blood cultures and no deaths from GBS infection occurred in study infants. The fact that 7 fullterm infants developed GBS septicemia during this period suggests that the virulence of the organism had not decreased.

	Livebirth > 500g			Livebirth 500-2499g		
	# Live Born	# GBS Deaths	Mortality Rate/1000	# Live Born	# GBS Deaths	Mortality Rate/1000
Jan '69-May '74	17238	10	0.58	1208	10	8.28
Jun '74-Nov '77	15010	1	0.07	983	0	0

Assuming no change in GBS colonisation rate, these data support the hypothesis that systemic P from birth will prevent mortality from GBS in LBW infants ($\chi^2 = 6.45$).

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CEPHALEXIN IN CYSTIC FIBROSIS: A PLACEBO-CONTROLLED STUDY. Vera A. Loening-Baucke, Elaine H. Mischler, Martin G. Myers (Spon. by Kabir Younoszai), University of Iowa Hospitals, Department of Pediatrics, Iowa City.

The efficacy of cephalexin monohydrate 50 mg/kg/d was evaluated in a placebo-controlled, double-blind, crossover study of patients with cystic fibrosis. Over a 2 year period, 17 patients with mild-moderate disease were treated for 4 month intervals with either cephalexin or placebo. One patient developed drug-related vulvovaginitis and dropped-out of the study. Although colonization by cephalexin-resistant *S. aureus* was not observed, 2 patients had an increase in sputum concentration of *Ps. aeruginosa* during antibiotic therapy.

Patients previously colonized with $\geq 10^6$ CFU/ml *Ps. aeruginosa* of *H. influenzae* had no alteration in flora. Six of 8 patients colonized with $\geq 10^6$ CFU/ml *S. aureus* had a decrease in *S. aureus* concentration to $< 10^4$ CFU/ml during treatment with cephalexin. During periods of drug treatment, significantly reduced *S. aureus* colonization (7% versus 26% of cultures), increased weight gain (1.2 versus 0.2 kg/4 months) and less hospitalizations (2 versus 15) were observed ($P < 0.01$). There were no differences in pulmonary function tests, chest x-rays, or sputum production during periods of antibiotic or placebo treatment.

Cephalexin, when compared to placebo, altered the microbial flora and clinical course of some patients with cystic fibrosis.

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INGUINAL HERNIAS IN PATIENTS WITH CYTOMEGALOVIRUS INFECTION. V.P. McCarthy, J.A. Ware, D.R. Stewart, M.F. Lenahan & C.T. Cho. Depts. of Peds. & Surg.

University of Kansas Medical Center, Kansas City, Kansas. An increased incidence of inguinal hernias has been noted in infants with congenital cytomegalovirus (CMV) infections. However the significance of this association is still poorly defined.

The incidence of inguinal hernias was determined retrospectively in 67 infants and children with documented cytomegalovirus infections. A prospective study was also conducted to define the incidence of cytomegalovirus infection in 24 consecutive patients presenting for surgical repair of an inguinal hernia.

The occurrence of inguinal hernias in patients with congenital or acquired cytomegalovirus infection revealed an overall incidence of 9.6% (7/67) but an 18.4% (7/38) incidence was noted among congenitally infected infants.

No. of patients with CMV	No. of patients with hernia (%)
congenital 38	7 (18.4)
acquired 29	0 (0.0)

Prospectively, cytomegalovirus infection was demonstrated in 3 of 24 (12%) infants and children with inguinal hernias. Our data, though limited, further document the association of inguinal hernias in cytomegalovirus infections.

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SECRETORY IMMUNOLOGIC RESPONSE TO RESPIRATORY SYNCYTIAL VIRUS INFECTION IN INFANTS: ANTIBODY ON AND OFF EPITHELIAL CELLS. Kenneth McIntosh, Joyce McQuillin,

Phillip S. Gardner. University of Colorado Medical Center, Department of Pediatrics, Denver; University of Newcastle-upon-Tyne, Department of Virology, Newcastle-upon-Tyne, England.

We studied the secretory immunologic response to respiratory syncytial virus (RSV) infection in 22 infants hospitalized for bronchiolitis or pneumonia. Nasopharyngeal secretions were obtained by suction every other day during hospitalization and (in most) once in convalescence. Shed epithelial cells were spotted on slides and acetone-fixed. These were then examined by double-labelling immunofluorescence techniques for RSV and IgG, IgA or IgM antibody. Cell-free secretions were titrated by indirect fluorescence for anti-RSV IgG, IgA and IgM.

In 19 of 22 infants IgA was seen coating RSV-infected nasal epithelial cells in the first available secretion (day 1 to 3 of hospitalization), and in all 22 by the 4th day. Cell-free anti-RSV IgA was never evident in the first specimen and appeared, on the average, on the 6th hospital day. In contrast, cell-associated IgG was seen in only 12 infants, often as late as the 4th day. Cell-free anti-RSV IgG appeared in all infants, on the average by the 4th day. RSV antigen was universally present through the 5th hospital day, and in 1/2 of infants for 8 days.

Both IgA and IgG antibody appear in secretions of RSV infected infants much earlier than previously suspected, and their possible roles both in cure and in mucosal immune complex disease must be seriously considered.