997A A COMPROMISED INTERFERON RESPONSE: AN ETIOLOGIC FACTOR IN REYE'S SYNDROME. K.R. Rozee, S.H.S. Lee, E. Arcinue and J.F.S. Crocker. Dalhousie Univ. and Izaak Walton Killam Hospital for Children, Halifax, N.S. and the Detroit Children's Hospital, Detroit. Depts. of Pediatrics and Microbiology.

Young mice treated with Toximul MP8, a polyoxyethylene ether based emulsifier, show an increased mortality when infected with encephalomyocarditis virus (EMC) than do control mice. Lymphocytes taken from these emulsifier treated mice respond poorly to interferon induction, as compared to controls. Interferon also protects control mice against EMC infection but such protection is reduced in emulsifier-treated mice. This enhanced lethality to EMC in emulsifier-treated mice may be due to compromised interferon response in these animals.

Blood samples were obtained from a group of Reye's patients in the acute and convalescent phase of their disease. Lymphocytes were induced to synthesize interferon by Newcastle Disease Virus. Peripheral blood lymphocytes from convalescent patients and controls responded well to interferon induction. Lymphocytes taken in the acute phase of children with Reye's syndrome produced significantly less interferon than those from recovering patients or controls. Since Reye's syndrome is an unusual response on the part of the host following a virus infection our data would appear to implicate interferon as part of this altered response.

998 DEVELOPMENT OF MENINGITIS FOLLOWING LUMBAR PUNCTURE (LP) IN CHILDREN WITH BACTEREMIA. Barry Dashefsky, David W. Teele, Tamara Rakusan, and Jerome O. Klein. Boston Univ. School of Medicine, Dept. of Pediatrics, Boston City Hospital, Boston.

To determine the association of lumbar puncture performed during bacteremia with subsequent meningitis, we reviewed records of 253 episodes of bacteremia occurring from Sept. 1971 to Dec. 1980. Children had blood drawn for culture and were subsequently identified as having bacteremia: S. pneumoniae (201), H. influenzae (49), N. meningitidis (3). LP was performed at initial visit in 36; no child had meningitis at this time. After culture(s) 123 bacteremic children received no antibiotics initially (I), and 130 with an identified focus such as otitis or pneumonia received oral antibiotics initially (II). Meningitis later developed in 11:

	I LP	No LP	II LP	No LP
Meningitis	5	1	3	2
No meningitis	11	106	17	108
	P = .0001		P = .024	

There is a significant association with LP performed during bacteremia and later development of bacterial meningitis. The association is strongest for children whose bacteremia was due to H. influenzae. These data do not show that the association is causal. Children who later developed meningitis may have been more ill at first visit and thus were selected for LP.

999 SCREENING TESTS FOR ASYMPTOMATIC PYELONEPHRITIS IN CHILDREN WITH ILEAL CONDUIT URINARY DIVERSIONS. Charles A. Davis, Morris D. Dixon, Jr., and Sandra Petti (Spon. by Marshall Klaus) Case Western Reserve University, Rainbow Babies & Childrens Hospital, Dept. of Pediatrics, Cleveland, Ohio

Urines were obtained from myelomeningocele patients with intact urinary tracts and by double lumen catheterization from myelomeningocele patients with ileal conduit urinary tract diversions. Urines were subjected to quantitative culture, LDH isoenzyme analysis, lysozyme concentration measurement, and testing for antibody coated bacteria. Individuals with anatomically intact urinary tracts and $< 10^5$ organisms per ml of urine had virtually no positive results by the three ancillary assays. Individuals with ileal conduits and $< 10^5$ organisms per ml of urine had normal lysozyme levels and negative antibody coating tests, but the majority had positive LDH isoenzyme assays. When $> 10^5$ organisms were present, individuals with ileal conduits were more likely to have positive results by all three assays than were those with intact urinary anatomy. Among patients with ileal conduits, changes in LDH isoenzyme positivity did not correlate with bacteriuria symptoms or antibiotic therapy.

As judged by lysozymuria and testing for antibody coated bacteria, $> 10^5$ bacteria per ml appears to represent significant bacteriuria in patients with ileal conduit urinary diversion. Lysozymuria and antibody coating of urinary bacteria may be useful in identifying patients with ileal conduit diversions who are at risk for chronic pyelonephritis.

1000 ADVERSE EFFECTS OF METHICILLIN, NAFCILLIN, AND OXACILLIN IN PEDIATRIC PATIENTS: A PROSPECTIVE STUDY. Sherry M. DeBolt, Milap C. Nahata, Dwight A. Powell, and Milo D. Hilty, Ohio State University Colleges of Med and Pharm and Children's Hospital, Dept of Peds, Columbus, Ohio.

Methicillin (M), nafcillin (N) and oxacillin (Ox) are commonly used antimicrobial agents, but limited prospective data are available about the incidence of their side effects in pediatric patients. Sixty-nine patients (newborn-18 yr) were evaluated prospectively for adverse effects associated with M, N or Ox. Hematologic parameters (hemoglobin, hematocrit, WBC with diff, platelet ct, and reticulocyte ct), hepatic enzymes (SGOT, SGPT) and renal function tests (BUN, serum creatinine and urinalysis) were obtained before the institution of treatment and at frequent intervals (min every 5 d) during the course of therapy. Culture and sensitivity, daily body temp and concomitant drugs were recorded. Three of 28 patients receiving M developed eosinophilia (eos, 705-1547/cu mm) within 5-8 days of therapy. Of 32 patients on N, 2 developed neutropenia (neut, 642-752/cu mm) within 4-13 days of therapy; 3 developed eosinophilia (eos, 1030-2171/cu mm) within 1-4 days of therapy; and 1 developed elevation of liver enzymes (SGOT 179 RFU, SGPT 98 RFU) on day 3 of treatment. Of 9 patients receiving Ox, 1 developed elevation of liver enzymes (SGOT 158 RFU, SGPT 409 RFU) on day 15, and 1 developed neutropenia (neut 684/cu mm) on day 11 of therapy. All laboratory values returned to normal with the discontinuation of therapy. Our data suggest that appropriate laboratory tests should be monitored to detect occurrence of adverse effects to M, N and Ox.

1001 BREAST ABSCESSES IN ADOLESCENTS. Timothy F. Doran, Nathan Litman, (Spon. by Gerald Nathenson), Albert Einstein College of Medicine, Montefiore and North Central Bronx Hosps., Dept. of Pediatrics, Bronx, New York.

The occurrence of breast abscesses has not previously been studied in an adolescent female population. We identified 12 patients at our institution over the past 4 years with breast abscesses. The patients ranged in age from 12 to 19 years and were all post-pubertal. None of the patients were menstruating at the time of presentation, nor were any post-partum or breast feeding. Only 3 patients were febrile; all were treated with antibiotics and 11 required surgical drainage. The infections were caused by Staphylococcus aureus in 7 patients, Staphylococcus epidermidis in 1, mixed anaerobes in 2, and both Group B Streptococcus and Staph epidermidis in 1. Ten of the 12 abscesses were subareolar. Both patients with anaerobic infections had several recurrences and required surgical excision of breast tissue. The anaerobic organisms identified were typical of normal oral flora. Our data indicate that (1) breast abscesses are not rare in the non-puerperal adolescent female and (2) anaerobes are important pathogens in this disorder and result in greater morbidity than breast abscesses due to staphylococci. This study demonstrates the need for anaerobic as well as aerobic culturing of breast abscesses and antibiotic and surgical management based on etiologic agents.

• 1002 QUANTITATIVE PROTECTION STUDIES IN A SUCKLING RAT MODEL OF GROUP B STREPTOCOCCAL SEPSIS. Gerald W. Fischer, Samuel R. Wilson, Kenneth W. Hunter, and Val G. Hemming. Uniformed Services University of the Health Sciences, Department of Pediatrics, Bethesda, MD. 20014

Group B streptococci (GBS) are a major cause of sepsis and meningitis in the newborn. Neonatal susceptibility to GBS infection appears to be associated with a deficiency of opsonic antibody to GBS. The present studies were designed to determine if human antibodies protective for one type III GBS strain provide uniform protection for other type III strains. IgG was affinity purified from pooled human immunoglobulin using a staphylococcal protein A immunoabsorbant. The relative titers of IgG antibodies to group B and type III (native and core) antigens were determined. To evaluate protection, suckling rats were challenged S.Q. with each of 4 different clinical isolates of GBS type III followed immediately by various concentrations of purified IgG given IP. With 5 or 10 µg of IgG significant survival occurred in animals challenged with one strain (17/20, 85%), whereas this amount of antibody afforded no protection against the other 3 strains (0/27). Administration of 20 µg of IgG provided some protection for 2 of these strains (7/23, 3/9), but uniform lethality occurred with the fourth strain even when more than 40 µg IgG was given. These studies demonstrate that four type III GBS strains require different amounts of human IgG for protection. Strain variability must be examined in future investigations of vaccine induced or passively acquired IgG for protection against GBS disease.