1062 INITIAL THERAPY FOR OSTEOMYELITIS AND SEPTIC ARTHRITIS WITH CEFOXITIN. Lisa M. Dunkle, Shehla H. Naqvi, John S. Venglarcik and Terry L. Dwelle (Spon. by William J. Keenan), St. Louis University, St. Louis University School of Medicine, Cardinal Glennon Memorial Hospital for Children, Department of Pediatrics/Adolescent Medicine, St. Louis, MO.

Single drug therapy with a broad spectrum antibiotic is a desirable option for patients with bone and joint infections of unknown etiology. We initiated treatment for 23 children, ages 6 months-15 years (mean 6.5 years) with cefoxitin. The infecting organisms proved to be S. aureus-10, Strep. pneumoniae-2, H. influenzae b-2, and S. epidermidis, B. melaninogenicus, Moraxella sp. and Ps. fluorescens-1 each. No agent was identified in 6 cases. Initial clinical response was good in 21 cases (91%). One primary resistant organism (Ps. fluorescens) and one cephalosporin-tolerant S. aureus failed to respond. A third child with infection due to H. influenzae b developed meningitis after 3 days. These 3, and 6 patients with negative cultures, were dropped from the study. All other patients did well and received $\bar{x}$ 15.7 days IV cefoxitin before switching to oral antimicrobials. Adverse reactions included allergic rash (1), mild eosinophilia (2) and mild elevation of SGOT (1). Cefoxitin was discontinued for reason of the rash; all other reactions resolved when the drug was discontinued at the completion of therapy. Neutropenia (PMN <1000/mm^3) was seen in 2 patients but resolved spontaneously. Urinary reducing substances were detected in 5 cases (22%). Cefoxitin appears to be satisfactory initial therapy for osteomyelitis and septic arthritis in pediatric patients; it should not be used for infants <2 years of age where H. influenzae b is suspected, due to the possible development of meningitis.

1063 ORAL RIFAMPIN/TOPICAL BACITRACIN THERAPY AS AN ADJUNCTIVE MEASURE TO CONTROL EPIDEMIC METHICILLIN-RESISTANT S. AUREUS IN A NEONATAL UNIT. Lisa M. Dunkle, William J. Keenan, G. Randall Andrews, Dagmar A. Schnader, Robert E. Fleming and Cindy A. Slaten, St. Louis University, St. Louis University School of Medicine, Cardinal Glennon Memorial Hospital for Children, Department of Pediatrics/Adolescent Medicine, St. Louis, MO.

Several months after institution of intensive infection control measures in a neonatal intensive care unit with a high infection rate due to methicillin-resistant S. aureus (MRS), patients and personnel colonized with the organism were treated with rifampin (R), bacitracin (B) and hexachloraphene (H) in an attempt to reduce the reservoir of MRS. Infection rates in the 6 months prior to the treatment (Rx) period were 11-22% and colonization rates ranged from 26-49%. 16 patients colonized with MRS in nose, cord or trachea and 5 personnel colonized intranasally were included in the study. Patient sites of MRS colonizations included nose-4, cord-2, nose and cord-6, nose and trachea-4. Rx regimen included R 20 mg/kg/day x5 days, B ointment to nose, cord, tracheostomy site x6 daily and H bath with rinse daily x2 days. Personnel received R 600 mg/day x5 days and B ointment intranasally 6x daily. Repeat cultures showed no MRS colonization in 9/16 (56%) patients and 5/5 personnel 2-8 weeks after Rx. Failure of Rx was not statistically associated with any particular site(s) of colonization. Adverse reactions to Rx included vomiting (3/16) and transient hypertransaminasemia (2/16). In the 4 months following the study period, the MRS monthly infection rate was 2-4% and the colonization rate was 6-20%. RBH Rx may offer an additional method of controlling epidemic MRS colonization when standard measures prove inadequate.

●1064 INTERACTION OF INFLUENZA WITH HUMAN NASOPHARYNGEAL MUCOSA. K.M. Edwards, D.S. Stephens, P.Snyder, P.F. Wright. Vanderbilt Univ., Nashville,Tn. and Emory Univ. Atlanta, Ga.

Previous studies of influenza A infections in animal models have stressed the tropism of this virus for the upper respiratory tract. To assess the interaction of influenza A virus with human respiratory tissue, adenoids, consisting of ciliated epithelium with underlying lymphoid follicles, were maintained in organ culture. Influenza growth was demonstrated when the organ culture was inoculated with wild type influenza A/Alaska (H3N2). The serologic status of the donor influenced viral replication, with a mean titer of 5.2×10^3 pfu/ml of virus released into the supernatant of organ cultures from 9 seronegative children at 48 hours and only 4.5×10^2 pfu/ml of virus released from 8 seropositive children. Vigorous ciliary activity of the nasopharyngeal tissue could be seen for up to 96 hours in organ cultures of uninfected tissue, while ciliary activity ceased after 24 hours in influenza infected tissue. By 24 hours after influenza infection, light microscopy of the organ culture mucosa revealed sloughing of the epithelial cells and infiltration of the submucosa with inflammatory cells, unlike control tissue. Fluorescent microscopy of infected organ cultures localized influenza antigen to the epithelial surface 4 hours after infection and by 24 hours, fluorescence was noted in submucosal locations. Lymphocyte populations from 12 adenoids have also been characterized. Mean values were 43% B cells, 29% T cells, 19% OKT 3^+ cells, 12% OKT 4^+ cells and 5% OKT 8^+ cells. Studies measuring specific in vitro anti-influenza antibody production by these lymphocytes are now in progress. The adenoids provide an intact organ culture system to study the pathogenesis of influenza A infections and the resultant local immune response.

†1065 POLYACRYLAMIDE GEL ELECTROPHORESIS (PAGE) OF ROTAVIRUS RNA AFTER SERIAL PASSAGE AND IN CHRONIC INFECTION. Joseph Eiden, Genevieve Losonsky, Steve Vonderfecht, John Johnson, Robert Yolken. Johns Hopkins Medical School, Baltimore, Maryland.

Rotaviruses are important causes of serious gastroenteritis in infants and children. New strains of rotavirus emerge during the course of an epidemic, but little is known about the mechanisms by which this occurrs. It is not known, for example, if significant alterations occur in the rotavirus genome during direct transmission from one individual to another. We investigated this question through the analysis of the distinctive electropherotypes produced by high resolution PAGE of rotavirus dsRNA from an animal model and from children with severe combined immunodeficiency disease (SCID) who had prolonged excretion of rotavirus.

To test the stability of the rotavirus genome during multiple infections, a strain of murine rotavirus was serially transmitted for more than 10 passages over a period of several months. No changes in the rotavirus electropherotype occurred during this period of infection. In the case of human infection, rotavirus was isolated from multiple stool specimens collected from two SCID patients over the course of several weeks and analyzed for RNA patterns. Each specimen contained more than one strain of rotavirus as determined by examination of the PAGE electropherotype. Marked changes were noted in rotavirus electropherotypes from different specimens from each patient. Recombinant events or recurrent infection with multiple rotavirus strains might account for the appearance of new strains of human rotavirus and might contribute to the chronic diarrhea in such patients.

1066 PSEUDOMONAS AERUGINOSA (PA) OSTEOMYELITIS IN CHILDREN: SIGNIFICANCE AND CLINICAL CHARACTERISTICS. Stephen J. Elliott and Stephen C. Aronoff (Spon. by William T. Speck) Case Western Reserve University School of Medicine, University Hospitals of Cleveland, Department of Pediatrics, Cleveland, Ohio.

PA is usually regarded as an infrequent cause of childhood osteomyelitis. The records of 144 children below 19 years of age and hospitalized between 1972 and 1981 were retrospectively reviewed. Osteomyelitis was defined as signs of localized infection and radiographic or radioisotopic demonstration of an osseous lesion. 104 patients fulfilled these criteria. S. aureus, no etiologic agent, and PA occurred in 60.5%, 15.4%, and 10.6% of the cases respectively. Compared to all other cases, children with PA osteomyelitis were significantly older (mean 8.0 yrs. vs 13.4 yrs. $p < .001$), lacked fever at admission (60.9% vs 18%, $p < .02$), and lacked bacteremia (60% vs 0%, $p < .03$). The relationship between penetrating trauma or cutaneous drainage and PA infection was highly significant ($p < .005$). Although statistical significance was not achieved, at the time of presentation children with PA osteomyelitis had lower erythrocyte sedimentation rates, lower peripheral white blood cell counts, and longer duration of symptoms when compared with other causes of osseous infection. PA is a significant cause of adolescent osteomyelitis and is characterized by signs of local, rather than systemic infection.

1067 ACIDOSIS IS A PRESENTING FEATURE OF CHLORAMPHENICOL TOXICITY. Linda S. Evans and Martin B. Kleiman. Dept. Peds., Indiana Univ. Sch. Med., Indpls, IN. (spon. by R. Schreiner)

Chloramphenicol (CAP) is an inhibitor of mitochondrial protein synthesis and impairs energy production through interference with oxidative phosphorylation. Metabolic acidosis associated with lactic acidemia has not been described as a presenting feature of CAP toxicity. Three critically ill children 6 mos to 11 y/o (congenital hypoaldosteronism, brain stem dysfunction, Reye's syndrome) received CAP (75-100 mg/kg/d) and had normal pH and stable cardiovascular, renal and pulmonary function for 48 hrs. Each developed profound acidosis (arterial pH 7.13-7.22) 60-108 hrs after beginning CAP. Initial CAP conc. were 61, 80, and 30 mcg/ml and subsequent levels decreased; CAP was 30 mcg/ml in the child with Reye's syndrome, a condition with recognized mitochondrial dysfunction. SGPT conc. were 386, 2,620, and 145 IU/l when acidosis was recognized. Serum lactate was 6.6 and 8.8 mM in 2 patients prior to CAP and increased to 11.1 and 17.1 mM. Mean anion gap increased from 16.8 to 32.3 mM in the 3 patients. Hypotension followed acidosis by 6-19 hrs and was severe in one and mild in 2. Other signs of gray baby syndrome were absent. CAP was stopped in each patient and resolution of acidosis and hypotension paralleled the fall of serum CAP. 1) Unexplained metabolic acidosis is an early sign of CAP toxicity; 2) pre-existing lactic acidemia, hepatic dysfunction, and/or mitochondrial dysfunction may predispose to CAP toxicity; 3) toxicity may occur with CAP levels in a therapeutic range in Reye's syndrome.