769 ORBITAL CELLULITIS IN CHILDREN. Andrew M. Gellady, Stanford T. Shulman, and Elia M. Ayoub. University of Florida College of Medicine, Dept. of Pediatrics, Gainesville.

Orbital cellulitis is an infection involving the orbit and adjacent periorbital tissue without involvement of other facial tissues. The bacterial etiology, in contrast to that of facial cellulitis, has not been clearly defined. To gain insight into this problem, bacteriological and clinical data on 86 children with orbital cellulitis were reviewed. Peak age incidence was 2-4 yrs. and in 84/86 cases the involvement was unilateral. Differences in clinical presentatfon enabled identification of two distinct groups. Group I consisted of 42 children with an adjacent soft tissue focus of infection and included 30 children with recent trauma to the area. Group II included 44 patients with no apparent focus of infection. Blood cultures obtained on 21 patients in Group I were positive in only one; the isolate was S. aureus. In Group II, 10/31 blood cultures were positive; 8 isolates were H. influenzae and 2 were S. aureus. The difference in incidence of bacteremia was significant ($p < 0.02$). S. aureus and/or Group A streptococcus were recovered from wound or conjunctival cultures in 74% of 39 cases in Group I and 24% of cases in Group II ($p<0.001$). Thus, the bacterial etiology of orbital cellulitis varies with clinical presentation. Patients with cellulitis and an adjacent soft tissue focus of infection or trauma are likely to have gram-positive pathogens. Patients lacking such a focus are similar to children with facial cellulitis in that bacteremia is encountered frequently and H. influenzae is the common etiological agent.

770 THE SYNERGISTIC AND ANTAGONISTIC RELATIONSHIP OF E. COLI AND P. AERUGINOSA IN NECROTIZING FASCIITIS AND MYOSITIS. Lewis F. Gold, Russell W. Steele and Melvin Baden, Dept. of Ped., Brooke Army Med. Ctr., San Antonio, TX.

The third and youngest neonate with necrotizing fasciitis and myositis resulting from E. coli is reported. Within 24 hours of diagnosis, the patient underwent radical surgery with identification and sensitivities of the etiologic agent. Mitogen stimulation of lymphocytes and immunoglobulins were normal. In an assay using acridine orange as a vital stain for bacteria, the phagocytic and bactericidal capacity utilizing the recovered K-1 antigen E. coli were felt to be normal. After treatment of the E. coli, the patient expired from an overwhelming septicemia resulting from P. aeruginosa. Although this might be interpreted as a secondary infectious process, we postulate that the presenting infectious process of necrotizing fasciitis and myositis was the result of a synergistic effect of two organisms rather than that of a single infectious agent with subsequent secondary infection.

A synergistic relationship between E. coli and P. aeruginosa was demonstrated in a rat model thus revealing a pathophysiologic process for necrotizing fasciitis and myositis. In-vitro studies indicated that recovery of multiple synergistic organisms is impeded by an antagonistic relationship based on the variable concentrations of organisms resulting from differential growth rates. This suggests that broad spectrum antibacterial coverage is indicated from the inception even though additional organisms are not identified.

771 IMMUNOGENICITY OF GROUPS A AND C MENINGOCOCCAL POLYSACCHARIDES OF DIFFERING MOLECULAR WEIGHTS.

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Two lots of bivalent groups A and C meningococcal polysaccharides were evaluated in children 2-10 years old. Molecular weights of both the A and C polysaccharides were 100,000-300,000 in Lot S and >2,000,000 in Lot X. 68 children received a single dose of vaccine (34 Lot S, 34 Lot X) and 45 received 2 doses of vaccine 2 months apart (27 Lot S and 18 Lot X). Antibody responses were assessed by indirect immunofluorescence (FA) and by bactericidal activity (BA). Pre-immunization FA titers of anti-A and anti-C were 5.9 and 6.7. One month post-primary, anti-A FA titers were 33.5 with Lot S and 134.1 with Lot X ($p < .05$); BA titers were 15 and 388 ($p < .01$). Significant anamnestic responses to A polysaccharide were not observed after either Lot S or Lot X. Anti-C FA titers one month post-primary were 32.0 after Lot S and 121.4 after Lot X ($p < .05$); BA titers were 45 and 697. Neither enhancement nor suppression of anti-C responses were seen after the second dose of vaccine with either lot. Protection of infants with vaccines similar in size to Lot S has been achieved against group A, but not group C meningococcal disease. The immunogenicity of large molecular weight vaccines similar to Lot X in infants is currently under investigation.

772 INFLUENCE OF MATERNAL CORTICOSTEROID (CS) ADMINISTRATION ON NEONATAL NEUTROPHIL COUNT. Steven J. Gross and Phillip I. Mieburg (Intr. by Frank A. Oski). SUNY, Upstate Med. Ctr., Dept. of Peds., Syracuse, N.Y.

Measurements of absolute neutrophil and band counts are now utilized with increasing frequency for the early diagnosis of infection in neonates. Unfortunately all the factors that may influence the absolute neutrophil count (ANC) during this period of life have not been elucidated. For this purpose ANC were determined at 3, 12-15, 48 and 72 hrs. of life in 4 groups of Caucasian premature infants who were appropriate weight for gestational age. One group did not develop respiratory distress syndrome (RDS), one group did, while a third group were born after their mothers received corticosteroids (CS) for 3 days prior to delivery - some of these infants developed RDS and others did not. The ANC ($x10^3/mm^3$ ± 1SD) in these infants are depicted below:

		HOURS			
RDS	CS	3	12-15	48	72
0	0	3.4 ± 2.2	6.7 ± 4.1	3.7 ± 2.1	3.8 ± 2.3
+	0	4.1 ± 2.9	8.2 ± 4.7	7.0 ± 7.6	5.0 ± 4.8
0	+	17.5 ± 5.6	15.7 ± 5.3	8.6 ± 4.7	8.0 ± 2.3
+	+	2.1 ± 1.0	7.7 ± 7.3	5.4 ± 5.7	1.6 ± 0.1

These studies illustrate that the administration of CS to mothers significantly increases the ANC of their offspring if they do not develop RDS ($p < .005$). The CS had no effect in infants who developed RDS. The ANC in neonates is influenced by maternal CS therapy and may be of predictive value in identifying those CS treated patients who will develop RDS.

773 SERRATIA MARCESCENS IN THE INTENSIVE CARE NURSERY (ICN) AND ITS CONTROL. Richard W. Hall, Mark E. White, William R. Collie, Alice G. Beard, Robert W. Arrington, Terry Yamauchi. Ark. Child. Hosp., Ark. Dept. Health, Univ. of Ark. Med. Sci., Little Rock, AR.

During March through July, 1976, an outbreak of Serratia Marcescens was studied in our ICN to determine the clinical course and epidemiology of 23 culture positive infants. Cultures were obtained from the environment, cord, stool, and pharynx of all infants born in the nursery during the outbreak, and handwashings, nasopharynx and vagina and/or rectum of all nursery personnel. Environmental cultures were negative, but 4 of 76 handwashings were positive after provodine-iodine scrubbing. Eleven symptomatic infections were recognized. Six positive cultures were obtained from blood, 2 from cerebrospinal fluid, 1 from peritoneal fluid, 3 from urine, and 1 from eye drainage. There were 5 probable cases of pneumonia. Serratia contributed to 2 deaths. Twelve infants were colonized but asymptomatic. Septic infants presented with a typical clinical picture of food intolerance, abdominal distention, tremors of the upper extremities and vasoconstriction. A study was performed using as controls infants brought into the ICN immediately before and after the culture positive infants. Significant factors were respirator care ($p < .001$), hyperalimentation ($p < .001$), multiple antibiotics ($p < .001$), and length of hospital stay ($p < .001$). The bacteria had the same serotype and characteristic antiobiogram (resistant to all antibiotics except chloramphenicol and amikacin). Spread was controlled by strict cohorting.

774 MECHANISMS OF ATTACHMENT BY A PATHOGENIC STRAIN OF E. COLI (0111/B4) TO INTESTINAL MUCOSA IN PRE- AND POST WEANLING RATS. Moshe Hirschberger, David Mirelman, and M. Michael Thaler. University of California, Department of Pediatrics, San Francisco.

Recent findings suggest that adhesiveness of certain strains of E. Coli to mammalian cells may be mediated by bacterial antigens with affinity for mannose residues of surface glycoproteins. We investigated the possibility that attachment of E. Coli to intestinal mucosal cells may be inhibited or reversed by mannose polymer. Mucosal scrapings from duodenum (D), jejunum (J), ileum (IL), and colon (C) of 7 day-old and 3 week-old rats were preincubated with a ^{3}H-thymidine-labeled strain of E. Coli associated with severe infantile diarrhea (0111/B4). Mannose polymer (mannan) or sucrose were added, incubation terminated after 30 min at 37°C, tissues washed free of unattached bacteria, and bound radioactivity counted. Mucosa from 7 day-old rats bound 8% of added bacteria in D, 30% in J, 14% in IL and 25% in C. Binding to mucosa from 3 week-old rats was comparatively minor (0.6% in D, 0.5% in J, 1.2% in IL, 2.6% in C). Incubation with mannan reduced binding in all segments of 7 day-old intestine by 93 to 98%, whereas inhibition in 3 week-old specimens averaged 20%. These results indicate that E. Coli 0111/B4 attach to pre-weanling intestine by a specific mechanism which may be reversed by mannose derivatives. Binding to post-weanling mucosa is much less effective, and is either partly mediated by a different mechanism, or is relatively non-specific.