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INDUCTION OF GENTAMYCIN RESISTANCE BY VISIBLE LIGHT. Richard A. Polin, William T. Speck. The Children's Hospital of Philadelphia, University of Pennsylvania and Rainbow Babies & Children's Hospital, Case Western Reserve University.

Recent studies have demonstrated the ability of visible light or phototherapy to modify the intracellular DNA of prokaryotic and eukaryotic cells. The present study was undertaken to determine the effect of phototherapy on drug resistance in prokaryotic cells using a tester strain of gentamycin sensitive E. coli. A growing population of the tester strain was inoculated onto agar plates containing minimal medium and gentamycin. The plates were divided into two populations, one which was illuminated while the other was kept in the dark to serve as a control. The cells were illuminated under a standard phototherapy unit (Dura Test Vita Lite). The unit was protected from direct sunlight and air cooled to maintain the cultures at 23°. The sample distance from the light source was adjusted to maintain a fluence rate (450 nm) of 141 μWcm^2. An increased mutation frequency to gentamycin resistance was seen in the irradiated population. The relationship between light induced antimicrobial resistance and the recent emergence of drug resistant bacteria in neonatal nurseries needs to be determined.

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EARLY ANTIBIOTICS IN GROUP B STREPTOCOCCUS(GBS) INFECTION. S.P. Pyati, P. Amma, R.S. Ramamurthy and R. S. Pildes. Cook County Children's Hosp., Chgo. Med. Sch. and Univ. of Ill., Depts. of Ped., Chgo., Ill.

The possible role for early antibiotics in improving survival of GBS (<96 hr) infection was studied in 29 neonates born between Jan. '76 to Oct. '77. All had positive blood and/or CSF cultures. Mean ± (S.E.) birth wt. was 1790 ± 163 gm (r.680-3544) and gestation, 33 ± 0.9 wk (r.25-42). Clinical diagnosis was respiratory distress 22, meningitis 3, sepsis 3, apneic spells 1. Ampicillin or penicillin plus gentamicin were started before 4 hr in 12, 4-8 hr in 4, >8 hr in 11. Two were not treated: one 680 gm died and one 2495 gm lived. Overall mortality was 58.6%; 92% in ≤1500 gm, 50% in 1501-2000 gm and 25% in >2000 gm. There was no sig. difference in mortality between those treated ≤4 hr (8/12) and those after 4 hr (8/15). Age at onset of therapy was not sig. different in those who died (7.8 ± 2.2 hr) from those who lived, 29 ± 10 hr. Immediate post mortem cultures were still positive in 11 who had been treated for 19 ± 9 hr and negative in 3 (therapy, 44 ± 12 hr). All 15 infants with radiographic HMD died despite antibiotics started at 5.2 ± 1 hr. Birth wt., Apgar or survival time were similar in HMD infants treated ≤4 hr or those >4 hr. All 7 infants with mild respiratory distress and radiographic Type II RDS lived:3 treated ≤4 hr, 3 > 4 hr and 1 not treated. These data suggest that early antibiotics did not improve survival of infants with GBS who presented with HMD. Whether early therapy may be beneficial in neonates who present with mild respiratory distress could not be answered.

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ASSOCIATION BETWEEN MATERNAL COLONIZATION WITH GROUP STREPTOCOCCI (GBS) AND PREMATURE RUPTURE OF MEMBRANES (PROM). J.A.Regan, S.Chao & L. S. James, Coll. P & S, Columbia Univ.,Depts. Ped. & Obstet, N.Y.

In a prospective study of 1684 women admitted with ruptured membranes, in labor or for induction, 231 were found to have cervical cultures positive for GBS. In 16% of the colonized population membrane rupture occurred a minimum of one hour prior to onset of regular contractions (PROM) vs. 8% of the total population studied ($p<.005$ $X^2 = 15.1$, 1 df). Conversely, of the 136 women with PROM, 37 (26%) were colonized with GBS compared to the overall colonization rate of 14% in the total population studied ($p<<.005$, $X^2 = 18.6$, 1 df). Thus a significant association was found between maternal GBS colonization and PROM . Furthermore, there were 16 (7%) pre-term deliveries at or prior to 32 weeks among 231 colonized women vs. 30 (1.8%) among the 1684 women studied. ($p<<.005$, $X^2=29.2$, 1 df) 8 of the 16 pre-term births in the colonized population followed PROM.

Antenatal screening at 32 weeks gestation has become an established approach to the detection of maternal GBS colonization. The rationale for this schedule is that screening at 32 weeks allows time for therapeutic attempts to eradicate colonization prior to delivery at or near term. Our findings suggest that screening and treatment prior to 28 weeks might reduce the risk of preterm delivery since it appears that the presence of the pathogen in the mother may predispose to PROM and premature delivery prior to the 32nd week of gestation.

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CELL MEDIATED IMMUNITY IN MOTHERS AND THEIR OFFSPRING WITH CYTOMEGALOVIRUS (CMV) INFECTION. David W. Reynolds and Paula H. Dean, University of Alabama in Birmingham, Department of Pediatrics, Birmingham, Alabama.

Lymphocyte transformation to CMV was determined for 7 patients with natal (N-CMV) and 17 with congenital (C-CMV) infection as well as their mothers. Antigens consisted of heat inactivated intracellular virus and a control. All subjects were tested against strain AD169 and ½ of them against a clinical isolate. The mean stimulation index (S.I.) of 11 seronegative adults was 0.7 (± 0.3) irrespective of the sera used. Mean indices for 10 seropositive normal adults were 21.7 in autologous sera (A.S.) and 34.8 in homologous seropositive sera (H.S.). Of the 7 children with N-CMV, 1/7 responded in A.S. and 2/2 in H.S. (mean maximal S.I. {m.m.S.I.}, 3.3). Among the 15 with inapparent C-CMV, 4/14 were positive in A.S. and 7/9 in H.S. for a total of 9 responders (m.m.S.I., 7.5). Neither of 2 with disease were positive. For the total group, 3/13 at ≤12 months of age reacted whereas 7/11 over 12 months did so. Seven of 9 mothers of children with N-CMV were positive as follows: 5/7 in A.S. and 3/3 in H.S. (m.m.S.I., 11.4). Among women with congenitally infected offspring, 13 of 15 responded, i.e., 7/14 in A.S. and 6/9 in H.S. (m.m.S.I., 8.4). Both mothers of affected infants reacted (m.m.S.I., 3.6). Among the 23 responding patients assayed in both sera, 9 stimulated in H.S. only whereas the converse was observed only once in this group and not at all in normal adult controls. In general, both antigens stimulated equally well; however, 3 children and 1 mother failed to respond to the wild strain.

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FAVORABLE RESPONSE TO TRANSFER FACTOR IN GENERALIZED BATTEY BACILLUS INFECTION. Gilberto E. Rodriguez, Richard T. Meyer, Nancy B. McWilliams, Edwin L. Kendig and Harold M. Maurer. Medical College of Virginia, Dept. of Pediatrics, Richmond, Virginia.

An 11 year old white girl presented with an 8-month history of thigh pain and weight loss. Skeletal survey showed multiple lytic lesions with radiographic appearance of "eosinophilic granuloma". Tibial biopsy showed chronic osteomyelitis. Bone marrow culture grew Mycobacterium avium-intracelluare (Battey bacillus). A PPD-B skin test was positive with 27 mm. induration.

A 6-drug anti-mycobacterial regimen was instituted but the disease progressed over the next 9 months with development of new bony lesions and subcutaneous abscesses with sinus tract formation at aspiration sites. Cultures of aspirated material again grew Battey bacillus and PPD-B skin test became negative.

Immunologic evaluation revealed leukocytosis, an elevation of gammaglobulins, sed. rate, B-cells and serum lyzozymes. Phagocytosis and monocyte chemotaxis were normal.

Nine months after diagnosis she received 2 units of Transfer Factor prepared from a highly reactive PPD-B donor. Four and 6 weeks later one additional unit was given. Over the next few months her clinical status improved dramatically with healing of sinus tracts, weight gain and gradual resolution of bony lesions. Although the sed. rate remains elevated, the leukocyte count and immunoglobulins are approaching normal values.

This experience suggests that further clinical trials with Transfer Factor are warranted in such Battey bacillus infection.

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HUMAN ROTAVIRUS (HRV) AND HEAT LABILE ENTEROTOXIN (LT) DETECTION IN A PEDIATRIC POPULATION. William J. Rodriguez, H.W. Kim, Harry Greenberg, Susan Seigel, Waheed N. Khan, Carl D. Brandt, Robert Chanock, Albert Kapikian and Robert H. Parrott. Children's Hospital and George Washington University, Washington, D.C., and National Institutes of Health.

Between July 1976 and June 1977, 236 hospitalized patients (116 gastroenteritis "GE" and 120 control "C"), and an outpatient population of 85 patients (36 "GE" and 49 "C") were studied for evidence of infection with bacterial enteric pathogens, enterotoxins and viruses. 46% of "GE" inpatients and 42% of "GE" outpatients, 15% of "C" inpatients and 14% "C" outpatients had potential enteric pathogens. HRV was the most commonly detected agent in gastroenteritis (36% of 152). Using the Y-1 mouse adrenal CA assay, at least 4 isolates from each patient were tested for LT production. 120 were also tested by a radioimmunoassay (RIA) for LT. LT-producing (LT+) coliforms were found in 8% of hospitalized "GE" vs. 4% of hospitalized "C." No difference in LT detection rate was noted in the "GE" outpatient vs. their outpatient "C." LT-producing organisms were found in 7.6% of all gastroenteritis patients and 4% of controls. Among hospitalized HRV negative "GE," 8.4% (6/71) had LT+ as opposed to 3% (3/110) "C" subjects. Approximately 90% of patients with LT+ coliforms were younger than 2 years of age. A limited number of patients have had their sera tested (by a blocking RIA test) for evidence of infection with LT. Only 12% (1/8) have had fourfold seroconversion following illness associated with recovery of coliform producing LT. LT's role in the mediation of gastroenteritis in our population needs to be further elucidated.