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A LONGITUDINAL STUDY OF INTERACTION BETWEEN ENVIRONMENTAL LEAD AND BLOOD LEAD CONCENTRATIONS DURING PREGNANCY, AT DELIVERY AND IN THE FIRST 6 MONTHS OF LIFE. A. Harold Lubin, Albert L. Caffo, Robert Reece (Spon. by Milo D. Hilty) Ohio State University, College of Medicine, Children's Hospital, Department of Pediatrics, Columbus, Ohio.

Sequential evaluations of pregnant women from three cities in the second trimester, at delivery, and of their infants indicate that lead circulating in the bloodstream is readily exchanged between mother and fetus and is present in the newborn. There is no significant change in lead concentration as pregnancy progresses, indicating no hormonal influence within pregnancy as is found in other trace metals. Erythrocyte protoporphyrin concentration is twice normal in the cord and neonate samples, with no correlation to lowered serum iron. Mean blood lead concentrations are lower in maternal, cord, and neonate samples from Columbus than in the same samples from Boston and New Bedford, Mass.

		Mother		Cord Blood		Infant	
	N	Pb	EP	Pb	EP	Pb	EP
Columbus, Oh	46	6±2	27±9	7±3	54±20	9±4	62±25
Boston, Mass	30	8±4	26±12	8±4	54±25	10±3	54±26
New Bedford, Mass	22	9±3	30±12	8±5	64±28	10±4	61±32

Environmental lead concentrations are also lower in Columbus, especially in drinking water, and in dust, to a lesser extent. No abnormally high blood leads have been found in our population, but mothers with levels at the higher range of normal have offspring with concomitant levels. Children will be followed for two years to evaluate the relative contribution of various environmental factors to increases in body lead burden.

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TREND IN NEONATAL BACTERIAL INFECTION AND THE INCIDENCE OF RARE GRAM NEGATIVE (RGN) SEPTICEMIA. H.Modanlou, O.Ortiz & R.Henke (Spon. by D.Mosier). Dept of Neonatology/Pathology, Miller Children's Hospital, Long Beach, CA

A prospective study was designed to investigate the changing trend in neonatal septicemia (positive blood and/or CSF culture) for its type and incidence in our NICU. In neonates meeting the generally accepted clinical criteria for septicemia, blood and CSF cultures were obtained. During a 2-year period 68 were found to have septicemia. Three were excluded because of multiple congenital anomalies. The incidence of gram positive septicemia was the same as gram negative (48vs52%). Overall mortality was 29% with 23&35% mortality in gram positive and gram negative respectively. The predominant gram positive bacteria was beta-hemolytic S. group B (34%) followed by S. aureus (5%). The most common gram negative bacteria was E. coli (17%) followed by K. pneumoniae (14%), RGN (11%) & P. aeruginosa (8%). The 7 RGN septicemias were caused by Citrobacter diversus (3), Acinetobacter anitratus (1), H. parainfluenza (1), P. multicida (1) and S. marcescens (1). Considering that both K. pneumoniae and P. aeruginosa septicemia occurred in neonates with chronic lung dicease while on respirator (12 of 13 pts), the group of RGN constitutes the 3rd most common cause of neonatal septicemia which was associated with meningitis in 43% of cases while overall meningitis was 19%. This study reveals a changing trend in neonatal septicemia with beta-hemolytic S. group B as the most common and the RGN bacterias as more significant pathogens than previously reported. Proper bacteriologic identification of the RGN and use of appropriate antibiotic therapy are emphasized.

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FEVER AND PRESUMED SEPSIS IN INFANTS ≤ 6 MOS. OF AGE Leo R. Muido and Leonard B. Weiner (Spon. by Frank A. Oski) Dept. of Peds., SUNY Upstate Med. Ctr., Syracuse, New York

This retrospective study evaluated the differences and outcome of children ≤6 mos. of age admitted with the diagnosis of fever, possible sepsis and apnea; none with foci of serious infection. There were 44 pts. in Gp. A (bacterial negative disease), 6 in Gp. B (bacterial proven disease), 5 in Gp. C (latent disease) and 17 in Gp.D (apnea). Historical information obtained: age, admission temperature, preadmission duration of illness, incidence of GI or URI symptoms, and concurrent family illness. Lab data included: weight, CBC, chest x-ray, CSF analysis, blood cultures and urinalysis and culture. Mean age was Gp. A: 7.1 wks, Gp. B: 13.2 wks, Gp. C: 17.2 wks, Gp. D: 7.8 wks. Mean temperature was similar in gps. A thru C (38.6°C, 39°C, 39.4°C). Preadmission duration of illness was longer in Gps. B & C. There was no concurrent family illness in Gp. B, vs. 50% in Gp. A. Absolute band counts did not correlate with bacterial disease; 1400/m^3 in Gp. A, 900/m^3 in Gp. B, 2800/m^3 in Gp. C. Absolute neutrophil counts (ANC) were more reliable; Gp. A: 4700/m^3, Gp. B: 7600/m^3. Discharge diagnoses in Gp. B included 2 UTIs and 4 episodes of sepsis. Gp. A included viral illness (21), URI (6), fever (5), gastroenteritis (4) and others. Gp.C included aseptic meningitis (1), otitis media (2), viral pneumonia (2); all developing after admission. Gp. D included apnea (13), URI (4). The incidence of proven bacterial disease was 8.3% and signficant differences in age, preadmission duration of illness, concurrent family illness and ANC were noted.

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MEASLES IN A PARTIALLY IMMUNIZED POPULATION. Martin G. Myers, Laverne Wintermeyer and Donald J. Slymen (Spon. by Robert G. Thompson), University of Iowa Hospitals, Department of Pediatrics, Iowa City.

During the investigation of an outbreak of measles in a partially immunized rural community, a spectrum of illness was observed which ranged in severity from a brief febrile illness to one characteristic of measles. Unimmunized children had more illness ($p = 0.009$) and more measles ($p < 0.001$) than immunized children. However, a large proportion of both the immunized and unimmunized children developed a milder illness epidemiologically and serologically associated with measles. The proportion of immunized and unimmunized children who developed the milder, measles-associated illness was not statistically different ($p = 0.20$).

Employing a trichotomous logistic model, it was demonstrated that the probability of developing both measles and measles-associated illness decreased with increasing age of immunization ($p < 0.001$) but was unaffected by the interval since immunization ($p \geq 0.12$). The age at which immunization could be expected to prevent measles in 95% of cases was 5 years. Reimmunization against measles after 5 years of age may be necessary to effect eradication of epidemic measles. Based upon this model, reimmunization would also be expected to reduce the occurence of measles-associated illness.

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PERINATAL DEATH RELATED TO PREGNANCY HYPERTENSION AND PROTEINURIA. Richard Naeye (Spon. by Nicholas M. Nelson) Pennsylvania State Univ., College of Medicine, Department of Pathology, Hershey, Pa.

The study determined the frequency of placental and fetal disorders responsible for hypertension related fetal and neonatal deaths in a prospective study of 53,518 pregnancies. 3.2% of the pregnancies were placed in the hypertension-proteinuria category when one or more maternal diastolic blood pressures were recorded at 85 mm Hg or more with 1+ or greater proteinuria. The perinatal mortality rate for these pregnancies was 37.9/1000 births and 17.2/1000 for normotensive mothers without proteinuria($P<.01$). 42% of the excess mortality associated with hypertension and proteinuria was due to large placental infarcts, 15% to placental growth retardation, 13% to abruptio placentae and the remaining 30% to other disorders.

The perinatal mortality rate for pregnancies that were characterized by hypertension without proteinuria was 26.6/1000 births. 35% of their excess mortality was due to large placental infarcts, 15% to premature rupture of the fetal membranes, 12% to placental growth retardation, 4% to abruptio placentae and the remaining 34% to other disorders. Thus, overall about two thirds of the excess perinatal mortality associated with maternal hypertension during pregnancy was due to three disorders, placental infarcts, marked placental growth retardation and abruptio placentae. The study found hypertension related lesions in decidual and uterine arteries that appear responsible for these three disorders.

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CAUSES OF THE EXCESSIVE PERINATAL MORTALITY ASSOCIATED WITH PROLONGED GESTATIONS. Richard L. Naeye, Pennsylvania State University, College of Med., Hershey, Pa.

The study analyzed placental structure and the causes of excessive perinatal mortality associated with prolonged gestations using data from a prospective study of 53,518 pregnancies. Postterm infants had a 30% greater frequency of acute inflammation in the subchorionic plate of the placenta than did term infants ($P<.02$). Such inflammation is a marker for amniotic fluid bacterial infections. The postterm placentas did not show any significant increase in those lesions that are characteristic of "aging" or reduced uteroplacental perfusion, i.e. fibrotic villi, excessive syncytial knots, infarcts and decidual necrosis at the margin of the placenta.

The perinatal mortality rate was 20.9/1000 births for infants born after 42 weeks of gestation and 11.7/1000 for those born between 38-42 weeks. The perinatal mortality rate due to congenital malformations was 113% greater, amniotic fluid infections 160% greater, abruptio placentae 100% greater, placental growth retardation 280% greater, placental infarcts 81% greater and umbilical cord knots 88% greater in the postterm than in the term infants. The excess mortality due to congenital malformations was entirely in infants with hypoplastic adrenal glands. Thus, only about one quarter of the excess perinatal mortality associated with prolonged gestations was due to placental disorders that lead to placental insufficiency. The clinical indications for inducing delivery in postterm gestations may need revision.