

● **535** RISING INFANT MORTALITY IN AN AREA OF HIGH ACCESS TO NEONATAL INTENSIVE CARE. L.R. First, P.H. Wise, H. Hersee, J. Rideout, B. Boardman, G.A. Lamb. (Spon. by J.B. Richmond). Harvard Medical School, Boston University Medical School, Children's Hospital, Boston City Hospital, Boston.

Reports from several cities have suggested recent increases in infant mortality (IM). We studied recent trends in Boston, a city where 93% of infants are born in centers with Level III neonatal intensive care. Linked birth/death vital statistics files were analyzed, and infant and maternal medical records for all resident infants who died from 1980 through 1983 were reviewed. The data suggest a plateauing in the survival of low birth weight (LBW) infants and recent increases in high birth weight (HBW) and postneonatal mortality. These increases were related to conditions generally associated with poor access to medical care. Birthweight distributive effects were minimal over this time period.

TRENDS IN BIRTH WEIGHT-SPECIFIC, POSTNEONATAL, AND TOTAL IM

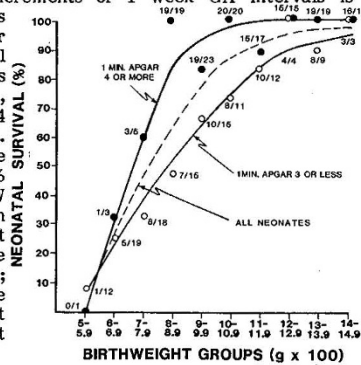
	1969-71	1977-79	1980-81	1982-83
LBW (≤ 2500 g)	138.0	108.0	76.3	83.1*
HBW (> 2500 g)	3.7	1.2	1.1	2.7*
POSTNEONATAL	5.1	3.7	3.1	3.5*
INFANT	21.2	16.4	12.4	13.8*

(rates per 1000 livebirths) * $p < 0.01$

These data suggest that in populations with longstanding access to high quality neonatal intensive care, annual incremental improvements in LBW survival may become minimal. In this setting observed increases in HBW and postneonatal mortality may cause overall infant mortality rates to rise.

536 EFFECT OF APGAR SCORE ON SURVIVAL OF THE VLBW INFANT. David E. Fisher, John B. Paton, Stephen A. Myers, Pritzker Sch. of Med., Univ. of Chgo.; Michael Reese Hosp., Depts. Peds. and OB/Gyn, Chicago, Ill.

To plan appropriate obstetric and pediatric intervention for pregnancies resulting in VLBW infants, the physician needs to be informed of outcome data at the delivering institution. We reviewed 9552 live births from Jan '82 through Aug '84 when our crude neonatal mortality rate was 15.5 and our VLBW rate was 3.8%. Because of rapidly improving outlook for VLBW infants, BW and GA specific mortality analysis by 100g increments or 1 week GA intervals is essential. Of 137 neonates weighing 600-1499g, with Apgar score of 4 or more, the survival was 93%. For 190 neonates between 23 & 32 weeks GA, with 1 min. Apgar score of 4 or more, survival was 94%. When the 1 min Apgar score was 3 or less, survival was 51% and 57% for comparable BW and GA groups. Prolongation of pregnancy is the most important factor to improve outcome for the VLBW infant; when this is not possible assuring optimal condition at birth becomes the highest priority.



† **537** AEROBIC EXERCISE AND ATHEROSCLEROTIC RISK FACTORS IN ADOLESCENTS. Raymond R. Fripp, Robert Winter, James Hodgson, Peter O. Kwitterovich, Victor Whitman, H. Gregg Schuler. The Pennsylvania State University College of Medicine, The Milton S. Hershey Medical Center, Department of Pediatrics, Hershey, PA

The effect of a 7 week aerobic exercise program on atherosclerotic risk factors was assessed in 65 adolescent white males (mean age 15.8 yrs). Each subject was evaluated before and after the program for body weight, body mass index (BMI) (kg/m^2), % fat, systolic and diastolic blood pressure, maximum oxygen consumption (MVO_2), exercise duration (ED) and fasting plasma lipids (cholesterol (CHL), triglyceride (TGL), high density lipoprotein (HDL-C) and low density lipoprotein (LDL-C)). Mean weight (\pm SD) was 70.7 ± 16.5 kg before and 71 ± 16.5 after training (p NS). BMI was 23.2 ± 4.6 and 23.2 ± 4.7 (p NS). % fat decreased by 7.9% from 20.3 ± 6.9 to $18.7 \pm 6.3\%$ ($p < 0.001$). Systolic and diastolic blood pressure remained unchanged, MVO_2 increased by 10.8% from 45 ± 6.5 to 49.9 ± 7.8 $\text{ml}/\text{kg}\cdot\text{min}^{-1}$ ($p < 0.0001$) and ED increased from 20.5 ± 2.6 to 21.1 ± 2.5 min ($p < 0.01$). Plasma lipids (mg/dl) were similar pre and post exercise: CHL 154 ± 32 and 152 ± 31 , TGL 87 ± 46 and 92 ± 40 , HDL-C 46 ± 12 and 45 ± 9 and LDL-C 90 ± 21 and 88 ± 27 . These results demonstrate that with the exception of obesity, atherosclerotic risk factors are not modified by an effective aerobic training program in adolescent males. This is at variance from that reported in adults undergoing aerobic training.

† **538** AN OUTBREAK OF PSEUDOBACTEREMIA CAUSED BY *EWINGELLA AMERICANA*. Sherry Gardner, Kathy Kabat, and Stanford T. Shulman. Dept. of Pediatrics, Northwestern University Med. School, Children's Mem. Hosp., Chicago.

Between September, 1981, and April, 1984, *Ewingella americana* was recovered from blood cultures from 21 patients in the intensive care units and emergency room of a 265-bed pediatric hospital. Because clinical presentations were generally not suggestive of Gram-negative bacteremia, we began an epidemiologic investigation for a source of pseudobacteremia. *E. americana* is a new genus and species in the family *Enterobacteriaceae*, previously known as CDC enteric group 40. It has been reported as a pathogen only once. A case-control study showed that cases were much more likely than controls to have had blood obtained for coagulation profiles with cultures (15/19 vs. 4/39 controls, $p = 3.4 \times 10^{-7}$). Coagulation tubes had been prepared with crystalline sodium citrate and citric acid in distilled deionized water. Blood for both coagulation studies and culture was occasionally instilled into the screw-top coagulation tube before blood culture bottles were inoculated. We hypothesized that if the citrate solution were contaminated, the needle or syringe hub could have transferred *E. americana* to the blood culture bottles, resulting in false positives. *E. americana* was recovered from all of 80 unused coagulation tubes and from no other environmental sources. Personnel obtaining blood for multiple studies should adhere to strict aseptic technique. Laboratories should consider using sterile evacuated coagulation tubes rather than tubes containing potentially contaminated home-made anticoagulant.

† **539** SURVIVAL AND SHORT-TERM OUTCOME OF INBORN "MICROPREMIES". J.S. Gerdes, S. Abbasi, V.K. Bhutani, F.W. Bowen. (Spon: A.M. Bongiovanni). Univ. of Pa. Sch. of Med. and Pennsylvania Hospital, Dept. of Peds., Philadelphia

The mortality and major morbidity of 104 consecutive micro-premie live born deliveries (1982-1984) in an inborn perinatal center was examined by retrospective chart review. "Micropremies" are defined as AGA infants with BW 500-1000gm and < 28 weeks gestation.

Wks. Gest.	24	25	26	27	28	25-28
n	7	19	25	31	22	97
\bar{x} BW (gm)	651	735	740	860	910	814
Survival	0%	39%	58%	83%	76%	67%

There was no difference between survivors (S) and non-survivors (NS) in PROM, C-section, or Apgar scores. There was a significant difference between S and NS for pneumothorax (9% vs 28%) and for mean maximum FIO_2 (.53 vs 1.00). Survival rates by sex and race: white females 61%; white males 38%; black females 90%; black males 70%. Morbidity rates among survivors: Sepsis 20%; NEC 17%; IVH 42% (Grade 3 or 4, 11%); PDA 50%; ROP 64% (Grade 3 23%, Grade 4 1%); BPD 65%; severe BPD requiring $\text{O}_2 > 3$ mos. 18%; apnea 69%; seizures 11%. Mean days on ventilator was 37 (range 0-103), and average length of stay was 93 days (range 61-200), excluding 3 infants who were hospitalized for 6-18 months in chronic care facilities. One infant is blind from ROP; 2 had shunts for hydrocephalus; 1 required tracheostomy for subglottic stenosis. Conclusions: The acceptable prognosis for "micropremies" supports aggressive perinatal management as low as 25 weeks gestation. Race and sex are important determinants of outcome in these infants.

540 FACTORS IN THE POSSIBLE NOSOCOMIAL SPREAD OF *HAEMOPHILUS INFLUENZAE* TYPE b (HIB). Janet R. Gilsdorf and Gerald Herring. (Spon. by Robert P. Kelch) C.S. Mott Children's Hospital, University of Michigan Medical Center, Ann Arbor, MI

Serial nasopharyngeal (np) cultures for Hib were obtained from twenty-three children (ages 2 months to 9 years) with invasive Hib disease during systemic antibiotic therapy (ampicillin, chloramphenicol, or both). During the first 12 hours of antibiotic therapy, 4 of 10 (40%) np cultures were positive; during the first 24 hours of therapy, 6 of 25 (24%) were positive. None of the 74 cultures obtained after 25 hours of therapy (median = 5 days) were positive for Hib. Four of six children who had received no antibiotics at the time of the initial np culture were culture positive and had moderate (21 to 100 cfu/plate) or many (> 100 cfu/plate) Hib present. The positive cultures obtained more than three hours after the first dose of appropriate therapy had rare (< 5 cfu/plate) Hib present.

Reconstruction experiments to investigate the survival of Hib in the environment were performed using 10^3 , 10^5 or 10^7 cfu Hib mixed with tracheal secretions and applied to various surfaces. Using 10^3 cfu, all surfaces tested had no Hib recovered beyond 15 min. after contamination. Using 10^5 or 10^7 cfu, Hib was recovered from stainless steel, plastic laminate countertop and plastic gloves for at least 120 min. and from paper towels up to 15 or 45 min. after contamination. No Hib was isolated beyond 1 min. from cotton sheets.

These results suggest that respiratory secretion precautions for patients hospitalized with invasive Hib disease should be maintained for 48 hours after initiation of adequate antimicrobial therapy to prevent possible nosocomial spread of this organism.