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PRODUCTION OF BILIRUBIN PHOTOISOMERS (PIB) DURING PHOTOTHERAPY (PT) Richard E. McClead, Ronald P. Grogire, Angelo A. Lamola (spon. by Grant Morrow) Ohio State Univ. Col. of Med. Cols. Children's Hospital Dept. of Peds. Cols., Ohio and Bell Labs, N.J.

The Bilirubin Hematofluorometer measures albumin-bound bilirubin (AB) and total blood bilirubin (TB). A risk factor for developing kernicterus is the level of non-AB. The fraction of TB not bound by albumin is defined as $TB-AB/TB=AB$. In two studies we found that AB increased during PT. In 23 jaundiced pre-term infants, mean AB (\bar{AB}) increased during 4 to 24 hours of phototherapy (Pre-PT, $\bar{AB} \pm S.D. = 0.15 \pm 0.10$ and Post-PT, $\bar{AB} \pm S.D. = 0.24 \pm 0.11 = P < 0.005$, by paired t-test). In a study of 17 jaundiced pre-term infants, AB was determined serially during 24 hours of continuous PT; \bar{AB} increased by 0.1 ± 0.1 over Pre-PT values at 4 hours ($P < 0.005$) but declined to baseline by 12 to 18 hours. The photochemical behavior of bilirubin may explain the apparent rise in AB during PT. Radiation at 465 nm converts bilirubin to three water soluble PIB. This isomerization is associated with: an increase in absorbance at 470 to 530 nm (max 495 nm); a 22% decline in fluorescence intensity of PIB mixtures at 520 nm (excitation at 465 nm); and an altered circular dichroic spectrum. We suggest that the increase in AB during PT is due to the formation of albumin-bound PIB which fluoresces less intensely than AB and not to an increase in unbound bilirubin. The decline in AB during prolonged PT is unexplained. Conclusion: By converting bilirubin to photoisomers, phototherapy may reduce the body burden of neurotoxic bilirubin.

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AN ANIMAL MODEL OF NEONATAL SEPSIS: EARLY AND LATE HEMODYNAMIC AND METABOLIC SEQUELAE OF GROUP B BETA STREP (GBS) SEPSIS IN THE PIGLET. W. Meadow & P. Meus (Spon. by M.O. Beem) Dept. of Pediatrics, U. of Chicago & Dept of Surgery & Cardiac Surgery, Michael Reese Hospital, Chicago

Sepsis is 2nd only to respiratory distress as a cause of neonatal death. We have pursued an animal model and continued to study the hemodynamic & metabolic consequences of neonatal sepsis.

Newborn piglets (1-5 wks, n=5) were intubated, paralyzed, and monitored for aortic pressure (AOP), pulmonary artery pressure (PAP), left atrial pressure (LAP), pulmonary artery blood flow, taken as cardiac output (QPA), and mesenteric blood flow (QMES). Pulmonary artery resistance (RPA), mesenteric artery resistance (RMES), and peripheral resistance (RTP) were calculated. LAP was held constant.

GBS were infused I.V. continuously @ $1-5 \times 10^7$ organisms/kg/min. After 30 mins. QPA & QMES fell to 68% & 58% of control, AOP & PAP rose to 108% & 360% of control, and RPA, RTP, & RMES rose to 490%, 150%, & 160% of control. Blood pH and base excess were normal.

During the next 2-4 hours @ GBS infusion rates of $5-10 \times 10^7$ org/kg/min QPA & QMES fell below 50% of control, while PAP rose to 50% of control. At the end of this time period, base excess rose from -2.7 to -21.7, and AOP, which previously had been stable at 90-115% of control, fell precipitously to below 40% of control.

Conclusions: Early-GBS sepsis reveals pulmonary, peripheral, & mesenteric vascular hyper-resistance, diminished blood flow, preservation of systemic blood pressure, & normal acid-base balance. Later, as blood flow continues to decline, metabolic acidosis increases, peripheral vascular hyper-resistance reverses, and systemic blood pressure collapses.

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PHENYLEPHRINE INFUSION AS A MODEL FOR THE HEMODYNAMIC SEQUELAE OF GROUP B BETA STREP (GBS) BACTEREMIA IN THE NEWBORN PIGLET. W. Meadow & P. Meus (Spon. by M. O. Beem) Dept. of Pediatrics, U. of Chicago & Dept. of Surgery and Cardiac Surgery, Michael Reese Hospital, Chicago

Newborn piglets exposed to GBS bacteremia consistently demonstrate vascular hyper-resistance, diminished blood flow, elevated pulmonary artery pressure, and preserved systemic blood pressure. We evaluated the possibility of endogenous alpha sympathetic mediation of these hemodynamic events by observing effects of exogenous phenylephrine (alpha agonist) infusion in the newborn piglet.

Newborn piglets (1-5 wks) were intubated, paralyzed, and monitored for aortic pressure (AOP), pulmonary artery pressure (PAP), left atrial pressure (LAP), pulmonary artery blood flow, taken as cardiac output (QPA), and mesenteric artery blood flow (QMES). Peripheral resistance (RTP), pulmonary artery resistance (RPA), & mesenteric resistance (RMES) were calculated. LAP was held constant.

After control observations, phenylephrine was infused @ 2-8mcg/kg/min. Within 40 mins. QPA & QMES fell to 70% & 69% of control, while AOP & PAP rose to 145% & 118% of control. RTP, RMES, & RPA rose to 215%, 210% & 173% of control. These results were then compared to our previously reported sequelae of GBS infusion.

Conclusions: 1. GBS and phenylephrine both produce comparably diminished QPA & QMES. 2. For the same decrease in QPA, phenylephrine raises AOP more (145% vs 108%) and PAP much less (118% vs 360%) than does GBS. 3. Phenylephrine consistently increases RTP more than RPA (215% vs 173%) whereas GBS raises RPA much more than RTP (490% vs 150%). 4. Hemodynamic sequelae of GBS are likely mediated by more than simply alpha effect.

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ACCURACY OF SKIN-SURFACE CO₂ MEASUREMENT IN SICK PRE-TERM AND TERM NEONATES. T. Allen Merritt, Sunanta Liyamasawad, Christian Boettlich and John G. Brooks (Spon. by Donald L. Shapiro) Univ. of Rochester School of Med., Strong Memorial Hospital, Department of Pediatrics, Rochester, NY

To evaluate the accuracy and clinical usefulness of a heated CO₂ electrode at 43° (Novamatrix Medical Systems) for measurement of skin surface pCO₂ (ssPCO₂), we made 162 simultaneous determinations of ssPCO₂ and arterial PCO₂ (PaCO₂). Arterial samples were drawn from an umbilical artery catheter, and the PaCO₂ was measured within 3 minutes using an Instrumentation Laboratories Micro 13 and Model 326 temperature controller that was calibrated immediately before each measurement. PaCO₂ values were corrected for any abnormal body temperatures. We obtained 2-19 paired samples per infant from 22 infants (gestational age=27-40 weeks) during the first 12 days of life. The skin electrode was calibrated every 4 hours by one investigator, and applied to the infant's chest or abdomen. Over a 4 hour period there was no significant drift of the electrode calibration and no significant change in ssPCO₂-PaCO₂ (Δ). Over the range PaCO₂ 26-78 torr, PaCO₂ was linearly related to ssPCO₂ ($PaCO_2 = .371 ssPCO_2 + 17.46$) ($r = .79$). To pursue the factors which determine Δ , we performed step-wise multiple regression analysis revealing that the only significant independent variables were ssPCO₂ and postnatal age [$\Delta = .79 CO_2$ (torr) - 1.2 age (days) - 23.9 ($r^2 = .86$)]. Insignificant independent variables were blood pressure, gestational age, birthweight, hematocrit, or weight at age of study. Our data support that ssPCO₂ using a heated electrode is an accurate non-invasive method of PaCO₂ measurement in sick neonates.

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CIRCULATORY EFFECTS OF HYPERINSULINEMIA IN THE NEAR TERM OVINE FETUS. J. Ross Milley, Adam A. Rosenberg, Anthony F. Philipps, Richard A. Molteni, M. Douglas Jones, Jr., and Michael A. Simmons. The Johns Hopkins University School of Medicine, Department of Pediatrics, Baltimore, Maryland

Ovine fetal hyperinsulinemia is associated with increased oxygen consumption, increased glucose uptake and decreased arterial O₂ content. We used eight mixed breed ewes with 114-132 day singleton pregnancies to measure the magnitude and distribution of fetal cardiac output in order to define the causes of insulin-induced hypoxemia. During an uninfused control period and again after 12, 24 and 48 hours of exogenous insulin infusion (0.05U/kg/hr), we measured umbilical vein-artery whole blood concentrations of glucose and oxygen. We used the distribution of each of four microsphere injections to determine total cardiac output and organ blood flow. Insulin infusion was associated with increasing insulin concentration following a 12 hour lag period. We confirmed an increase in cardiac output (+19%, $P < 0.02$) and a progressive fall in arterial O₂ content (0.33mM to 0.23mM, $P < .001$). During insulin infusion, blood flow increased to the brain (+50%, $P < 0.05$), adrenal (+56%, $P < 0.01$), and carcass (+44%, $P < 0.02$). Placental blood flow did not change during infusion and fell when expressed as a percentage of cardiac output (45% to 34%, $P < .002$). The hypoxemia during insulin infusion occurs as a result of increased O₂ consumption without a corresponding increase in placental blood flow. If similar effects occur in humans, hyperinsulinemic hypoxia may account for the near term fetal and neonatal mortality among women with poorly controlled diabetes.

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Effects of prolonged and slow-rise inflation pressures on resuscitation of asphyxiated newborn babies. A D Milner, H Vyas, A Boon, I E Hopkin. Cit Hospital, Nottingham, UK.

Previous studies have shown that resuscitation using standard techniques produces a much smaller tidal volume compared to the spontaneously breathing baby, and rarely leads to the formation of a functional residual capacity (FRC). Construction of inflation pressure/tidal volume loops suggested that there were always opening pressures: again very different from the pattern seen in spontaneously breathing babies. Inspection of traces indicated that the lung volume had not stabilised after one second's inflation. We have therefore resuscitated a further group of babies, five born by Caesarean section and four by vaginal delivery, maintaining the initial inflation for a mean of five seconds. This effectively doubled the inflation volume compared to the previous study (from 18.6ml, range 0 - 62.5ml; to 33.6ml, range 16.9 - 70ml). On all occasions this led to the formation of an FRC (mean 15.9ml, range 1.7 - 23.2ml). On five occasions inflation pressure was raised slowly over 2 - 3 seconds. Air started to enter the lungs as soon as inflation commenced in four of the five babies. Our conclusions are:

- 1) that prolonged initial inflation will lead to better lung expansion;
- 2) the apparent opening pressures seen in the previous study were not due to surface tension forces in the lung.