1308 BLOOD PRESSURE MONITORING IN NEONATES: COMPARISON OF UMBILICAL AND PERIPHERAL ARTERY PRESSURE MEASUREMENTS. Warwick Butt, Hilary Whyte, Dept. Ped., U of Toronto & Div. of Perinatal Med. & Res Inst., Hosp for Sick Children, Tor. (Spon. Paul R. Swyer).

Continuous monitoring of blood pressure is now an integral part of modern neonatal intensive care. The most widely practised method is via an indwelling umbilical arterial catheter for which normal values have been established.

In the last few years peripheral artery cannulation has become an increasingly popular technique. Maintenance of blood pressure with volume expansion & inotropes is becoming increasingly recognised as important in the overall management of infants in neonatal intensive care units. As such it is of paramount importance to know whether the normal values established for umbilical arterial catheters can be used for peripheral arterial measurements.

Infants of varying gestational ages (26-39wks), weights (740-3200g) & post-delivery age (days 1-7) with both umbilical & peripheral arterial catheters in place were available for study. (Usually infants with necrotising enterocolitis having an umbilical catheter replaced with a peripheral arterial catheter - radial/tibial). 34 simultaneous pressure recordings were done in 11 infants. There was an excellent correlation between the 2 recordings r=0.98 for systolic + r=0.97 for diastolic pressures. Therefore, normal pressure graphs obtained by umbilical arterial pressure measurements are applicable for peripheral arterial catheters.

1309 IDIOPATHIC APNEA OF INFANCY: POST-NEONATAL SYNDROME. Joan L. Caddell, HGB, NICHD, NIH, Bethesda, MD.

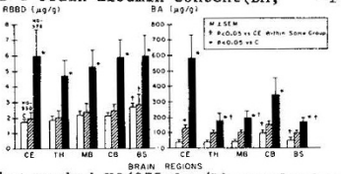
A retrospective study was conducted at NIH and St Louis U Dept. Peds. in 51 infants with post-neonatal apnea who were admitted to Cardinal Glennon Mem. Hosp. for Children, St Louis, between 12-1-74 and 12-31-82. None had received a prior course of magnesium (Mg) supplements, There were two groups: 1) 29 premature infants of 31.5±0.6 wk. gestation age (mean±SEM) and 1462±99 g birth wt., who had idiopathic apnea as neonates, were apparently well when discharged, and suddenly had apnea at 70.6±6 days of age; and 2) 22 infants of 39.9 wk. gestation age and 3293±150 g birth wt., with an onset of apnea at 49.6±12 days of age. Initial findings were similar in 1) and 2), except for lower hemoglobin in 1). Over 50% of the 51 infants had cyanosis, mucus secretions, extremes in heart rate on EKG, respiratory distress, and neuromuscular irritability. Over 20% had intermittent hypertonicity. The clinical chemistry profile often showed a shock pattern: metabolic acidosis, hypoxemia, hemoconcentration, hyperkalemia, hyperchloremia, hyperglycemia, elevated glucose and/or protein in cerebrospinal fluid and urine, and abnormal urinary sediment. These were transient changes; many normalized in 24-48 hrs. About 60% of chest X-rays showed pulmonary infiltrates or hyperinflation. There was usually high retention of an IM Mg load. Four died. Within a narrow time frame, Mg deficient weanling rats experience a syndrome similar to that of the infants in respect to physical, chemical, EKG, and lung changes; and mortality. Mg deficient dog pups have similar attacks. Mg deficiency may be a cause of the syndrome in infants. Mg deficiency is preventable; the matter deserves careful study.

1310 A RETROSPECTIVE EVALUATION OF MAGNESIUM SUPPLEMENTATION IN APNEIC INFANTS. Joan L. Caddell. HGB, Nat'l Inst. Child Health and Human Devel., NIH, Bethesda, MD

A retrospective clinical analysis was undertaken at NIH and St Louis U Dept. Peds. in 249 infants who presented with apnea at 0 to 9 mo. of age at the Cardinal Glennon Mem. Hosp. for Children, St Louis, from 12-1-74 to 12-31-82. The study was an attempt to assess whether or not Mg supplementation was beneficial. There were three groups: 201 premature and 26 term neonates, both with apnea neonatorum; and 22 infants (21 term), who first presented with apnea at 49.6±12 days of age. Of the 249 infants, 83 (about one-third) received a minimum course of magnesium (Mg) supplementation, and 167 did not. Mg was given IM as 50% $MgSO_4 \cdot 7H_2O$, 0.1 ml/kg body wt./day, usually for 5 days; or PO as 10% $MgCl_2 \cdot 6H_2O$, 1 ml/kg/day, usually for 14 days. For purposes of analyzing the data, 5 days of Mg therapy by either route constituted a course for premature neonates, 4 days for others. All premature neonates had apnea, bradycardia, and cyanosis, and 90% had the respiratory distress syndrome; 61 were Mg-treated, 140 were not. More untreated premature neonates received theophylline to control apnea and/or bradycardia ($P<0.0005$). A follow-up study of all infants showed that Mg-untreated infants had more readmissions with apnea ($P<0.0005$) and bradycardia ($P<0.0005$) than Mg-treated. Some degree of respiratory distress was seen in 79%, and neurological findings, in 55% of the readmissions for apnea. Untreated infants had a higher incidence of gastroesophageal reflux ($P<0.0005$). All Mg-treated infants survived; nine untreated infants died, three as crib deaths. Further evaluation of Mg in apnea of infancy can be strongly recommended.

†1311 HYPEROSMOLALITY(HO) AUGMENTED REGIONAL BRAIN BILIRUBIN DEPOSITION(RBBD) IN NEWBORN PIGLETS(P). G.H.Burgess,B.S.Stonestreet,W.J.Cashore,W.Oh. Brown Univ.Women & Infants Hosp.,Dept.of Ped.,Providence, RI.

This study was designed to explore the mechanisms of previously shown enhanced bilirubin brain deposition in urea induced HO. 28 2-4 day old P were infused with bilirubin(B)to raise & maintain serum B(SB) to approximately 8mg/dl. 14 P served as isoosmotic controls(C),7 P received urea infusions to raise serum osmolality to 330mOsm//L(HO330)& 7 P to 375mOsm/L(HO375). During 60 min of HO, SB was similar among the 3 groups(7-9mg/dl). Serum unbound B(UB) was also similar between HO330&C. In contrast, UB was 2.7-fold higher in HO375 vs C($p<0.01$). Microsphere measured regional brain blood flow(RBBF)was similar in cerebrum(CE), thalamus(TH),midbrain(MB),cerebellum(CB),& brainstem(BS)among the 3 groups. RBBD & brain albumin content(BA, ^{125}-I-albumin) are:



We conclude that marked HO(375mOsm/L) resulted in enhanced RBBD probably due to HO-related increase in UB & opening of the blood-brain barrier(BBB) to B. Furthermore, opening of the BBB to B may be more pronounced in the CE than in the other regions studied.

†1312 CONCENTRATION AND FLOW RELATIONSHIPS FOR ENTRY OF UNBOUND BILIRUBIN (BR) IN RAT BRAIN. William J. Cashore and Michael Silberberg. Brown Univ., Women & Infants Hosp., Dept. of Ped., Providence, RI

To estimate the influence of concentration (C), time (t) and rate of flow (F) on unbound bilirubin (BR) uptake across the blood-brain barrier, we injected unbound BR into the carotid artery in 22 adult rats. BR concentrations were between 1 and 10mg%; injection rates were 4.4, 6.0, 8.6, and 12 ml/min.; and injection times were from 10-60 sec. Permeability of BR and capillary surface area were assumed constant. After saline perfusion of the carotid arteries, brain BR was measured by chloroform extraction and diazotization. At $t \geq 20$ sec, arterial unbound BR concentrations (C) $\geq$1mg% produced visible staining of the injected hemisphere. Values of (C x t) uncorrected for flow, were correlated with brain BR: $r = 0.540$, $r^2 = 0.29$, $p<0.01$. Correction of (C x t) for relative flow changes (F) as determined by changes in injection rate improved the correlation of (C x t x F) with brain BR to $r = 0.834$, $r^2 = 0.70$, $p<0.001$. (C x t) accounted for 29% of the variance in brain BR independent of flow, while correction of (C x t) for different injection rates (C x t x F) accounted for 70% of the variance in brain BR. We conclude that whole brain uptake of injected unbound BR in adult rats is related to carotid artery injection rate as well as to arterial concentration of unbound BR and time of exposure. These findings are consistent with rapid entry of unbound BR into the brain when blood flow and the blood-brain barrier are normal, and with the hypothesis that increased brain blood flow may increase brain deposition of BR.

†1313 FATAL EARLY-ONSET GROUP B STREPTOCOCCAL INFECTION WITH A NORMAL CBC. Robert D. Christensen, Gerald Rothstein, Harry R. Hill and Robert T. Hall, University of Utah and Children's Mercy Hospital, Kansas City.

Changes in blood neutrophil (neut) number and in degree of left shift are used as diagnostic clues when neonatal sepsis is suspected. However, we report a pitfall in reliance upon CBC changes. We observed 4 cases of fatal group B streptococcal (GBS) sepsis with a normal neut count (mean 7730;range 6140-9610) and a normal immature|total(i/t) neut ratio (0.04;0.00-0.09). In 2 cases this was used as a reason to withhold antibiotics. In 2, the CBC was repeated prior to death (0 and 150|mm^3). In an animal model of lethal GBS infection, we determined the length of the "latent period" between bacterial inoculation and CBC changes. Neonatal and adult rats received GBS after which blood and marrow were serially examined. The neonates died by 24 hours, but no change in CBC occurred until after 4 hours when an increase in i|t ratio (0.40±0.05, control vs 0.58±0.3 infected, mean±SEM, $p<0.05$) and a decrease in marrow neut (78±0.4% of control, $p<0.05$) was seen. In adult animals, CBC changes were evident after only one hour; i|t ratio increased from 0.25±0.05 to 0.52±0.04 ($p<0.001$) and marrow reserves dropped to 77± 3% of control, ($p<0.05$) consistent with rapid release of marrow neut. These experiments demonstrate a significant delay, in neonates, between bacterial inoculation and mobilization of storage neut. Therefore, CBC changes during infection are delayed. A normal CBC is expected during the first several hours of neonatal sepsis and antimicrobial therapy must not be withheld because the CBC is normal.