

1410 USE OF A PORTABLE TRANSCUTANEOUS OXYGEN MONITOR (TcPO₂ MONITOR) DURING NEONATAL TRANSPORTS.

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647 of 1308 neonatal transports (1979-83) had TcPO₂ monitoring by a battery operated portable monitor. The inspired oxygen concentration (FiO₂) at start and end of transport was compared with the 661 infants who did not have TcPO₂ monitoring during transport.

	FiO ₂	FiO ₂ Changed During Transport	
	Unchanged	(FiO ₂ ↑)	(FiO ₂ ↓)
No TcPO ₂	307	354 (159↑)	(195↓)
TcPO ₂ on transport	159	488 (117↑)	(371↓)

Use of the TcPO₂ monitor resulted in significantly ($p > 0.00001$) more FiO₂ changes during transport. This was especially so with decreases in FiO₂ during transport ($p > 0.00001$). TcPO₂ monitoring, by giving quantitative measures of oxygenation, gives the transport team more confidence in making FiO₂ changes during neonatal transports than by reliance on clinical judgement of oxygenation status alone. This helps in preventing both hyperoxia and hypoxia, and their deleterious consequences to the neonate.

1411 CEREBRAL INTRAVENTRICULAR HEMORRHAGE (CVH) IN INBORN INFANTS 500-1250 GMS AT BIRTH. Savitri P. Kumar,* Endla K. Anday,* Steven G. Gabbe,* and Maria Delivoria-Papadopoulos. Dept. of Peds. and Ob-Gyn, Univ. of PA. Sch. of Med., Phila., PA.

To determine the incidence and to identify factors associated with the development of CVH, 84 infants delivered at the Hospital of the Univ. of PA. from 7/81-12/82 of birth weights between 500-1250 gms and who lived for at least 24 hrs were evaluated for CVH using a linear-array real-time ultra sound scanner. Twenty-four infants (28%) were identified as having CVH. Mild hemorrhage (Gr I or II) was noted in 17/84 infants while 7/84 infants had a Gr III or IV bleed so that 29% of bleeds were significant. The relation of CVH to gestational age & birth weight was examined: 15/40 (37%) infants of 25-28 wks gestation and 10/44 (23%) infants 29-32 wks gestation had CVH ($p = NS$); 13/39 (33%) infants 500-1000 gms and 11/45 (26%) infants 1001-1250 gms had CVH ($p = NS$). Perinatal factors such as PROM, antenatal dexamethasone, Apgar score, or mode of delivery did not affect the incidence of bleeding. However, 20/24 infants with CVH had premature labor (PML) while 29/60 infants without bleed had PML ($p = < 0.01$). Postnatal factors such as severity of RDS, pneumothorax, hypernatremia, hypoxia, hypercarbia, or admission pH were not significantly different in the infants with CVH. In summary, the overall incidence of Gr III or IV CVH among inborn very low birth (VLBW) infants was 8% with the only significant association being with PML. In contrast to outborn VLBW infants where the incidence of CVH is as high as 60%, in the inborn VLBW infants, PML and intrapartum stress seem to be more important factors in the genesis of CVH because the availability of immediate neonatal resuscitation minimizes postnatal factors which may also be involved in the development of CVH.

1412 WEANING OF PREMATURES FROM A NEUTRAL THERMAL ENVIRONMENT TO AMBIENT THERMAL ENVIRONMENT.

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The ability of a newborn to maintain temperature within the normal range in ambient air is one of the criteria used for discharge. At present, there are no clear cut criteria as to when a growing premature can be safely moved into an open crib.

This study was conducted prospectively in healthy growing prematures who had attained a weight of 1500 gms. and who were taking 120 to 150 cal/k/day of a 20-cal/oz formula. Infants were divided into two groups assigned alternately. Group I infants were weaned from the incubator at 1700 gms. and Group 2 were weaned at 1800 gms. Any infant whose axillary temperature was below 36c in the crib was considered a failure. Daily caloric and fluid intake were recorded. The length of hospitalization from the time of entry into the study (1500 gms.) to the time of discharge were compared.

Thirty infants are included to date. Preliminary findings show that infants in Group I stayed an average of 5.4 days less than those in Group 2. None of the babies from either group lost weight after weaning. Weight gain ranged from 0 to 70 gms. Two babies from each group were considered failures. (Supported by March of Dimes, Birth Defects Foundation, Summer Sciences Research Programs for Medical Students).

1413 EFFECTS OF NEONATAL HYPERTHYROIDISM ON SOMATIC GROWTH AND ON SUBMANDIBULAR GLAND AND SERUM-EPIDERMAL GROWTH FACTOR CONCENTRATION IN ONE MONTH OLD MICE. J. Lakshmanan, U. Beri, J. Perheentupa, T. Macaso. H. Kim, D.A. Fisher. Harbor-UCLA Medical Center, UCLA School of Medicine, Department of Pediatrics, Torrance, CA

We examined the consequences of neonatal hyperthyroidism on somatic growth and SMG- and serum-EGF levels in one month old female mice. Littermate pups were randomly divided into control (vehicle injected) and thyroxine (T4) treated (0.4 ug/g BW/day) groups. Animals were treated with vehicle or T4 from birth (day 0) through day 6 and sacrificed on day 31. T4 treatment resulted in acceleration of both tooth eruption and eyelid opening, and in addition, caused growth retardation (Body weight: control vs T4-treated 22.3±0.5g and 16.0±0.8g $P < 0.001$; and nose to rump length: control vs T4-treated (cm) 9.3±0.1 and 8.2±0.1 $P < 0.001$). SMG and serum EGF concentrations were measured by RIA with results as follows:

Group	Treatment	SMG-EGF (ng/mg Protein)	Serum-EGF (pg/ml)
A	Vehicle	224 ± 41	219 ± 9
B	Thyroxine	5 ± 1*	384 ± 61*

* $p < 0.001$

T4 treatment inhibited the normal ontogenic rise in SMG-EGF and increased serum EGF levels. Conclusion: Neonatal hyperthyroidism accelerates maturation but inhibits growth. Speculation: This may be due to augmented tissue EGF production while SMG maturation is impaired.

1414 DELAYED FEEDINGS FAILS TO PREVENT NECROTIZING ENTEROCOLITIS IN VERY LOW BIRTH WEIGHT NEONATES. H. Birenbaum, S.G. Ostertag, E.F. La Gamma, (Spon. P.A.M. Auld), Perinatology Ctr., N.Y.H.-Cornell Med. Ctr., N.Y.

To test the hypothesis that delayed feedings would lower the incidence of necrotizing enterocolitis (NEC) in infants less than 1500 g, we compared the incidence of NEC in two matched groups of newborns. High risk infants (38) were selected from 160 consecutive admissions over a 1 year period, based on cumulative risk scoring of their illness during the first 3 days of life. One group (N=20) was held NPO for two weeks, receiving nutrition parenterally, while the other (N=18) was begun on incremental enteral feedings of dilute infant formula or breast milk. During the first 14 days of life the incidence of NEC in the NPO group was 40% (8/20) compared to 5% (1/18) in the fed group ($p < 0.02$). The incidence of NEC in the second 14 days of life, when all infants were receiving enteral nutrition, was 16% (2/12) and 17% (3/17) for the NPO and fed groups respectively. After 28 days, an additional 20% (2/10) of the originally NPO group acquired NEC, while there were no further cases in the fed group (0/14). Overall, the incidence of NEC was 60% (12/20) in those infants held NPO early in life and 22% (4/18, $p < 0.02$) for those fed early in life (initiation of feedings $\bar{x} = 7 \pm 2$, range 4-11 days). In other words, of the cases of NEC observed, 56% (9/16) occurred in infants who were never fed. These data show that being held NPO for 2 weeks postnatally does not lower the incidence of NEC and in fact may promote its occurrence. Thus, early dilute, incremental feedings may paradoxically serve to protect the immature gut.

1415 EFFECTS OF NaHCO₃ ON CEREBRAL BLOOD FLOW (CBF) DURING HYPOXIA AND ACIDOSIS IN NEWBORN PIGLETS. Abbot R. Luptook (Spon. by C.R. Rosenfeld), Southwestern Med. Sch., Department of Pediatrics, Dallas, Texas.

A reciprocal relationship exists between CBF and arterial O₂ content (CaO₂) during hypoxic hypoxia. Prolonged hypoxia may be complicated by metabolic acidosis further decreasing CaO₂. To investigate if NaHCO₃ lowers CBF by increasing pH and CaO₂, CBF (microspheres), osmolarity, and arteriovenous differences of O₂ content and blood gases were obtained in 14 ventilated piglets. Measurements were obtained during control (C), prolonged isocapnic hypoxia (50 min) to produce metabolic acidosis (H+A), and following infusion of saline (Gr1, n=6) or 2mEq/kg NaHCO₃ (Gr2, n=8) during hypoxia. At C pH was 7.44±.01(\bar{x} ±SE) in Gr1 and 7.41±.01 in Gr2. H+A resulted in similar reductions in pH (Gr1=7.21±.02 and Gr2=7.21±.03), but differences occurred after the infusions, (Gr1=7.15±.03 vs. Gr2=7.30±.03, $p < .05$). In each Gr CaO₂ paralleled pH but did not differ between Gr. CBF (ml/min/100g) in Gr1 was 120±17 at C, increased to 287±60* during H+A and 259±31* during saline. Similarly, CBF rose in Gr2 from C of 116±12 to 251±23* during H+A, and 221±34* with NaHCO₃. Although cerebral O₂ delivery, extraction, and uptake changed (due to the extent of CaO₂ reduction), both Gr were comparable. No changes in osmolarity occurred. It appears that the magnitude of change in pH induced by 2 mEq/kg NaHCO₃ does not alter CBF. It is speculated that even larger increases in pH produced by NaHCO₃ may not affect CBF due to changes in O₂ affinity. (* $p < .05$ vs. control).