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PREVALENCE OF CENTRAL APNEA IN PREMATURE NEWBORNS. Jose Lopes, Nestor Muller, Margaret H. Bryan, and A. Charles Bryan. Research Institute, Hospital for Sick Children, Toronto, Ontario, Canada.

It has recently been suggested that most apneas observed in preterm infants are either mixed or obstructive. However, our results indicate that the majority of the spells are central. We studied 10 preterm infants, BW = 1226 ± 196 gr, gestational age 29 ± 1.6 weeks, and postnatal age 19 ± 10 days (mean ± S.D.). Seven out of the ten infants had intracranial hemorrhage (ICH) diagnosed by ultrasound. We recorded diaphragmatic EMG activity with surface electrodes, motion of the rib cage and abdomen with magnetometers and air flow using a nasal thermistor. Apnea was defined as cessation of air flow for more than 20 sec, or more than 10 sec, if followed by bradycardia (H.R. < 100 bpm). Spells of absent airflow with diaphragmatic EMG activity were called obstructive (O), without EMG activity (C), and spells with both patterns mixed apnea (M).

Infants	(C)	(M)	(O)	Total
No ICH (3)	9	4	0	13
ICH (7)	41	7	9	57

In only two infants (with ICH) was the predominant type mixed apnea. Our results are not in agreement with previous data suggesting that all apneas in infants with ICH are mixed or obstructive. We conclude that apneas in preterm infants are predominantly central.

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EFFECT OF WATER BALANCE ON CLINICAL OUTCOME IN LOW BIRTH WEIGHT (LBW) INFANTS. John M. Lorenz, Leonard I. Kleinman, Uma Kotagal, U. of Cincinnati Coll. of Med., Dept. of Pediatrics

The clinical effects of fluid inputs designed to maintain different degrees of negative water balance during the first 5 days of life were determined prospectively in 68 LBW infants. AGA infants with birth weights of 750-1500 g were matched for birth weight (BW) in 250 g increments, RDS or no RDS, asphyxiated or not, and inborn or outborn. Each infant was randomized to either Group I -- fluids managed to allow 1-2% loss of BW per day to a maximum loss of 8-10%, or Group II -- fluids managed to allow 3-5% loss of BW per day to a maximum loss of 13-15%. The mean 5 day cumulative fluid input in Group II was 230 ml/kg less than in Group I, yet Group II lost only 45 g/kg more than did Group I (8% of BW lost in Group I vs 12.5% of BW lost in Group II, $p < 0.01$). There were no statistically significant differences ($p > 0.20$) in incidence of clinically significant PDA, intracranial hemorrhage, BPD, NEC, hypernatremia, excessive weight loss ($> 20\%$ BW), mortality, time on O_2 or ventilator, or time to discharge. Group II had a higher incidence of hypoglycemia (serum glu < 40 mg%) than Group I (12/34 vs 1/34, $p < 0.001$). Hypoglycemia occurred only in infants receiving less than 5 mg/kg/min glucose for at least 2 days. We conclude that fluid input in LBW infants can be flexible to allow 1-5% loss of BW per day to a maximum loss of 8-15% during the first five days of life, as long as more than 5 mg/kg/min of glucose is provided.

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ABILITY OF LOW BIRTH WEIGHT (LBW) INFANTS TO MAINTAIN FLUID HOMEOSTASIS. John M. Lorenz, Leonard I. Kleinman, Uma Kotagal, Univ. of Cincinnati

The ability of the LBW infant to control water balance during the first 5 days of life was studied prospectively in 68 infants. Matched AGA infants with birth weights (BW) of 750-1500 g were randomized to either Group I -- fluids managed to allow loss of 1-2% of BW per day to a maximum loss of 8-10%, or Group II -- fluids managed to allow loss of 3-5% of BW per day to a maximum loss of 13-15%. All infants were cared for in convectively heated incubators with 80-95% relative humidity and abdominal skin temp maintained at 35.5-36.0°C. Mean fluid inputs were as follows:

	Fluid Input (ml/kg/day)					5 Day Cumulative
Day	1	2	3	4	5	Input (ml/kg)
Group I	84	98	125	141	144	594 ± 24 (SE)
Group II	68	68	68	76	83	366 ± 12 (SE)

Group I lost a mean of 80 g/kg and Group II 125 g/kg over 5 days. Mean cumulative caloric input in Group I was 216 ± 10 Cal/kg and in Group II 143 ± 5 Cal/kg ($p < 0.01$). When weight loss was corrected for caloric loss (assuming caloric expenditure of 45 mg/kg/day and 4.8 cal/gm dry tissue metabolized), cumulative water loss was 78 ± 6 g/kg in Group I and 110 ± 10 g/kg in Group II ($p < 0.02$). A 230 ml/kg difference in fluid input was required to produce a mean difference in total body water of only 32 g/kg. Thus the LBW infant is able to maintain water balance within a comparatively narrow range over a broad range of fluid inputs by regulating water excretion. This ability to handle higher fluid inputs relatively well allows higher caloric input.

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OUTCOME OF INBORN VS. TRANSPORTED HIGH-RISK INFANTS. Lula O. Lubchenco, L. Joseph Butterfield, Virginia D. Black, Edward Goldson, Catherine S. Manchester and

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The outcome of 694 births of infants weighing < 1500 grams from two sources was compared. 301 births occurred in 5 metro area hospitals (MH) with Level I nursery facilities; the high-risk infants were transported to Level III nurseries. 393 infants were born in University Hospital (UH) where Level III intrapartum and neonatal care are available. Fetal deaths were twice as frequent in the MH population as in the UH group, 25 vs. 12.5% ($p < 0.01$). Neonatal deaths were slightly increased in the MH, 37 vs. 30% but not to a significant degree. Infant deaths occurred in 3 and 4% respectively in MH and UH. Outcome at approximately 12-24 months was the same in both groups. Because 61% of the UH population in this weight group comes from in utero transport patients from the region, this population was reviewed. Fetal deaths occurred in 9.5% of total maternal transport births. Neonatal deaths were similarly reduced (27%) and outcome slightly improved 33%. When the in utero transport patients from the MH were credited back to these hospitals the fetal death rate was 22% compared to the UH rate of 17% ($p = NS$). Maternal transport patients constitute a population which differs from that of the MH and UH. Data on maternal complications, neonatal morbidity, birth weight, gestational age, socioeconomic status, type of transport and long-term outcome in relation to these factors is in preparation.

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CEREBRAL AND RETINAL HYPOPERFUSION AS A POSSIBLE CAUSE OF RETROLENTAL FIBROPLASIA - HYPOTHESIS TO EXPLAIN NON- O_2 RELATED R.L.F. Jerold F. Lucey, Jeffrey D. Horbar, and M. J. Onishi. Department of Pediatrics, Univ. of Vermont College of Medicine, Burlington, Vt. 05405

Retrolental fibroplasia has now been reported to occur in over 190 infants in which oxygen administration was not a factor. Ashton injected glass ballotini into kitten retinal arteries and produced vasoproliferative changes "exactly as occur in retrolental fibroplasia." (Brit. J. Ophthalmol. 49:225, 1965) He demonstrated that vasoproliferation is not due to O_2 toxicity per se but is "merely a sequel of the obliteration of the vessels induced by oxygen." Our hypothesis is that R.L.F. may develop in very low birth weight infants who have cerebral/retinal hypoperfusion/ischemia, at birth or in the neonatal period. These infants have impaired cerebral blood flow autoregulation, and a high incidence (40-80%) of intracranial bleeding. This hypothesis would explain the high incidence of non- O_2 related R.L.F. in anencephalic infants and the rising incidence in very low birth weight infants < 1000 grams with intracranial hemorrhage. We have observed two infants with severe R.L.F., whose clinical course (asphyxia, shock, intracranial bleeding) supports our hypothesis. In one infant cerebral hypoperfusion was documented. Imperfect $TcPO_2$ and arterial blood gas monitoring did not reveal prolonged significant hyperoxemia. Our observations suggest that hyperoxemia is not the only cause of R.L.F., cerebral hypoperfusion and retinal ischemia may produce the same picture. Careful O_2 monitoring alone will probably not eliminate R.L.F.

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HYPOTHERMIA IN TRANSPORTED NEONATES: REWARMING EFFECT OF RADIANT HEAT VERSUS WARMED AIR INCUBATORS. Andrew Macnab, Edward Lau, Marilyn Kaga & Margaret Pendray. (Spon by Syd Segal) University of British Columbia, Vancouver B.C.

Of 231 neonates transported to this centre in the last year 64 (28%) had a core temperature of $< 36^\circ C$ when examined prior to transport. Two types of transport incubator are in use; a conventional system with circulating warmed air (WA) and one using a radiant heat (RH) source. Allocation of incubators depends on availability and is largely random. Review of transport records shows 27 (42%) of the hypothermic infants rewarmed in transit with a rise in temperature above $36.5^\circ C$. 10 (25%) of the 40 infants nursed in WA units rewarmed and 17 (70%) of the 24 transported under RH. In cold babies WA unit environmental temperature is set $2^\circ C$ above skin temperature and the skin servo of the RH unit at $36.8^\circ C$. Mean weights for rewarmed and persistently cold groups were comparable, as were time of day, season and mean duration of transport. Lowest incidence of rewarming was on short transports (< 2 hours from initiating transport care to admission). In 2 infants who failed to rewarm core temperature was unrecordable prior to transport and with 2 others equipment malfunction occurred. 6 (20%) of those who remained cold in WA units actually lost body heat in transit. Neonatal mortality was 17% in the rewarmed compared with 37% among those who remained cold. Although initial morbidity and management are relevant, among hypothermic infants transported to this centre those nursed in radiant heat transport incubators appear to have a greater potential for re-warming than those in warmed air units, and those that do rewarm have a lower neonatal mortality than those who remain cold.