1265 COMBINED CARDIOTONIC AND VASODILATOR THERAPY FOR MYOCARDIAL DYSFUNCTION ASSOCIATED WITH HYPERTENSION IN ASPHYXIATED PREMATURES.

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Infants with acute heart failure due to perinatal asphyxia may have elevation of systemic blood pressure from adrenergic stimulation. This afterload elevation is probably deleterious for myocardial function and tissue perfusion. Treatment needs to be directed towards increasing cardiac contractility and reducing afterload. Dopamine and chlorpromazine were given at 2-8 and 1-2 ugm/kg/min respectively, to 6 preterm infants with hypertension following severe perinatal asphyxia. This therapy improved hemodynamics in all infants: the heart rate did not increase significantly, mean arterial pressure decreased from 52±4 to 46±4, and skin PCO_2, obtained with an unheated electrode, decreased by 8%. These results suggest that improvement in hemodynamics and tissue perfusion occurred without production of myocardial stress by tachycardia or hypotension. The use of an inotropic agent alone for heart failure in the presence of hypertension, may worsen the condition. The combination of dopamine, inotropic and moderate chronotropic agent with chlorpromazine, rapidly active, short acting vasodilator, successfully treated acute hypertension and transient left ventricular dysfunction. The association of hypertension and heart failure in asphyxiated preterm infants may be neurogenic or of vascular origin. In both, appropriate choice of therapy may be significant determinor of outcome.

1265A OXYGEN TENSION ($tcPO_2$) HEART RATE, BREATHING, AND SLEEP DURING INFANCY FOLLOWING PRETERM BIRTH.

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Five six-hour daytime studies on each of 13 preterm infants, 6 healthy (HPTI) and 7 with previous persistent oxygen dependency (PPOD) were carried out between 36 weeks GA and six months post term and compared with those in normal term infants (NTI). All babies showed a rise in heart rate between term and one month with a subsequent fall to six months. Only the early heart rates were higher in preterm infants and highest in PPOD infants [e.g. at Term. NTI 116±2.9 QS 123.7±2.1 AS; HPTI 136.9±1.5 QS 140.5±1.9 AS; PPOD 151.7±2.8 QS 154.7±2.8 AS]. Heart rates were always higher in active (AS) than in quiet sleep (QS). Respiratory rates were only higher at term in HPTI and PPOD [42±3 QS 43±2.5 AS; NTI 32.7±2.2 QS 35.8±1.8 AS]. $TcPO_2$ rose during the first month, post term, was highest at term in HPTI; and was lower at all ages in PPOD infants. Only in HPTI at 36 weeks GA was $tcPO_2$ lower in active sleep. No NTI or HPTI had $TcPO_2$ < 50 torr (64 studies) whereas 11 of 34 studies in PPOD were < 50 torr: 7 of these were lower in QS. Some preterm infants following PPOD may have lowered oxygen tensions during sleep for several months [eg 3 months post-term PPOD 58.4±2.8 torr vs healthy 78±3.4 torr]. Neither tachycardia nor tachypnea or apnea correlated with this. Interstitial lung disease or diminished oxygen chemosensitivity may be the cause. Future sleep-related respiratory disorder and intellectual impairment should be considered. FSSID NO. 30.

1266 TRANCUTANEOUS pO_2 ($TCpO_2$) DURING INTRAVENOUS FAT INFUSIONS IN LOW BIRTH WEIGHT INFANTS.

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Trancutaneous pO_2 was measured continuously during consecutive control and infusion periods of equal length in low birth weight infants given intravenous fat emulsion at a rate of 195 ± 24 mg/kg of triglyceride per hour for 5 hours. The infants averaged 1000 gms. (range 750-1480 gm) and 28.1 weeks gestation (range 25-31 wks.) at birth, were studied at age 7-21 days, and were on stable FIO_2 and/or ventilator settings during the study periods. Plasma triglycerides were measured before the infusion, at 3±0.5 hours during the infusion, and 4 hours post-infusion for each patient. $TCpO_2$ values for each patient were matched at corresponding 30 min. intervals (i.e., at 0-30 mins., 30-60 mins., etc) for 69 control and 69 infusion periods of 30 mins. each, and the matched periods were analyzed by a paired t-test, as shown:

Triglycerides, mg/dl		$TCpO_2$, mmHg	
Before 39±18*	$p<0.001$	Control (n=69	62.4±12.0* $p=n.s.$
During 93±22	$p<0.001$	Infusion (n=69)	62.5±14.3
After 56±19		*Mean S.D.	

In 6 infants breathing spontaneously, respiratory rates were 55± 11/min. during control periods and did not increase during fat infusions. Apneic spells occurred during 11% of control periods (8/69) and 7% of infusion periods (5/69). Low birth weight infants>1 week of age and in stable respiratory condition appear to tolerate intravenous fat at 200 mg/kg/hr with no clinically detectable effects on respiratory rate, frequency of apnea, or O_2 transport to tissues as estimated by $TCpO_2$.

1267 ENDOGENOUS OPIATES CAUSE NEONATAL DEPRESSION FOLLOWING FETAL ASPHYXIA.

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Naloxone, a specific opiate antagonist, greatly modifies the ventilatory response to asphyxia in the newborn rabbit (Chernick et al, Ped. Res. 14:357, 1980). Do endogenous opiates affect the neonatal adaptation to birth following fetal asphyxia? Pregnant does (day 30) were given naloxone (1 mg/kg I.V.) or saline (2.5 ml I.V.), and 5 minutes later placed in a chamber containing 7% CO_2 in N_2. The does expired at 3-4 min., the fetuses were delivered at 10 min., and kept warm by a heating lamp. A 'blinded' observer assessed the pups at 1,3,5,10,15 and 30 min. post delivery using a scoring technique for respiration, color, muscle tone, response to stimulation and general activity. Pups in the naloxone group (n=27) had significantly higher scores than those in the saline group (n=24) for the first 15 min. of life but by 30 min. the difference was not significant. Similar experiments were done in which pregnant does were given 7% CO_2 in N_2. At 10 min. I.P. fetal injection through the uterine wall of either saline or naloxone (0.5 mg/kg) was done and the fetus delivered. However, saline injected pups had high scores even at 1 min. and these scores remained high and not different from naloxone treated pups. Thus, endogenous opiates cause neonatal depression during the first 15 min. after birth following fetal asphyxia and this depression is reversed by naloxone and intraperitoneal injection per se. (Supported by M.R.C. Canada and the Children's Hospital of Winnipeg Research Foundation Inc.)

1268 A LONGITUDINAL ASSESSMENT OF THYROID FUNCTION IN THE VERY LOW BIRTHWEIGHT (VLBW) INFANT.

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Assays of serum T3, T^4 and TSH were performed on cord blood & then serially at 3, 24 and 72 hrs; at 1 week and weekly until 6 wks in 11 VLBW infants(<1200gm). In response to parturition,there was a TSH surge(cord 7.1 ± 3.6mu/ml; 3 hrs 8.7 ± 5.1mu/ml)and elevation of T^4(cord 7.1 ± 1.8µg/dl; 3 hrs 9.3 ± 3.6µg/dl). TSH then fell to a nadir at 72 hrs(2.1 ± 1.0mu/ml); T^4 at 3 wks(4.1 ± 2.3 µg/dl). Subsequently T^4 increased; at 6 wks(5.3 ± 2.7µg/dl). T3 closely paralleled T^4 with a nadir at 2 wks of 66.1 ± 36.2ng/dl, rising to 106.9 ± 22.0 at 5 wks. Analysis of data revealed significantly lower($p<.05$)T^4 in infants with RDS(n=5)vs those without RDS(n=6)at 24 and 72 hrs as well as at wks 1, 2, 3 and 6. T3 and TSH were not significantly different in these groups. Comparison of survivors(n=8)vs non-survivors(n=3)revealed significantly lower T^4 in the latter at 3 hrs($p<.05$), 2 wks($p<.005$), 3 wks($p<.005$) and 4 wks($p<.05$). T3 and TSH were not statistically different. These normative data are in general agreement with previously reported values in larger preterm infants, reflecting the pattern in fullterm infants albeit of a significantly less degree. No treatment is recommended for preterm infants with low T^4 since these values increase to normal over several weeks. Reassessment of this recommendation seems warranted in light of the above which indicates some of these infants do not recover; their relative hypothyroid state may not be a physiologic response to their illness but significantly related to morbidity and mortality.

1269 POSSIBLE RELATIONSHIP BETWEEN MATERNAL ANESTHESIA AND NEONATAL JAUNDICE: EFFECT IN VITRO OF ANESTHETICS ON THE NEONATAL RED CELL.

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Neonatal jaundice has been correlated with use of anesthetics in the mother. These membrane-active agents cross the placenta & lead to measurable blood levels in the newborn. We investigated the effect in vitro of 3 commonly used agents on filterability of neonatal red cells (RBC). Cord blood was obtained from 10 infants whose mothers had received no medication or anesthetics prior to or during labor. RBC were washed and resuspended to a hematocrit of 5% in pH 7.4 Kreb's phosphate buffer, & incubated for ½ hour at 37°C with either buffer or anesthetic agent (1 or 2 µg/ml). Filterability was tested using a modification of Teitel's method.

INCUBATION MEDIUM	FILTRATION $T_{½}$ (Min.,Mean ± SD)	p VALUE
Buffer	6.0 ± 1.2	--
Mepivicaine	5.8 ± 1.5	N.S.
Lidocaine	6.4 ± 0.9	N.S.
Bupivicaine	9.7 ± 1.5	<.001

For the 3 agents tested, no significant difference was seen in $T_{½}$ at the 2 concentrations tested. At both 1&2 µg/ml, the $T_{½}$ of bupivicaine-treated RBC was significantly prolonged when compared with buffer-treated RBC. These results complement available clinical information linking the use of bupivicaine with neonatal jaundice, and suggest that this agent may be adversely affecting the deformability of neonatal RBC, leading to premature red cell destruction.