†1435 EFFECT OF VARYING HEAD POSITION ON CEREBRAL 31-P NUCLEAR MAGNETIC RESONANCE (NMR) SPECTROSCOPY.

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Previous studies in neonates have reported changes in intracranial pressure (ICP) and internal jugular vein (IJV) outflow following changes in head position. To determine the effect of varying head position on cerebral metabolism in healthy infants, brain spectra of phosphorus-containing compounds by 31-P NMR were measured in 6 term and 4 preterm, appropriate-for-gestational-age infants, each with their head in various positions: Body in right and left lateral position, head neutral; supine, head turned 90° right; prone, head turned 90° left. Spectra of phosphomonester (PME), inorganic phosphate (Pi), phosphodiester (PDE), phosphocreatine (PCr) and ATP were obtained using a 2 turn surface coil. PCr/Pi, a critical measure of bioenergetic reserve, intracellular pH, and PME/βATP were calculated. The mean value for PCr/Pi in the term babies in neutral position was, left = 0.93, right = 1.08. The PCr/Pi following head rotation 90° to the right and in prone position with head 90° to left, was 1.05 and 1.04, respectively. The PME/βATP in term infants was left = 1.47 and right = 1.81, and after 90° head rotation while supine, 4 of 6 infants showed a $\geq$10% decrease in PME/βATP. Preterm infants with head in neutral position had a mean PCr/Pi of 0.99 and 0.91 on the left and right, respectively. Following head rotation, PCr/Pi was 1.11 and in the prone position it was 0.85. Intracellular pH showed no correlation with changes in head position. These data suggest that alterations in ICP or IJV which may accompany changes in head position are not of sufficient magnitude to affect cerebral metabolism in healthy term and preterm infants.

†1436 EFFECT OF POLYCYTHEMIA ON FIBRINOGEN DISAPPEARANCE RATE IN PUPPIES.

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I have shown in previous work that polycythemia in the newborn dog will cause a disease similar pathologically to necrotizing enterocolitis(NEC) in human infants. To explore the pathophysiology of this disease, the effect of polycythemia on fibrinogen disappearance rate was studied in 38 puppies (3-14d). All pups received an exchange transfusion removing 65cc/kg of blood and transfusing 85cc/kg of either whole blood (control, C, resulting Hct.=37), or packed red blood cells(polycythemia, P, resulting Hct.=68). NEC was found in 15 of 19 P and 4 of 19 C pups($p<.01$). The concentration of Fibrinogen degradation products measured by tanned red cells hemagglutination inhibition at 2 & 4hr. post transfusion was 36% higher in the P than the C pups (NS). 125I Fibrinogen and Evan's blue (an albumin marker) were injected 2hr after transfusion and the concentration of clottable labelled fibrinogen and albumin tracer were measured at ½ hr & 2 hrs after injection. The fraction of the tracer that disappeared over the 1½hr period was calculated. In previous experiments pups have been shown to have stable plasma volumes during this period. In the P group 45±18 SD% of the clottable fibrinogen disappeared vs only 28±15% in the C group ($2p<.01$). In the P group 36±21% of the albumin tracer disappeared vs 31±12% in the C group(NS). Thus polycythemia in the newborn dog is associated with an increased disappearance of clottable fibrinogen not associated with a general increase in protein disappearance rate. The most sensative currently available test for fibrinogen degredation products was inadequate to demonstrate this event.

●1437 THE ROLE OF BLOOD COAGULATION IN THE PATHOPHYSIOLOGY OF NECROTIZING ENTEROCOLITIS IN POLYCYTHEMIC PUPPIES.

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We have shown that polycythemia can cause necrotizing enterocolitis in the newborn dog. In order to determine the role of blood coagulation in this syndrome the following experiment was performed. Seventeen newborn (3-14 day old) dogs were made polycythemic by a transfusion of 50cc/kg of adult dog packed red blood cells. Group one (9 pups) received 500 units/kg heparine prior to the transfusion and 50 units/kg/hr thereafter. Group 2 (8 pups) received only the transfusion and served as controls. Both groups of pups were fed puppy formula at 8cc/kg/hr beginning 1 hour after the transfusion. Hematocrit and viscosity were similar in the two groups after transfusion: Hct 69 ± 5SD in group 1 and 68 ± 5 in group 2(NS); viscosity at 115/sec, 9.4 ± 2.0 in group 1, 9.2 ± 1.5 in group 2(NS, also NS at other shear rates). Necrotizing enterocolitis was diagnosed at 24 hr after transfusion or at death by autopsy. Necrotizing enterocolitis was seen in 0 of 9 heparinized pups and 4 of 8 control pups ($p<.05$, Fisher's exact test). An intact coagulation system is critical to the pathogenesis of necrotizing enterocolitis in polycythemic puppies.

1438 APNEA AND VENTILATORY CORRELATES DURING THE NEONATAL PERIOD AND EARLY INFANCY.

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We studied 5 term infants (BW 3.57±0.2 kg; GA 40±0.4 wks) and 5 preterm (BW 2.14±0.25 kg; GA 34±0.5 wks) on 5 to 7 occasions each during the first 3 months of age. A total of 59 epochs (1062 min) of quiet sleep (QS) and 54 epochs (660 min) of REM were analyzed in term infants; 71 epochs (1437 min) of QS and 69 epochs (879 min) of REM in preterm infants. In term infants 80% of apneas (>3 sec) were central, 1.8% obstructive, 0.4% mixed and 17.8% of the breath-holding type. In preterm infants 96% were central, 0.5% obstructive, 1.7% mixed and 1.8% breath-holding. The number of apneas decreased from 140/hr at 5 days of age to 30/hr at 120 days in preterm infants; in term infants apneas per hour changed from 10 at 5 days to 20 at 30 days, and to 5 at 120 days ($p<0.025$). The number of apneas/hour was similar in both sleep states. In QS, $\dot{V}_E$ was 0.157 L/min/kg and 0.153 at 10 and 120 days; V_T was 6.7 ml/kg and 6.2; f was 24 breaths/min and 26; and P_ACO_2 was 41 mmHg and 40. P_AO_2 and $TcPO_2$ increased with age in preterm infants only ($p<0.05$). These findings suggest; 1) apneas in the newborn and early infancy are primarily central and more frequent in preterm than term infants; 2) breath-holding type of apneas are prevalent in term infants but negligible in preterm; 3) lower $TcPO_2$ in preterm infants during the first month of life may explain higher incidence of apneas; and 4) respiratory output and alveolar ventilation remain remarkably constant despite a dramatic change in the rate of apnea.

1439 ABNORMALLY PROLONGED CAFFEINE ELIMINATION IN BREAST FED INFANTS.

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Four exclusively breast fed infants (BFI) on caffeine (C) maintenance therapy for apnea of prematurity have been followed for a 5 month period with serial blood C dosage (pre and post-dose) allowing calculation of C $t_{\frac{1}{2}}$. BFI were compared to 14 formula fed infants (FFI). Contrary to FFI who presented a sharp increase in C elimination with post-conceptional age, C $t_{\frac{1}{2}}$ decreased very slowly in BFI. This is expressed by some of our data at different post-conceptional age in BFI and FFI with respectively at 39-42 wks: 83.7 h (99.5-71.5) vs 61.7 h (100-26.8); at 47-50 wks: 74.7 h (90.0-58.7) vs 30 h (63-11.2); at 55-58 wks: 51.2 h (58-44.4) vs 11.8 (30-1). One BFI did not show any maturation with a steep decrease of C elimination from 80 h at 40 wks to 102 h at 44 wks and 372 h at 54 wks.

Mean pre-dose was 4 times higher in the BF group at 51-54 wks: 11.5 mg (16.1-8.3) vs 2.8 ± 2.6 mg, and 6 times higher at 55-58 wks: 7 mg (7.4-6.4) vs 1.1 ± 1.1 mg. These data have serious therapeutic implications and confirm that monitoring C is mandatory, particularly in BFI who can show abnormal C accumulation.

These data show a progressive discrepancy with post-conceptional age in caffeine elimination between BFI and FFI strongly suggesting that maternal hormones in breast milk repress or inhibit the normal maturation of C elimination.

1440 *SEQUENTIAL MEASUREMENT OF URINARY MECONIUM INDEX (UMI) IN INFANTS WITH MECONIUM ASPIRATION SYNDROME (MAS).*

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Aspiration of meconium before or during birth may cause respiratory distress of varying severity. Neonates with MAS frequently pass dark urine after birth. Previous investigators have suggested that a water-soluble substance derived from meconium may be absorbed through the lungs and excreted in the urine. This substance has a spectrophotometric absorption peak at 405 nm which has been used in the calculation of the UMI. We prospectively studied 14 babies with the diagnosis of MAS. For each patient, the UMI was determined in the first postnatal urine (UMI_1) and in a second urine sample obtained at approximately 24 hours of age (UMI_2). Four patients demonstrated an increase in UMI_2. As a group, these babies had more complicated hospital courses than did the remaining ten infants, who, in fact, exhibited a marked decrease in UMI_2.

	Increased UMI2 n=4	*Decreased UMI2 n=10*	*P*
Persistent Fetal Circulation	*4*	*1*	*0.001*
Pneumothorax	*2*	*2*	*0.090*
Hypoxic Encephalopathy	*3*	*1*	*0.006*
Mech. Vent. > 5 days	*3*	*1*	*0.006*

Sequential measurement of UMI may be useful in predicting morbidity and possible outcome in neonates with MAS, and may alert the clinician to the possibility of extrapulmonary disease in babies who remain seriously ill but who demonstrate a fall in UMI.