

1416 ROLE OF BLOOD FLOW IN CEREBRAL LACTIC ACID DELIVERY (LD) AND UPTAKE (CMR). Abbot R. Luptook, A. Michael Porter, and Jan Peterson, (Spon. by C.R. Rosenfeld) Southwestern Med. Sch., Dept. of Pediatrics, Dallas, Texas.

Although glucose is the primary fuel for the brain, alternative substrates are available. Whether or not a substrate is important depends on bulk delivery to and transport into the brain. We examined LD (mM/min·100gm) and CMR, (mM/min·100gm) before, during, and after hemorrhagic hypotension in 12 ventilated piglets. Cerebral blood flow (CBF, microspheres) and arteriovenous differences of lactic acid were obtained during control (C), after 15 min of hypotension (H), and 10 (R-10) and 90 (R-90) min after rapid reperfusion with whole blood. Mean arterial pressure (mmHg) fell from 89 ± 2 ($\bar{X} \pm SE$) to $36 \pm 2^*$ during H, and rose to 89 ± 3 and 85 ± 2 at R-10 and R-90, respectively. Arterial lactic acid (La), rose from $1.93 \pm .21$ to $7.11 \pm .67^*$ mM during H, increasing further to $9.21 \pm .95^*$ at R-10; by R-90 La fell to $3.41 \pm .69^*$. CMR was similar at C, H, and R-90 ($.01 \pm .07$, $.01 \pm .01$ and $.01 \pm .01$, respectively), but rose 10-fold at R-10, $.10 \pm .03^*$. CMR mirrored LD; i.e., unchanged during C, H, and R-90 ($.17 \pm .02$, $.25 \pm .05$ and $.25 \pm .06$, respectively), but increased 10-fold at R-10 to $1.07 \pm .16^*$. Changes in LD reflect those of La and CBF (ml/min·100g), the latter falling from 98 ± 8 to $41 \pm 8^*$ during H, but increasing to $131 \pm 15^*$ at R-10, and decreasing to 83 ± 11 at R-90. Furthermore, CMR correlated with LD ($r = .68$, $p < .001$). The importance of CBF in substrate delivery and uptake in newborns is demonstrated, as well as the role of La as substrate for cerebral metabolism. (* $p < .05$ vs. control).

1417 NON-INVASIVE MONITORING OF ARTERIAL O₂ SATURATION IN NEWBORN INFANTS. Fergus Leahy, Asher Tal, Hans Pasterkamp, John Marks, University of Manitoba, Children's Hospital, Winnipeg, Manitoba.

Ear oximetry is a simple non-invasive method for measurement of arterial O₂ saturation (SaO₂) in adults and older children. To evaluate the practicability and accuracy of ear oximetry in newborn infants, we have measured SaO₂ in 12 newborns, ages 2.1 days \pm 1 (SD) using Biox II-A oximeter and its small ear probe. 15 SaO₂ measurements were done during mechanical ventilation (for HMD, diaphragmatic hernia, sepsis) and compared to actual (CO-OX) and calculated SaO₂ from simultaneously obtained arterial blood. Blood pressure was in normal range at the time of study. Bilirubin and Hb levels were noted. There was excellent correlation between SaO₂ measured by ear oximetry and CO-OX values ($n=12$; $r=0.987$) and calculated SaO₂ ($n=13$; $r=0.989$) (range: 43-99%). SaO₂ measured on the scrotum correlated equally well to CO-OX SaO₂ ($n=6$; $r=0.979$). Bilirubin and Hb levels had no effect on differences between the various SaO₂ measurement methods. Ear oximetry is an accurate and practical method for monitoring SaO₂, especially in chronic hypoxemia situations during the neonatal period. Transcutaneous PO₂ monitoring will be preferred for preterm infants at risk of hyperoxia. However ear oximetry has the advantages of easier application of the probe with less risk of skin burns, and thus longer continuous monitoring of SaO₂ in hypoxic newborn infants.

1418 NECROTIZING ENTEROCOLITIS CAN BE CAUSED BY POLYCYTHEMIA IN THE NEWBORN DOG. M.H. LeBlanc, C. D'Cruz, K. Pate (spec. by B. Batson), Dept. of Ped. and Path. Univ. of Miss. Med. Center, Jackson, Mississippi.

Although necrotizing enterocolitis (NEC) has been associated with polycythemia in human infants, they have not been shown to be causally related. To elicit a possible causal relationship the following experiment was performed. 46 unanesthetized puppies were studied (age 6-14d). Normovolemic polycythemia Hct 70, was induced in 19 pups by exchange transfusion with 75cc/kg of red blood cells (RBC). Hypervolemic polycythemia, Hct 70, was induced in 14 pups by transfusion with 50cc/kg of RBC. 13 pups received exchange transfusion with whole blood and served as controls, Hct 40. Gross autopsy was performed on all pups at 24 hrs post-transfusion or at death. NEC was defined as areas of violaceous discoloration of the bowel associated with blood in the intestinal lumen. Although lesions appeared throughout the bowel in some pups, involvement of the distal small bowel was most common. Diagnosis was confirmed by microscopic examination. Microscopic changes ranged from mucosal hemorrhage and necrosis to transmural necrosis, hemorrhage and inflammation. Submucosal pneumatosis intestinalis was seen in two cases and air was discerned in ileocecal lymph nodes in one case. Both gross and microscopic lesions appear similar to that in NEC in human infants. NEC was seen in 11/19 pups with normovolemic polycythemia, 7/14 pups with hypervolemic polycythemia, and only 1/13 controls ($p < .01$). Necrotizing enterocolitis can be caused by polycythemia in the newborn dog.

1419 THE EFFECT OF POLYCYTHEMIA ON PLASMA VOLUME IN NEWBORN DOGS. M.H. LeBlanc, K. Pate, Dept. Peds. (spec. B. Batson), Dept. Peds. Univ. MS Med. Ctr., Jackson.

The effect of polycythemia (Hct 64-80) on plasma volume (PV) was studied in 27 unanesthetized, splenectomized puppies (age: 6-14, post-splenectomy: 5-13d). Normovolemic polycythemia (N) was induced in 9 pups by exchange transfusion (ExN) with 75cc/kg of red blood cells (RBC). Hypervolemic polycythemia (H) was induced in 11 pups by transfusion of 50cc/kg of RBC. 7 pups received ExT with 75cc/kg of whole blood and served as controls (C). PV (^{125}I Fibrinogen) and red cell volume (RCV, by ^{51}Cr) were measured prior to and 1, 2, and 4 hrs after transfusion, with the pups receiving no oral intake. Pups were fed 8cc/kg/hr after 4 hrs, and measurements were repeated at 8 and 24 hrs. RCV rose with transfusion in N and H and remained elevated throughout the experiment. RCV did not change in C. PV fell slightly in the C group prior to 4 hrs and then rose to initial levels. PV rose in the N pups from $-50 \pm 7\%$ SD to $-35 \pm 10\%$ ($p < .001$), $-37 \pm 8\%$, $-38 \pm 9\%$, and after feeding to -29 ± 13 and 1 ± 18 ($p < .001$), 1, 2, 4, 8 and 24 hrs post-transfusion. PV fell in the H pups from $4 \pm 2\%$ to -24 ± 12 ($p < .001$), -35 ± 14 , -37 ± 14 , and then rose after feeding to -32 ± 9 and 0 ± 15 ($p < .001$). At 1, 2, 4 and 8 hrs PV in H and N was different from that in C ($p < .01$). Thus pups with induced N or H equilibrate rapidly to a PV determined primarily by the Hct and not initial PV. Since the increase in PV in N prior to feeding was rapid and occurred during a period of no fluid ingestion, it must be due to a decrease in capillary pressure secondary to the induced change in viscosity. A further increase in PV to control levels occurs when the pups are fed, which is probably caused by renal solute retention.

1420 INCIDENCE OF FEEDING INTOLERANCE IN VERY-LOW BIRTH WEIGHT INFANTS. Kwang-sun Lee, Rita Klein, Chuwen-yuh Kuo, William Zala, Hui-shin Wu, Abdul Aldousany and Stuart Berger, University of Chicago, Dept of Peds, Chicago, IL.

The relationship between initial modes of nutritional support and later feeding intolerance was examined. Infants with birth-weights (BW), $< 1,500$ gms, were randomly assigned to Gr. I, continuous nasogastric feeding (CNG) and Gr. II, parenteral nutrition (PN). Gr. I continued to have CNG regardless of their clinical conditions except feeding intolerance. Gr. II was supported by PN [3.6+0.9 (M±SEM) days] until clinical condition became stable and CNG started. Frequency of feeding intolerance in the first 30 days of life is shown below:

Group	n	Heme(+) stools	Pneumatosis	NEC	CNG failure
Gr. I CNG	24	54%	8%	25%	46%
Gr. II PN	32	66%	6%	24%	39%

NEC: Acute necrotizing enterocolitis. Differences, all, $p < .05$. Feeding intolerance in these infants, then, was examined by BW and duration of mechanical ventilation (MV) as shown below:

Group	n	Heme(+) stools	Pneumatosis	NEC	CNG failure
Gr. A	10	90%	20%	80%	100%
Gr. B1	25	44%	4%	4%*	16%*
Gr. B2	22	64%	5%	23%*	45%*

Gr. A: < 900 gms. Gr. B: 900-1,500 gms. Gr. B1 with MV, 0-7 days and Gr. B2 with MV, 7 days. Differences, both $p < .05$.

Results of this study suggests that 1) a short duration of initial PN does not prevent a later development of feeding intolerance and 2) feeding complications are significantly higher in infants with a prolonged mechanical ventilation.

1421 HOME CARE (HC) OF INFANTS WITH BRONCHOPULMONARY DYSPLASIA (BPD) AND OXYGEN DEPENDENCY (OD). Marta H. Lifschitz, Dan K. Seilheimer, Susan D. Thurber, Shirley Northrop, Geraldine S. Wilson, Mardina M. Desmond. Baylor College of Medicine, Texas Children's Hospital, Department of Pediatrics, Houston.

Newborns who develop BPD and OD have costly hospitalizations and prolonged separation from their families. HC was provided to 26 ventilator independent infants who had BPD and required supplemental O₂ to maintain a TcPO₂ $> 50-60$ mmHg. Practicability of HC was assessed in 13/26 at age > 6 months ($\bar{x} = 20.3$ mo). Mean GA was 28 wk. Birth weight 1307 g, days on ventilation 79, hospital days 179. Morbidity included 11 patients that had HMD, 8 IVH, 4 PDA, 3 persistent fetal circulation, 11 ROP, and 4 GE reflux. All parents were high school educated and had health insurance. Parents' training prior to HC included feeding techniques, handling of O₂ concentrator, administration of medication, and if indicated, use of monitor and CPR. Only few families required HC nursing. At present, 11/13 are off O₂ (mean age cessation, 9 mos), 10/13 are fed entirely P.O. and 3 (ages 15-41 mos) are still fed by N.G. tube or gastrostomy. Six/13 had 13 readmissions, 38% related to pulmonary and 62 to GI/feeding problems. Nine remain $< 5\%$ for corrected age in weight, 8 for length, and 4 FOC. Despite the physical demands of providing constant care at home, parents report that with HC life was better organized, attachment improved, and expenses reduced up to 96%. Few had initial difficulties with O₂ use. Feeding related problems are the major concern. **Conclusion:** Home care of oxygen dependent infants is a viable option to chronic hospitalization.