235 THE EFFECT OF ARGININE VASOPRESSIN (AVP) ON RENAL VASCULAR RESISTANCE (RVR) IN NEWBORN LAMBS DURING HYPOXEMIA (HPX). Nancy A. Ayres, Sindy Wear, and Jean E. Robillard. University of Iowa, Department of Pediatrics, Iowa City, IA.

HPX in newborn lambs results in a significant increase in AVP (Circ Res 52:179, 1983). Thus, to evaluate the role of AVP on RVR during HPX, doppler flow probes were placed on the renal arteries of 10 chronically catheterized newborn lambs (8-13 days) to allow continous monitoring of renal blood flow (RBF). Each lamb was used as its own control and randomly received either 100 ugm bolus of the specific AVP inhibitor (AVP-I), d(CH_2)5Tyr(Me)AVP, on the experimental day (EXP) or the vehicle used for AVP-I infusion on the control day (CON). Vascular responsiveness and effectiveness of AVP-I were evaluated by giving 100 mU bolus of AVP before and following HPX on each CON and EXP day. On both CON and EXP days HPX produced significant decreases in PO_2 from 88±4 to 36±2 mmHg and 84±7 and 32±3 mmHg respectively. Before (Pre-HPX) and following HPX (Post-HPX) AVP infusion increased RVR in animals receiving the vehicle alone but not in animals pre-treated with AVP-I. However, during HPX no significant changes were found in RVR, mean arterial blood pressure (MABP), and RBF between the CON and EXP days.

% Change in RVR, MABP and RBF at 5 minutes after AVP bolus or HPX

	Pre-HPX+AVP		HYPOXEMIA		Post-HPX+AVP	
	CON	EXP	CON	EXP	CON	EXP
RVR	40.7±9.7	4.2±5.7*	9.2±5.7	4.5±4.8	14.4±6.2	-5.5±4.8*
MABP	16.3±2.3	2.3±1.0*	-1.4±5.4	3.5±5.0	12.2±3.4	-8.0±5.4*
RBF	-14.6±6.4	-1.9±6.5	-9.3±10.0	-6.9±6.9	3.3±7.3	-0.8±4.8

* $p<0.05$; $\bar{x}$ ± SEM.

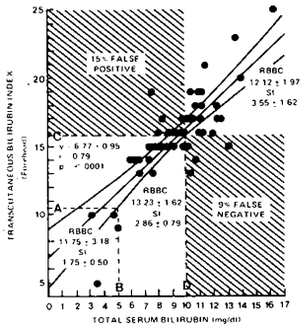
These data suggest that AVP does not play a significant role in modulating the RVR, renal flow, or MABP during HPX in newborn lambs.

238 USE OF A PORTABLE SUCKOMETER TO INVESTIGATE NUTRITIVE SUCKING PATTERNS IN INFANTS. Edward L. Bartlett, Jr. Sharon L. Murray, Robert J. Fink (Spon. by G.C. Rosenquist), Children's Hosp. Natl. Med. Ctr. and George Washington U., Washington, D.C.

An easy method for assessing nutritive sucking behavior during infancy would provide early identification of children at risk for feeding problems. We developed a portable suckometer to measure sucking amplitude across time and burst/interval patterns, and studied 54 normal infants (41 term, GA 38-42 wks., at 3.7 days ± 1.6 SD and 13 preterm, GA 34.5 wks. ± 1.8 SD at 41.5 wks. ± 1.6 SD post conception) in a newborn nursery for a mid-morning feed. Measurements were taken for the first 60 cc consumed or 20 minute feeding time calculated from the first (S), mid (M) and last two minutes (E) of the feed. Breast-fed infants were given D5W; bottle-fed infants received their usual formula (e.g. Isomil). Nutrient flow was determined by the infant's suck. Standard cross-cut nipples with a second precut hole (Ross Twist-on Nipple Unit) were attached to disposable graduated feeding tubes interfacing with an Ailtech miniature pressure transducer via Intramedic polyethylene tubing (PE-160). Results include: a) no significant differences on suck measurements for breast vs. bottle-fed, D5W vs. formula, male vs. female, preterm vs. term; b) a decrease across time (S→E) for number of sucks ($p<.001$), and number and length of bursts ($p<.001$). This normative information obtained from the portable suckometer provides a baseline for comparison with infants with feeding difficulty, and documents the ease of obtaining nutritive sucking measurements in a normal newborn nursery.

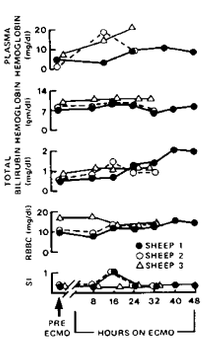
236 FLUOROMETRIC BILIRUBIN BINDING ASSAY (FBBA) AND TRANSCUTANEOUS BILIRUBINOMETRY (TcB) IN TERM NEWBORNS. Raul C. Banagale (Spon. by William F. Howatt), Univ. of Michigan, Department of Pediatrics, Ann Arbor, MI.

Fifty-four white full term newborns (gestational age 40.0±1.2 wks; birth wt 3447±440 gms), not under phototherapy, were studied. FBBA including reserve bilirubin binding capacity (RBBC) and saturation index (SI) were determined, as well as the bilirubin levels by TcB and modified diazo method. There is a significant linear relationship between serum bilirubin levels and values obtained by TcB (figure). This study differs from other published works on TcB by demonstrating the RBBC and SI levels of infants who belong to Area AB (RBBC=11.75±3.18, SI=1.75±0.5) and Area CD (RBBC=13.23±1.6, SI=2.86±0.79). These FBBA values including the RBBC (12.12±1.97) and SI (3.55±1.62) of the infants outside these areas are normal, demonstrating that this group of infants are not in danger of Kernicterus regardless of the TcB and serum bilirubin values at 3 days of life.



237 BILIRUBIN ALBUMIN BINDING PARAMETERS IN SHEEP DURING EXTRACORPOREAL MEMBRANE OXYGENATION (ECMO). Raul C. Banagale, John Toomasian, Cindy Nixon, Robert H. Bartlett (Spon. by William F. Howatt). Univ. of Michigan, Depts. of Surg. and Peds., Ann Arbor, MI.

Three sheep (average wt. 32 kg) were anesthetized, ventilated and cannulated for venoarterial (VA-ECMO) by-pass procedure (Trans Am Soc Artif Intern Organ 28:350, 1982). Bilirubin albumin binding parameters were determined using a Bilirubin Hematoflurometer (Pediatr Res 17:127A, 1983). Pre-VA ECMO values were: hemoglobin (Hgb) 8-10 gm/dl; plasma Hgb 1-7mg/dl; Fibrinogen (F) 110-245 mg/dl; Fibrin split products (FSP) 10 µg/ml and platelet count (Plt) 495-880x10^3/mm^3. During VA-ECMO, F was 96-225 mg/dl, FSP over 40 µg/ml and the Plt was 94-269x10^3/mm^3. As shown (Figure), the Hgb and bilirubin levels varied, with elevation of plasma Hgb. In spite of hemoglobinemia suggesting intravascular hemolysis, the reserve bilirubin binding capacity (RBBC) and saturation index (SI) values were normal during the study. The normal RBBC and SI values would make the development of bilirubin neurotoxicity unlikely during VA-ECMO.



● 239 **HYPOXIA FAILS TO CAUSE HYPERTROPHY OR HYPERPLASIA OF FETAL PULMONARY ARTERY SMOOTH MUSCLE CELLS IN VITRO** William E. Benitz, Daniel S. Lessler and Merton Bernfield Stanford University School of Medicine, Department of Pediatrics, Stanford, CA

Persistent pulmonary hypertension of the newborn (PPHN) is associated with both prenatal hypoxia and increased pulmonary arterial (PA) smooth muscle mass in both clinical (Gersony, J Pediatr 82:1103, 1973; Murphy et al, J Pediatr 98:962, 1981) and experimental (Goldberg et al, Pediatrics 48:528, 1971; Gersony et al, J Pediatr 89:631, 1976) circumstances. To assess the ability of hypoxia to directly produce increased PA smooth muscle cell (SMC) mass, we examined the effect of hypoxia on the proliferation and size of SMC cultured from fetal bovine pulmonary arteries. SMC were cultured under ambient oxygen tensions (pO_2) ranging from 2 to 145 torr. Both the rate of cell proliferation and the final cell density (10 days of culture) were decreased in cultures incubated at pO_2 less than 55 torr. Final cell densities at pO_2 of 2 or 24 torr were only 18% and 80%, respectively, of that seen at pO_2 of 85 to 145 torr ($p<0.002$). Oxygen tension did not affect SMC size, as the protein:DNA ratio was constant. Therefore, hypoxia does not produce either proliferation or hypertrophy of PA SMC in vitro. We conclude that prenatal hypoxia does not act directly on SMC in the PA to produce the increased PA SMC mass observed in infants with PPHN. Other mediators of this developmental abnormality must be sought. (Supported by NIH Grant HD06763 and a Medical Student Traineeship of the Cystic Fibrosis Foundation.)

240 LIPID CLEARING IN PREMATURE INFANTS. RESPONSE TO INCREASING DOSES OF INTRALIPID(IL): II-FREE FATTY ACIDS(FFA), TRIGLYCERIDES(TG) AND CHOLESTEROL(CHOL). S. Berkow, M.L. Spear, A. Gutman, G.E. Stahl, M. Hamosh, R.A. Polin, G.R. Pereira, P. Hamosh. Depts. Pediatrics, Georgetown Univ. Med. Center, Washington, DC and Children's Hospital of Philadelphia, Phila., PA.

Plasma levels of FFA, TG and CHOL were measured in 21 premature infants (gest. age 26-35 wks.), aged 1-8 days, maintained on total parenteral nutrition with heparin (1 U/ml) and given 1, 2 and 3 g IL/kg/day (over 15 h) on day 1, 2 and 3 respectively. Blood samples were drawn before and at the end of IL administration.

	FREE FATTY ACIDS		TRIGLYCERIDES		CHOLESTEROL	
Day	Before	After	Before	After	Before	After
1	0.19 ± 0.02	0.56 ± 0.11*	52 ± 8	131 ± 20**	86 ± 6	112 ± 9**
2	0.22 ± 0.03	0.90 ± 0.15**	45 ± 7	170 ± 30**	115 ± 11	131 ± 11+
3	0.31 ± 0.04	1.12 ± 0.16**	49 ± 7	214 ± 49*	153 ± 15	171 ± 18

FFA - mEq/l; TG & CHOL - mg/dl. All values mean ± SEM.
+ = $p<.05$; * = $p<.01$; ** = $p<.001$ - Before vs. After.

In all infants there was a transient, dose dependent increase in TG and a cumulative increase in CHOL. A significant positive correlation between FFA and TG levels ($r = 0.65$, $p<.001$) at 15 hr of TG infusion suggests that plasma TG at that time is a mixture of IL and VLDL newly synthesized from the released free fatty acids. A significant positive correlation between FFA and CHOL levels ($r = 0.58$, $p<.001$) at 15 hr of TG infusion suggests that the CHOL is also newly synthesized from FFA-derived acetyl-CoA. During the following 9 hours, the removal of the excess TG was adequate, but the cumulative increase in CHOL necessitates further study. (Supported by NIH grants HD-15631 and RR-00240).