● 253 ANATOMIC DISTRIBUTION AND QUANTITATIVE DEVELOPMENTAL CHANGES IN GUINEA PIG PULMONARY BETA RECEPTORS. Catherine Gatto, Virginia Seybold, Thomas Kulik, James Lock, and Dana Johnson. University of Minnesota Medical School, Departments of Pediatrics and Anatomy. Minneapolis, MN.

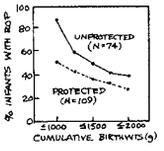
Quantitation using tissue homogenates has demonstrated an increase in pulmonary ß-receptors during development. However, techniques using disrupted tissue have not permitted the precise anatomic localization of pulmonary ß-receptors or identification of the structures where increases occur. Using ^{3}H-dihydroalprenolol, ß-receptors were radioautographically localized and quantitated in sections of newborn (NB) and adult (A) guinea pig lung. Specific binding on lung sections, defined as that prevented by 2µM propranolol (80-90% of total counts), was rapid and saturable. Scatchard analysis showed a single class of binding sites with a maximum binding capacity of 189+3 (NB) and 305+37 (A) fmole/mg protein ($p<.003$). Binding was of high affinity K_d = 1.46+.2 (NB) 1.26+.3 (A) nM (N.S.). The majority of ß-receptors were localized in the alveolar wall and airway epithelium (alveolar >> bronchiolar > bronchial) ($p<.0001$) and averaged 2.8 (A) and 1.3 (NB)-fold greater in number than ß-receptors in corresponding airway smooth muscle ($p<.005$). Arterial and venous smooth muscle had few demonstrable ß-receptors. The increased number of ß-receptors in the adult appeared to be due primarily to a 2.0+.2 fold increase in alveolar wall and airway epithelium as opposed to only a 1.3+.4 fold increase in the already low number in airway and vascular smooth muscle ($p<.003$). While receptor density may not necessarily correlate with response or physiologic importance, the role of ß-receptors in airway and alveolar epithelium/endothelium function deserves further investigation.

254 LACTATE UPTAKE BY THE FETAL LIVER. Christine A. Gleason, Abraham M. Rudolph, James D. Bristow, and Colin D. Rudolph, Department of Pediatrics, University of California, San Francisco, CA 94143

Lactate is produced by the placenta and is an important metabolic substrate for fetal lambs. However, utilization of lactate by the fetal liver specifically has not been assessed. We measured net hepatic lactate uptake in 26 normoxemic fetal lambs at 128 (121-138) days gestation, with catheters chronically maintained in descending aorta, inferior vena cava, right or left hepatic veins and umbilical vein (UV). Lactate and hemoglobin concentrations and O_2 saturations were measured in all vessels 3-7 days after surgery, and hepatic and umbilical blood flows measured by injecting radioactive microspheres into UV. VO_2 of each liver lobe was 1.60 ± 1.65 ml/min. Net lactate provided to the fetus from the placenta was 4.55 ± 5.04 mg/min, and total lactate delivery to the liver was 44.4 ± 21.2 mg/min. Net lactate uptake by each liver lobe was 2.06 ± 2.22 mg/min, so that total hepatic lactate uptake was similar to that provided by the placenta. However, hepatic extraction was only about 10% of the lactate delivered. Lactate is thus an important substrate for the fetal liver; calculation of lactate: oxygen quotients indicated that lactate was used metabolically for processes other than oxidation as a substrate.

● 255 EFFECT OF NURSERY ILLUMINATION ON THE INCIDENCE OF RETINOPATHY OF PREMATURITY (ROP) Penny Glass, George Washington University, Dept. of Psychology; Gordon Avery, Children's Hospital National Medical Center, Dept. of Neonatology; K.N. SivaSubramanian and Marshall Keys, Georgetown University Hospital, Depts. of Neonatology and Ophthalmology, Washington, D.C.

The preterm infant is subjected to prolonged exposure to ambient nursery illumination at levels that have been found to produce retinal damage in animals. The effect of light exposure in the nursery on the incidence of ROP was investigated in two Level III nurseries. One hundred eighty-three infants with birthweights ≤2000g. and GA≤34 weeks were admitted to the study. (Infants >1500g had to have received O_2). One group of infants from each hospital was protected from the ambient lighting by the addition of a neutral density filter to the incubator. The Control infants were from the regular nursery environments. The infants were routinely examined by ophthalmologists for ROP at discharge or shortly thereafter. The results indicate less incidence of ROP in the Protected Group, particularly for those with BW ≤ 1000g, the infants most susceptible to ROP (χ^2=19.79, $p<.001$). The trend is similar within each hospital. These findings are further substantiated by an epidemiological study of infants in the Protected Group who had been in the same nursery. Fourteen ROP infants were matched for BW with 10 Non-ROP infants. The chance of being in a bed next to the window, and therefore "sun-exposed", is 25%. These ROP infants were more likely to have been "sun exposed" than the Non-ROP infants (79% vs.10% respectively, $p<.01$). These associations between light level and incidence of ROP suggest that the ambient illumination in the hospital nursery may be one factor contributing to ROP. This study has important implications for the early care of infants at risk.



256 NEGLIGIBLE GLUCONEOGENESIS BY THE FETAL LAMB LIVER. Christine A. Gleason and Abraham M. Rudolph, Dept. of Pediatrics, Univ. of California, San Francisco, CA.

Previous studies of gluconeogenic capability of the fetus in vivo have yielded conflicting results, and the specific role of the fetal liver in gluconeogenesis has not been defined. We studied 7 fetal lambs with mean gestational age 130.5 days (range 122-140d). We placed catheters in maternal artery and vein and in fetal hindlimb artery (FA) + vein (IVC), right or left hepatic vein (RHV, LHV), and umbilical vein (UV). After 3-5 days recovery, ^{14}C-alanine (25 µCi bolus, 20 µCi/hr continuous infusion) or ^{14}C-lactate (40 µCi bolus, 30 µCi/hr continuous infusion) was infused into fetal IVC. Lactate and glucose concentrations, glucose specific activity, and substrate radioactivity were measured after 30 and 60 minutes. Radioactive microspheres were injected into UV to measure liver blood flow. ^{14}C-Glucose uptake and release by the liver was calculated; net labelled glucose was calculated as a percentage of ^{14}C-substrate perfusing the liver. Mean glucose uptake from the placenta was 6.00 mg/kg/min (range 2.13-16.0). Glucose uptake or release by each liver lobe was only 0.70 mg/min; (range -3.7 to 9.5). Only 0.35% (range 0-2.3%) of ^{14}C-substrate was converted to ^{14}C-glucose; ^{14}C-lactate (0.29%) and ^{14}C-alanine (0.38%) were not significantly different. There was a suggestion that the left lobe converted less substrate to glucose (0.03%) than did the right (0.48%).

We conclude that hepatic gluconeogenesis contributes negligibly to fetal glucose supply. Warnes et al. showed gluconeogenesis is present within minutes after birth. Our model permits the evaluation, in utero, of mechanisms that stimulate gluconeogenesis.

● 257 EFFECT OF HYPOXIA ON BRAINSTEM BIOGENIC AMINES. Jeannine L. Gingras-Leatherman, M. Colleen McNamara, and Edward E. Lawson. Dept. of Pediatrics, Univ. of North Carolina, Chapel Hill, NC

Prolonged severe hypoxia results in reduced concentration of brainstem biogenic amines (J. Neurochem. 21:783, 1973). To test whether this effect is found in shorter, less severe hypoxia we subjected 3 d/o and 21 d/o rabbits to 10% O_2 in thermal jacketed chambers. The rabbits were confined for 6h breathing 21% O_2 (RA) (grp I), 10m 10% O_2 every .5h (grp II) for 6h, 2h 10% O_2 followed by 4h RA (grp III), 4h RA followed by 2h 10% O_2 (grp IV). Using sensitive radioenzymatic assays, we determined the level of dopamine (DA), norepinephrine (NE) and serotonin (5HT) in specific brainstem nuclear groups. The nuclei (dorsal raphe, DR; locus coeruleus, LC; substantia nigra SN) were removed by micropunch technique. In general, DA was not affected by hypoxia and there were inconsistent changes in NE. The data for 5HT is presented below (%Δ from control):

	3 d/o: DR	LC	SN	21 d/o: DR	LC	SN	
grp II	-75*	-69*	48	68	-13*	73	n=6 both
grp III	-69	-68*	-20	62	50	460*	groups;
grp IV	-94*	-17*	-87*	99*	168*	333	* p<.05

The 5HT findings demonstrate that acute hypoxia decreases specific central neurotransmitters in young animals. The Grp III findings imply failure to recover from these effects. 5HT influences central control of cardiopulmonary function; suggesting a mechanism to explain the increased susceptibility of newborns to the effects of hypoxia.

258 HEMODYNAMICS OF ENDOTOXIN SHOCK IN NEWBORN PUPPIES. Andrew J. Griffin, Masakatsu Goto, Zenshiro Onouchi, Pipit Chiemmongkoltip. (Spon. by Dharmapuri Vidyasagar) Loyola University Medical Center, Dept. of Pediatrics, Maywood, IL

Reports on neonatal hemodynamics in Endotoxin shock(ETX) are sparse. This study reports 58 newborn mongrel puppies, 260-800 grams in whom cardiac output(CO), mean arterial pressure(MAP), systemic vascular resistance(SVR), and minute work(MW), were measured q10 min. in controls (N=30) and 2 experimental groups receiving Ecoli(ETX) at a dosage of 1.5 mg/kg(group A=15), and 10 mg/kg(group B=13). RESULTS: MAP: fell below controls at 50 min. in group B, and at 70 min. in group A with parallel fall thereafter. CO: decreased immediately to 60% of control in both groups, and continued to decrease throughout the experiment, with gradual convergence of both groups at 120 min. SVR: rose to 200% in both groups A and B, and remained elevated throughout. MW: fell in both groups to 55% of control. SUMMARY: Newborn puppies respond to ETX with ↓CO, ↓MW, ↑SVR, and late ↓MAP, in contrast to adults subjects. Quantifications of interventions are now possible with this model.

	MAP		CO(L/min/kg)		SVR (unit)		MW (unit)	
Group	A	B	A	B	A	B	A	B
0	53±2	59±2	.37±.03	.33±.03	.15±.02	.20±.02	249±20	270±20
5 Min	54±3	63±3	.24±.03	.18±.02	.29±.04	.43±.06	180±22	141±27
1 Hr	53±2	43±4	.19±.02	.14±.02	.26±.02	.25±.06	150±18	150±18
2 Hrs	27±2	14±2	.14±.01	.12±.02	.21±.02	.15±.03	54± 9	25± 5