1259 AMNIOTIC FLUID ANALYSES IN BABOON PREGNANCIES. Y.W. Brans, T.J. Kuehl, D.L. Shannon and P. Reyes. University of Texas Health Science Center, Department of Pediatrics, and Southwest Foundation for Research and Education, San Antonio, Texas.

Extreme similarities in the anatomy and physiology of the fetus-placenta-uterine unit in baboons and humans make the baboon an ideal model to investigate various aspects of reproductive physiology. Thirty-six amniotic fluid samples from 30 normal baboon pregnancies ranging in length from 50 to 173 days (term=180 days) were analyzed to obtain baseline data.

Amniotic fluid volume as determined by para-aminohippurate dilution ranged from 126 to 380 ml, with a mean ($\pm$ SD) of 269 ± 77.6 ml. Volume did not vary significantly over the period of the study. It correlated well with the volume determined at cesarean section by aspirating all the fluid prior to incising the uterus ($r=0.65$). Osmolality averaged 273 ± 8.3 mOsm/kg (range: 252-287). Creatinine (C) concentration ranged from 0.23 to 1.51 mg/dl and increased linearly with gestational length (G): $C=0.008 G-0.09$, $r=0.63$, $S_{yx}=0.248$. Protein (P) concentration ranged from 0.022 to 1.229 g/dl and increased as a power function of gestational length: $P=0.022 G^{0.7}$, $r=0.88$, $S_{yx}=0.3156$. PH averaged 7.70 ± 0.226 (range: 7.44-8.30). A spectrophotometric absorption peak was identified at 400 nm rather than at 450 nm: ΔOD_{400} averaged 0.047 ± 0.0686 (range: 0-0.290) and did not vary with gestational length.

While these values are of the same order of magnitude as those observed in human amniotic fluid, the data suggest that baboon amniotic fluid differs from humans' in a number of respects.

1260 NEONATAL EFFECTS OF SEVERE MATERNAL HYPERTENSION BEFORE 36 WEEKS GESTATION. Jane E. Brazy, Virginia Little, Judy Grimm (Spon. by G.W. Brumley) Duke University Medical Center, Dept. of Pediatrics, Durham, NC.

Neonatal problems associated with an abnormal fetal environment have been well documented for infants of diabetic mothers, but the effects of severe hypertension upon the infant have not been well defined. Twenty-eight infants 27-35 weeks of gestation, delivered of mothers with diastolic hypertension ≥ 110 mmHg (IHM) and 28 gestational age-matched control infants (CI) delivered of normotensive mothers without tocolytic therapy were compared to determine the effects of severe hypertension and its therapy on the preterm neonate. Eight hypertensive mothers (HM) were eclamptic; all HM received magnesium sulfate; 32% sedatives; and 79% antihypertensive medications. Thrombocytopenia and marked LDH elevation occurred in 50% of hypertensive mothers. 39% of IHM and 0% CI were small for gestational age ($p < .001$); 29% and 0% were microcephalic at birth ($p < .01$). Other significant differences between the two groups of infants were noted in the incidence of hypotonia, ileus, delayed stooling, delayed respiratory adaptation, thrombocytopenia, leukopenia, neutropenia, patent ductus arteriosus and birth asphyxia. Maternal thrombocytopenia and enzyme elevations were significantly associated with growth retardation, microcephaly, thrombocytopenia, neutropenia and birth asphyxia in the infant. Gastrointestinal hypomotility and hypotonia were probably drug related. Severe maternal hypertension has unique and significant effects upon the preterm neonate which reflect the severity of the maternal disease process.

1261 TRANSCUTANEOUS BILIRUBINOMETRY IN INFANTS; INFLUENCE OF RACE AND PHOTOTHERAPY. Audrey K. Brown, Mae Hee Kim, Praewpun Nuchpuckdee, Gerard Boyle. SUNY-Downstate Medical Center, Department of Pediatrics, Brooklyn, N.Y.

The transcutaneous bilirubin meter, TcBM, a non-invasive spectral device developed by Yamanouchi (with Minolta Co.) gives an objective index of cutaneous bilirubin. We have compared TcBM indices with total serum bilirubin (A.O. Bilirubinometer) in 29 white and 142 black infants and evaluated the influence of race and phototherapy. Correlation between TcBM and total bilirubin was strong in both black ($r=0.79$) and white ($r=0.87$) infants. TcBM indices in black infants were higher than in white infants at serum bilirubin levels less than 5mg/dl; this difference disappeared at bilirubins greater than 10mg/dl. At TcBM indices less than 13, serum bilirubins in all infants were less than 5mg/dl. TcBM indices greater than 20 were associated with serum bilirubin levels greater than 7mg/dl. In white infants receiving phototherapy, the correlation between TcBM indices and total serum bilirubin decreased but remained good ($r=0.79$); in black infants there was no longer a strong correlation ($r=0.36$). This discrepancy appears to be due to the tanning effect of phototherapy. In sum, in the absence of phototherapy this device is useful in screening both black and white infants for significant degrees of bilirubinemia. TcBM indices below 13 may preclude the necessity for an invasive bilirubin determination; indices above 20 suggest a need for such a procedure. This simple device avoids the pitfall of subjectivity inherent in any visual appraisal of icterus (including the Gosset Icterometer).

1262 PULMONARY OXYGEN TOXICITY, LIPID PEROXIDATION, AND VITAMIN E IN THE NEONATAL RAT. John R. Bucher, Ian Rendak, and Robert J. Roberts. Departments of Pediatrics and Pharmacology, University of Iowa, Iowa City, IA 52242.

We have demonstrated inhibition of lung growth and development as well as microscopic injury to result from exposure of newborn rats to 6 days of normobaric hyperoxia as low as FiO_2 0.4 (Peds. Res. in press). The role of lipid peroxidation in the origins of these effects was investigated by measuring lung vitamin E concentrations and expired pentane during hyperoxic exposure of newborn rats. Lung vitamin E increased from $3.9 \mu g/g$ at birth to $25.3 \mu g/g$ by day 3 of age in control animals. Denying pups access to dams prevented this increase. Hyperoxia ($FiO_2 > 0.95$, for 3 days) did not lower lung vitamin E concentrations in fed animals ($30.6 \mu g/g$). Expired pentane averaged slightly more in fed animals analyzed every 24 hours during a 3 day exposure to $FiO_2 > 0.95$ than in air exposed controls (2.8 pmoles/min/100 g vs. 2.5 , or 0.7 vs. 0.5 pmoles/ml O_2 consumed). Exposure of starved newborns to 20 hours of hyperoxia resulted in a dose-related lethality (FiO_2 0.21, 0/14; 0.4, 6/14; > 0.95 , 11/15) but lung vitamin E concentrations did not decrease (FiO_2 0.21, 3.1; 0.4, 3.9; > 0.95 , 3.4 $\mu g/g$). Pentane production in starved newborns fell during a 12 hour exposure to $FiO_2 > 0.95$ (2.4 to 1.5 pmoles/min/100 g). The failure of hyperoxia to reduce lung vitamin E levels coupled with the lack of substantial increases in expired pentane suggests that lipid peroxidation may not play a major role in pulmonary oxygen toxicity in the neonate. However, until the significance of small changes in expired pentane is determined, lipid peroxidation cannot be totally excluded from the mechanism(s) of oxygen-induced lung injury. Supported by NIGMS 12675 and GM 07069.

1263 THE INFLUENCE OF VITAMIN E ON OXYGEN-INDUCED LUNG INJURY AND DYSFUNCTION IN THE NEONATAL RAT. John R. Bucher and Robert J. Roberts. Department of Pediatrics and Pharmacology, University of Iowa, Iowa City, IA 52242.

Newborn rats were treated with Vitamin E (injectable, 100 mg/kg s.c., Hoffmann-LaRoche, Nutley, NJ) on day 0, immediately prior to placement in $FiO_2 > 0.95$, and again on days 2 and 4 of age during 6 day hyperoxic exposures. This treatment regimen resulted in lung vitamin E concentrations of 207%, 160%, and 160% of control on days 1, 3, and 6 respectively. Vitamin E treatment had no effect on the observed oxygen-induced retardation of lung growth (wet lung weight, 79% of control; whole lung DNA, 67% of control, and maximum lung volume at 30 cm of H_2O pressure, 76% of control), lung development (secondary septal development, 13% of control and capillary numbers per unit area 58% of control), or on the evolution of microscopic lung injury (presence of capillary engorgement and interstitial edema). Vitamin E treatment did prevent the dose related lowering of the maximal arterial PO_2 observed in the neonatal rats breathing a mixture of 99% O_2 , 1% halothane, following a 6 day exposure to FiO_2 0.21, 0.4 or > 0.95 .

Exposure Group (6-day FiO_2)	Maximal PaO_2		*Significantly different from control (FiO_2 0.21), and vitamin E ($FiO_2 >$ 0.95).
	Control	Vitamin E	
0.21	296 ± 7	-	
0.4	284 ± 13	312 ± 12	
> 0.95	$225 \pm 12^*$	273 ± 15	

Vitamin E treatment also prevented alterations in descending P/V curves seen following 6 day exposures of newborns to FiO_2 0.4 and > 0.95 . Vitamin E treatment appears to alter only a select few of the many pulmonary injury parameters associated with 6 day hyperoxic exposure. The significance and mechanism for these alterations remain to be determined. Supported by NIGMS 12675 and GM 07069.

1264 AFTERLOAD REDUCTION BY SODIUM NITROPRUSSATE (SNP) IN ASPHYXIATED INFANTS WITH HYPERTENSION AND HEART FAILURE. Luis A. Cabal, Ignacio Barrenechea, Elvezia Fumagalli, Bijan Siassi, Joan E. Hodgman. Univ. of So. Calif. Sch. of Med. LAC/USC Med. Center, Dept. of Peds., Los Angeles.

In adults, blood pressure is decreased by SNP due to arteriolar and venous dilating effect; however, its actions have not been established in newborn infants. To elucidate the latter, we examined the changes in heart rate, arterial and central blood pressures, echo and electrocardiograms, and pH. Tissue perfusion was evaluated using skin surface unheated PCO_2 electrodes. 5 infants developed left heart failure and refractory hypertension after severe fetal distress and neonatal asphyxia and were treated with digitalis and intravenous SNP ($2-6 \mu g/kg/hr.$). Before therapy all infants had: tachycardia, cardiomegaly, pulmonary edema, high central venous and arterial blood pressures. Increased left ventricular internal diameter and LA/Ao ratio, ECG ST depression, elevated CPK-MB2 isoenzymes, acidosis and elevated skin tissue PCO_2 with increased skin-arterial PO_2 gradients. Hemodynamic changes observed within 20 minutes of therapy were compared to values before therapy by paired t tests. With therapy there were no increase in heart rate, and a significant decrease in arterial and central venous blood pressures. Increases in local perfusion, tissue PO_2 , and arterial pH, with simultaneous decrease in skin PCO_2 were observed. These data suggest that, afterload reduction with SNP in infants with post-asphyxial hypertension and acute heart failure, produces improvement of cardiac pump function, tissue perfusion and oxygenation.