

†1433 HEPARIN PREVENTION OF CATHETER RELATED THROMBOSES. M.M. McDonald, M.L. Johnson, C.M. Rumack, R. Marlar, W.E. Hathaway, Univ Colo School Med, Dept Pediatrics and Radiology, Denver, Blood Bank of SE Wisconsin, Milwaukee.

Thromboses related to umbilical artery catheters (UA cath) contribute significant morbidity to neonatal intensive support. This prospective randomized study was begun to evaluate the efficacy of prophylactic heparinization in the prevention of UA cath related thrombi. To date, 19 preterm infants have been assigned to 1) therapeutic heparinization (20-25U/kg/hr) or 2) infusions of 1-2U/ml heparin (controls). Using 10MHz ultrasound (US) thrombi of 1-18 mm were imaged 1-7 days after insertion of a UA cath. Pulsed doppler velocity in the catheterized iliac was twice that of the uncatheterized iliac and increased to $\geq 3/1$ one to four days prior to the appearance of a thrombus on US. Aortography was performed to corroborate US and doppler findings. With these studies, thrombi were detected in 1/11 heparinized and 4/8 control infants ($p < 0.05$). Heparinization resulted in a \bar{x} Laidlaw clotting time of 153 sec ($N=100$) and heparin levels of 0.2-0.6U/ml without complications including ICH. Baseline protein C (pro C) levels were similar in each group and lower than in term newborns (0.25 vs 0.32U/ml). By day 6, no effect of heparin was noted on levels of AT-III (0.38 vs 0.37U/ml) or pro C (0.36 vs 0.37U/ml), while platelet counts were higher in heparinized infants (247 vs 164 $\times 10^9/l$). The heparinized infants had lower blood velocities in the catheterized iliac artery (48 vs 95 cm/sec on day 6). Therapeutic heparinization inhibits thrombus propagation along UA catheters resulting in less obstruction to blood flow.

†1434 SELECTIVE ELEVATION OF SYSTEMIC BLOOD PRESSURE IN AN ANIMAL MODEL OF PULMONARY HYPERTENSION: AN ALTERNATIVE STRATEGY FOR PPHN. William L. Meadow and Brian F. Rudinsky (Spon. by K. S. Lee), U. of Chicago Medical Center, Department of Pediatrics, Chicago, IL

In persistent pulmonary hypertension of the newborn (PPHN), pulmonary artery pressure (PAP) exceeds aortic pressure (AOP). Pharmacologic therapy directed at selectively reducing PAP more than AOP has been largely unsuccessful. We have developed an animal model of neonatal pulmonary hypertension, and have pharmacologically induced selective elevation of AOP as an alternative approach to PPHN.

Neonatal piglets ($n=6$) were intubated, anaesthetized, and ventilated. AOP, PAP, and cardiac output (CO) were measured directly and continuously. Pulmonary hypertension was induced by intravenous infusion of serotype 1b Group B Beta Streptococci (GBS) @ approximately 10⁷ organisms/kg/min.

During GBS infusion, PAP rose by 131 +/- 60% (S.D.), while AOP rose by 4 +/- 11%. CO fell by 34 +/- 13% in response to GBS. Phenylephrine (PE) was then infused into the septic piglet @ 0.3 mg/kg/dose. After PE, AOP rose by 29 +/- 13% from post-GBS/pre-PE values, while PAP rose by only 3 +/- 7%. CO fell by 29 +/- 19% after PE.

Conclusions: 1) Neonatal pulmonary hypertension can be modeled by GBS infusion in the piglet. 2) PE infusion selectively elevates AOP more than PAP in the septic piglet. 3) Selective elevation of systemic blood pressure may be a feasible therapeutic strategy in human infants with PPHN.

†1435 MEASUREMENT OF MEAN PEAK BLOOD FLOW USING DUPLEX ULTRASOUND IN THE NEONATE. James A. Menke, Paul H. Liu, Mohammed Bashiru, Rex Bickers, Richard McClead, Sponsored by Grant Morrow, Dept. Peds., OSU and Children's Hospital, Columbus, Ohio.

Duplex ultrasonography combines a range-gated Doppler flowmeter with real time 2-dimensional ultrasound imaging to yield measurements of blood velocity and cross-sectional area. Flow is calculated using the equation $F = V * A$ where V = velocity and A = cross-sectional area. To evaluate the validity of this technique we obtained measurements in an *in vitro* model of pulsatile flow (1) and in the dog using the carotid and brachial arteries. Flow calculations of mean peak flow were compared to direct (volumetric) measurements ($n = 50$) of mean flow in the *in vitro* model and indirect (surgically-placed electromagnetic flowmeter) measurements ($n = 40$) of mean flow in the dog. The correlation was 0.90 with the *in vitro* model and 0.80 with the dog model. We then measured mean peak blood flow in the anterior cerebral artery of 13 neonates through the anterior fontanelle. Flow ranged from 0.87 to 10.39 cc/min (mean 3.88 cc/min). We conclude that noninvasive measurement of blood flow with duplex ultrasonography is comparable to other established direct, invasive methods, and may be useful in the evaluation of the intracranial circulation in the neonate.

1. Miles, R.: Transmission of vibration from simulated arterial stenosis. Masters Thesis, Iowa State University, 1976.

1436 CHOLESTEROL, CAROTENE AND BIRTHWEIGHT IN SMOKING MOTHERS Jack Metcalf, Paul Costiloe, Warren Crosby, Harold Sandstead and CE Bodwell, Departments Pediatrics, Biochemistry, Obstetrics, Computer Center, Univ. Okla-Health Ctr., Oklahoma City, OK and USDA/ARS Research Labs, Grand Forks, ND and Beltsville, MD.

It has been suspected that nutrition during pregnancy might counteract the adverse effects of smoking on birthweight (BWT). In our previous prospective studies of ~2100 mothers, 47% were smokers who had 63% of the babies with BWT below our median (3300g). A prospective, randomized, controlled trial of a food supplement (WIC) from 19 weeks to term in 410 of these free-living mothers led to increased mean BWT +91g ($p=0.039$), which was greater in smokers (+115g, $p=0.034$), taking into account sex, gestational age, race, prenatal visits. In 288 mother/baby pairs multiple regression analysis revealed an interaction between maternal midpregnancy (19 weeks) plasma levels of cholesterol (chol), β carotene (βC) and smoking on BWT ($p=0.017$). Prospective data on the interactive effects of βC , chol and smoking on BWT apparently have not previously been noted. High βC ($> 116 \mu g/dl$) and low chol levels ($< 150 mg/dl$) were associated with significantly larger babies (+500g) in the smokers. If βC levels remained high from 19 to 36 weeks of gestation, heavy smokers delivered large babies. If βC levels decreased, the babies were small. In nonsmokers, no interaction between these nutrients on BWT was detected, but BWT was ~300g more with higher chol, i.e., from 116 to 272 mg/dl at midpregnancy, at all levels of βC . We conclude that certain plasma levels of β carotene and cholesterol in smokers might counteract the adverse effect of smoking on fetal growth.

1437 VITAMIN E DOES NOT AFFECT NEUTROPHIL FUNCTION IN PRE-MATURE NEWBORNS. H.J. Mewis, M.S. Rhee, R. Carroll, N. Carrasco, H. Riesenber, A. Bartoletti, A. Geis. (Spon. M.L. Cowger) N.Y. State Dept. Health, Center of Labs and Research, and Albany Medical College, Albany, N.Y. 12208.

Human prematures have low levels of Vit. E. While supplementation with Vit. E seems desirable, this may lead to decreased killing of ingested microorganisms by neutrophils (NP), as suggested by recent reports, thus further impairing newborn host defense. We studied the effects of oral α -Tocopherol (αT) administration to prematures on NP functions in a randomized double blind placebo(P) controlled trial. αT and P groups were of comparable gestational age ($T: 32 \pm 2.2$; $P: 32 \pm 1.6$ wks), birthweight, race, sex, Apgar score, and mode of delivery; 42 babies were in the αT group, 31 in P group. We studied serum vit E, polyunsaturated fatty acid composition, H_2O_2 induced hemolysis, ingestion (Phag) and killing (K) of *S. Aureus* 502-A(SA), by NP. At a dose of 15mg/kg/day, αT did not lead to a significant rise in vit E levels; Phag. and K were unchanged. A second and third group were given 50 and 100mc $\alpha T/kg/day$ po respectively; in these groups serum Vit E levels rose significantly over the level in P group and reached levels $\geq 0.8 mg/l$ in 77% of babies. H_2O_2 induced hemolysis decreased concomitantly. Phag. and K were unaffected however. Both αT and P groups had similar values which did not differ significantly, regardless of the time interval after start of therapy, at which the samples were taken. Our data suggest that supplementation of prematures with αT does not impair or improve NP function and may not be hazardous for these babies. (Supported by NIH HD 14864 and CSC Grant 2-MO-1-RR-00749)

1438 RIFAMPIN AND PENICILLIN THERAPY FOR ELIMINATING NASAL COLONIZATION OF TYPE III GROUP B STREPTOCOCCI (GBS) IN INFANT RATS. D. Delaplaine Millard, Stanford T. Shulman, Ram Yogev. Northwestern Univ. Med. School, Children's Mem. Hosp., Dept. of Pediatrics, Chicago.

Multiple antibiotic strategies have been used in attempts to eradicate GBS from colonized infants and women. However, no chemoprophylactic regimen has been successful in reliably eliminating GBS carriage. Because rifampin (Rif) has been utilized successfully to terminate nasopharyngeal colonization with meningococci, *H. influenzae*, or *S. pyogenes*, we tested the *in vitro* sensitivity of GBS to Rif and the ability of Rif to eliminate GBS from nasally colonized infant rats. The MIC of Rif for 18 strains of type III GBS ranged from 0.1-0.4 $\mu g/ml$. Atraumatic nasal inoculation of 1-2 day-old rats with 10^7 - 10^8 colony forming units of GBS (MIC = 0.1 $\mu g/ml$) twice daily for 4 days resulted in 100% GBS carriage for at least 7 days. Colonized animals were divided into 4 treatment groups: 1) saline, 2) oral Rif (20 mg/kg/dose q 12 hrs x 4 days), 3) intraperitoneal (IP) penicillin (PCN) (50,000 units/kg/dose q 12 hrs x 4 days), or 4) IP PCN & oral Rif. All 73 saline controls and 26/29 PCN-treated animals had continued GBS nasal carriage 36 hours after completion of therapy ($p=NS$). In contrast, only 10/37 Rif-treated animals and 5/42 PCN & Rif-treated animals remained GBS-positive. No rifampin-resistant GBS emerged. Rif or PCN & Rif is significantly more effective in eradicating GBS carriage, compared to saline or PCN ($p < 0.0001$). These data demonstrate that, unlike PCN, Rif (with or without PCN) is effective in eliminating GBS from nasally colonized infant rats. Clinical trials with rifampin appear indicated.