DIMINISHED ANTIOXIDANT ACTIVITY (AOA) OF NEWBORN 1444 PLASMA. J.A.Stockman, D.A.Clark and K.McClellan. Dept. of Pediatrics, SUNY, Syracuse, New York.

Newborn infants appear to be unusually susceptible to oxidative damage. In order to determine if the overall antioxidant potential of one biological fluid, plasma, is demonstrably different in newborns, cord blood plasma from healthy term infants was compared with adult control specimens using the thiobarbituric acid (TBA)-malonyldialdehyde (MDA) reaction as a measure of polyunsaturated lipid autoxidation in a homogenate of brain tissue. In this assay, a 5.0 ml homogenate of calf brain is permitted to incubate with 50 μ l plasma at 37°c for 1 hour. MDA concentrations in the 1 hour and a zero time aliquot are measured by the TBA reaction following trichloracetic acid precipation of protein. AOA of plasma is defined as percentage inhibition of spontaneous autoxidation as measured in an untreated control homogenate. The AOA of cord plasma and adult plasma are as follows:

 $\frac{\text{Cord}}{26}$ Adult 116 22.6+5.6 7.6<u>+</u>3.3 AOA (% Inhib) p < 0.001

No correlation was observed between plasma alpha tocopherol levels and the AOA of either cord plasma or adult plasma. AOA of cord plasma is probably dependent on a wide variety of substances including metal complexes such as ceruloplasm, acids such as ascorbic acid and unsaturated lipid materials such as alpha tocopherol. The exact role of each of these and the relationship of plasma AOA to disease states remains unknown.

ASPHYXIA INDUCED ALTERATIONS IN SERUM AND RENAL CALCIUM, MAGNESIUM AND PHOSPHATE HOMEOSTASIS. B. Stonestreet, A. Laptook, W. Oh, R.C. Tsang. Brown U. Hosp., Providence, RI and U. of Cincinnati, O. Asphyxia has been shown to adversely affect neonatal calcium and magnesium homeostasis. We studied the effect of asphyxia on renal calcium, magnesium and phosphate homeostasis in 12, 1-6 day old newborn lambs. In 6 lambs, hypoxemia, hypercarbia and acidosis was induced by a 38ml/kg deadspace added to the airway for 20 min; the study continued for 140 min, 6 non-asphyxiated lambs were controls. Changes in asphyxiated lambs are shown: U = urinary concentration, V = volume, TRP = tubular reabsorption P. tubular reabsorption P.

	Baseline (B)	Asphyxia	Post-Asphyxia	
Time (min)		20	0-60	120-140
Plasma:	-			
Ca (mg/d1)	10.9±0.5	11.4±0.7	10.3±0.5	10.9±1.0
Mg	1.72±0.1	2.4±0.1*	1.7±0.1	1.8±0.1
P	8.8±0.5	9.9±1.1	9.8±0.5	7.6±0.6
UCaV(µg/kg/m	nin) 2.3±1.6	4.5±2.1	4.6±2.6	0.5±0.2
UMgV	1.5±0.8	4.5±1.8	5.1±3.0	0.8±0.4+
UPV	6.8±2.9	18.1±5.2	38.5±17.2*	5.2±1.6
TRP (%)	88±6	88±5	69±7	94±2
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M±SEM, *p<.05 compared to (B), +p<0.05 decrease compared to controls. Significant changes were not seen in control lambs. Thus, asphyxia induces hypermagnesemia and phosphaturia in newborn lambs; we speculate that magnesium may be released from intracellular stores and phosphaturia may be due to increased calcitonin or parathyroid release.

VASCULAR PROSTAGLANDIN I2 ACTIVITY AS A FUNCTION OF 1446 GESTATIONAL AGE. M.J. Stuart, D.A. Clark, J.B. Allen, J. Slott. SUNY, Upst. Med. Ctr., Dept. Peds, Syr, N.Y.

PGI₂ formation by vascular tissue plays an important role in maintaining the fetal-placental unit. Recently, an abnormality in PGI₂ production has been reported in fetal vessels from women with pre-eclampsia, when compared to term deliveries. The gestational ages of the infants evaluated was however <38 wks. (Prostaglandins 20:105, '80). Since a decrease in PGI2 formation by fetal vessels may be implicated in a variety of placental insufficiency syndromes, it is important to determine if PGI2 production differs in term neonates compared to prematures. We evaluated PGI2 activity from umbilical arteries of infants of various gestational ages in comparison to paired, full term control neogestational ages in comparison to paired, full term control neo-nates. Group I consisted of 7 infants (28 to 32 wks) who were compared to simultaneously evaluated 7 term neonates. Group II comprised 8 infants (33 to 36 wks) who were compared to 8 paired term controls. Mean uptake of ¹⁴C Arachidonic Acid (A.A.) into the vessels of the term controls and Grp I prematures was similar at 8603 ± 1314 (1SD), and 8555 ± 3281 cpm per 30 mgms tissue-The production of 6-Keto-PGF $_{1\alpha}$ (the end product of PGI $_2$) was similar (4.6 \pm 1.7% vs 4.5 \pm 1.4%). Values for Grp II prematures were also similar to paired term control values. Uptake of 14C Water also similar to parted term controls and Grp II prematures were similar at 10170 ± 3059 and 10023 ± 2322 cpm. 6-Keto-PGF $_{10}$ was also similar at $4.7\pm0.9\%$, and $4.8\pm1.2\%$. PGI $_{2}$ activity is not age dependent during the gestational ages of 28 weeks to term.

HARMFUL EFFECT OF LUMBAR PUNCTURE IN NEWBORN INFANTS. 1447 Shyan Sun, Kamtorn Vangvanichyakorn, Zenaida Aranda, Nora Ruiz. (Spon. by Robert Levine) CMDNJ,NJ Med. School, Dept. Neonatology, Newark, N.J.

It has been shown that sick newborn infants tolerate excessive handling very poorly especially in very low birthweight infants. We chose to monitor TcPO2 in 9 term & 7 preterm infants during spinal tap procedure. The indications of CSF tap in 9 term in-

disease. 7 preterm infants were tapped because of suspicion of sepsis, meningitis or intracranial hemorrhage, all had RDS. All showed TcPO2 reduction during positioning & tapping which took av. of 10 min. M Dur M 1 PO2

fants were sepsis & meningitis, none of them had obvious lung

Dur. Tap PO2 < 40 24.8 Torr 0.72 min Lung < 0.05 1657 21-80% 40.6 Torr RDS 5.4 min < 0.05

All preterm infants with lung disease showed significantly larger reduction in TcPO2 (40.6 vs 24.8 torr) during the procedure & took longer to recover compared to term infants. Only 2 out of 9 term infants suffered 3 min (av. 0.72 min) of hypoxia (TcPO2 < 40 torn) while 6 out of 7 preterm infants suffered 4.5 to 20 min. (av. 5.4 min) of hypoxia & resulted in clinical deterioration. We concluded that lumbar puncture in preterm infants with pulmonary insufficiency is a risky procedure which should be performed with utmost care. A prior hyperoxygenation to raise PaO2 to 80-90 torr would help to minimize hypoxia during the procedure. A continuous PO2 surveilance will definitely be helpful.

TRANSCUTANEOUS BILIRUBINOMETRY IN BLACK PRETERM IN-1448 FANTS ON COVERED & EXPOSED SKIN AREAS. Shyan Sun, Kamtorn Vangvanichyakorn, Lertluck Nutakul, Sunanda Ranee, Barbara Glista.(Spon. by Franklin Behrle) CMDNJ-NJ Med. School, Dept. Neonatology, Newark, N.J. Transcutaneous bilirubinometry (Yamanouchi & Minolta Co.) has

been shown to be useful in predicting serum bilirubin concentration in Japanese, white & black term infants (Yamanouchi, Lucey & this author). The same methodology was used to test its validity in 2 groups of preterm infants (Gr.A: No. 11, Wt. 750-1500 gm, 50 method 2.5-18.5mg%, Gr. B: No. 17, Wt. 1500-2500 gm, age 25 hrs-10 days, 50 determinations, serum bilirubin 1.2-14.2mg%). Gr. A showed best correlation at upper thigh (r=0.87, p<0.001, standard error of estimate 1.87) while Gr. B had best correlation at forehead (r=0.82 p < 0.001, standard error of estimate 1.74). Since most primies are born with fair skin which turns darker after exposure to daylight, it was assumed that Tc bilirubin reading at exposed & covered skin (a piece of aluminum foil 1 inch in diameter was patched on 1 thigh since birth) will be different. A simultaneous Tc bilirubin reading was obtained from the covered & exposed thigh on the 4th & 5th day in 6 preterm infants (Wt. 1245-1400 gm). 17 Readings were made on the covered area with a mean of 17.22 & SD of 1.34. The mean-reading on the exposed thigh was 17.50 with SD of 2.24. The student paired T test revealed no statistical significance of the readings from the 2 sites. Since the number is too few at this stage, more data will be needed to confirm this study.

ANTHROPOMETRIC CORRELATES OF CREATININE EXCRETION IN 1449 PREMATURE INFANTS. J.L. Sutphen (Spon. by R.J. Grand).

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Urinary creatinine excretion has been utilized to assess malnutrition, prorate the excretion of urinary metabolites and measure renal function. Creatinine output (mg per kg body weight) increases throughout childhood. There are no data describing the anthropometric correlates of creatinine excretion in the premature infant. Accordingly, we measured urinary creatinine output in timed 24-hour urine specimens obtained in the second week of life on 15 premature infants (mean weight 1.21kg, range 0.760 to kg; mean gestational age 29 weeks, range 26 to 33 weeks). The follower

kg; mean gestational age 29 weeks, range 26 to 33 weeks). The for lowing regression equations were obtained:

mg creat = 9.38 [birth weight in kg] - 1.51; r = .93, p < .001

mg creat = 0.81 [length in cm] - 22.02; r = .89, p < .001

mg creat = 1.21 [gest. age in weeks] - 25.56; r = .81, p < .001

Stepwise regression yielded no linear combination of these

variables that improved the estimate based on weight alone. In a similar manner, consideration of weight and length for age (expressed as percent of 50th percentile) did not improve the estimate based on weight alone. These data demonstrate that creatinine output in mg per kg increases by 15% between 0.7 and 1.5 kg birthweight. This increase parallels that in body protein content per kg obtained from published fetal body composition data in similarly aged infants. Creatinine output in premature infants therefore can serve as a reflection of growth of lean body mass.