†1469 TUBULAR DYSFUNCTION IN INFANTS WITH MECONIUM STAINED AMNIOTIC FLUID - DIAGNOSIS USING B2 MICROGLOBULIN.

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Urinary concentrations of B2 microglobulin (B2M) and creatinine were measured in normal term infants and in those born with meconium stained amniotic fluid (MEC). None of the infants or their mothers had conditions known to modify B2M excretion. Apgar scores for the normal and MEC infants averaged 8.8 and 7.7 respectively at 1 min and 9.0 and 8.2 at 5 min. Urinary B2M to creatinine levels (mg/gm) increased significantly ($p < .01$) in the normal infants from day 1 (1.5 ± 1.3 ; $n=29$) to day 3 (3.5 ± 2.8 ; $n=21$) of life. Compared to the normals, values for the MEC infants were significantly increased (4.7 ± 0.6 ; $n=25$; $p < .005$; day 1 and 12.9 ± 8.9 ; $n=26$; $p < .005$; day 3). Values in 12 of 25 MEC infants on day 1 and 13 of 26 on day 3 exceeded 2 SD above the normal mean. Urinary creatinine excretion was similar in the normal and MEC infants. We propose the increase in B2M levels from days 1 to 3 in normal infants is due to an increased glomerular filtration rate, increasing the filtered load. The elevated levels in the MEC infants indicates the existence of tubular dysfunction, probably mild acute tubular necrosis, in these infants even though other clinical evidence for this lesion was absent. This data supports the concept that MEC reflects stress in these neonates and indicates that urinary excretion of B2M is an extremely sensitive, noninvasive method for detecting even mild tubular damage in these patients. The existence of tubular injury in a high percentage of this patient population has not previously been suspected.

†1470 RED BLOOD CELL (RBC) MEMBRANE DEFORMABILITY AND WHOLE BLOOD VISCOSITY IN INFANTS OF DIABETIC MOTHERS (IDM).

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Impaired RBC deformability decreases RBC survival. In IDM, this may lead to obstruction in microvasculature resulting in NEC, other vascular complications, and hyperbilirubinemia. The present study was designed to evaluate RBC deformability using Ektacytometer (Blood, 61:889, 1983), whole blood viscosity, hematocrit (Hct) and clinical status in IDMs ($N=7$). Controls ($N=7$) were normal infants matched for race, sex, and gestation.

Two IDMs were LGA and 5 AGA, with GA 37-39 wks. IDMs had significant + in T_2 of deformability index (DI), greater RBC surface area and + in RBC water content compared to controls ($p < .05$). IDMs born to insulin-dependent mothers had greater + in RBC DI compared to class A diabetics. No correlation was found in IDMs between DI, viscosity, Hct, and HBA_{1c} levels. There was no significant difference in viscosity and Hct between IDMs and controls. One infant of class C mother had marked + in RBC deformability parameters with Hct of 66%, and normal viscosity of 12.1 cps at a shear rate of 11.5 sec^{-1} . He developed NEC and hyperbilirubinemia (peak bilirubin = 20.6 mg/dl).

Our study suggests that RBCs of IDMs are less deformable, without a change in whole blood viscosity compared to normal infants. This may result in significant clinical manifestations. The biochemical basis for this has been previously shown by us to be alteration in the lipid bilayer, demonstrated by + cholesterol and cross-linking of lipids in the RBCs of IDMs.

1471 ACUTE HYPOTHERMIC STRESS, TRANSIENT TRICUSPID INSUFFICIENCY AND EARLY MATERNAL-INFANT BONDING.

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Skin to skin contact between parturient mother and neonates has become an accepted practice in U.S. hospitals. Such contact is considered critical for better future maternal-infant bonding. Over the past six months we have observed transient tricuspid insufficiency secondary to hypothermic stress in 14 full term infants. All infants were product of normal pregnancy, labor and delivery with apgar scores ranging between 7 and 10 at 1 and 5 minutes respectively and all spent 30 to 60 minutes with mother beginning 30 minutes after delivery. At the end of such contact period infants developed grunting, nasal flaring, cyanosis and rectal temperature less than 96° F. All had a murmur characteristic of tricuspid insufficiency. Chest x-rays, EKG and Echocardiogram were essentially normal. Electrophoretic determinations of MB isoenzyme of creatine phosphokinase (CPK-MB) in serum of all infants were markedly elevated. Total serum creatine phosphokinase ranged from 391 to 2760 IU/L. MB isoenzyme percentage of total CPK ranged from 5 to 23 percent. Total creatine phosphokinase activity was measured at 37° C on a coulter - DACOS Automated Analyzer and an aliquot was subjected to agarose-gel electrophoresis. All infants had uneventful recovery within 48 to 72 hours of age. These observations suggest need for a careful maintenance of thermal environment of neonates during early maternal-infant bonding.

1472 OTITIS MEDIA (OM): A COMMON COMPLICATION OF NASOTRACHEAL INTUBATION (NTI) IN THE NEONATE.

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Auditory Brainstem Responses (ABR) are recorded in the Newborn Special Care Center at NBIMC prior to discharge. From 9/82 to 8/83 252 infants (INFS) had ABR evaluations. One hundred one INFS were ventilated through endotracheal tube. They were assessed by ABR and otoscopy at a mean postnatal age of 39 days (D). The mean birthweight (BW) of intubated INFS was 1895gms (580-4170), and mean duration of intubation was 5.8 D. NTI was used in 98 INFS, oral intubation in 3 and 18 had NTI and oral tubes. INFS <1250gms were intubated with 2.5mm portex tubes; INFS >1250gms had 3.0mm tubes. Conductive hearing loss (CHL) was documented by ABR. All INFS with CHL had OM by otoscopic exam. CHL occurred in 29% (20/101) of intubated INFS, but only in 4.6% (7/151) of non-intubated INFS. Only INFS with NTI had CHL.

	CHL	Normal	CHL: Relationship to side NTI (sNTI)
#INFS	29(29%)	72(71%)	CHL IPS BIL CON UNI
BW gms	1600	2007	sNTI 1 rare BIL
D NTI	9.3	3.5	#INFS 12(41%) 10(34%) 2(7%) 5(17%)

INFS with CHL were of significantly smaller BW and were intubated significantly longer. CHL occurred 41% ipsilaterally (IPS), 34% bilaterally (BIL), 7% contralaterally (CON) and 17% unilaterally (UNI) with BIL NTI. Of 11 INFS with CHL/OM who reached 6 month(MO) follow-up(FU), 9 (82%) resolved. At 3 MO FU 2 of 3 additional INFS had persistent CHL. Six MO FU on remaining INFS will be available for presentation.

†1473 HYPEROXIA INDUCED PULMONARY VASCULAR AND LUNG ABNORMALITIES IN YOUNG RATS AND POTENTIAL FOR RECOVERY

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We assessed the way duration of exposure to hyperoxia affects number and muscularity of pulmonary arteries and alveolar growth and which features show potential for recovery. Thirty-nine 10 day old Sprague-Dawley rats were used; 19/39 exposed to normobaric hyperoxia (F₁₀2) and 20 age matched controls (C); of 19 experimental animals, 6 were exposed for 2 wks. only (F₁₀2-2), 6 allowed to "recover" in room air for 2 wks. (F₁₀2-2+RA) and 7 exposed for a further 2 wks. (F₁₀2-4). At post mortem, ventricular weights (RV, LV) were obtained and lungs injected and fixed inflated for lung volumes (Vol) and morphometric analysis of arteries per 100 alveoli (A/100a), alveoli/mm², medial wall thickness of muscular arteries (%WT) and extension of muscle into peripheral arteries (%Ext).

Group	wgt/g	Vol/cc	RV/LV	A/100a	alv/mm ²	%WT	%Ext
F ₁₀ 2	46	2.56	.3319	2*	418	8.0	39**
C-2	45	2.64	.3302	3	415	7.5	6
F ₁₀ 2-2+RA	122	5.13*	.3193	4*	359**	12.4*	53**
F ₁₀ 2-4	95*	3.5	.3350	1**	374	8.6	85**
C-4	110	3.81	.3149	5	426	6.4	6

Mean values given: * $p < .05$, ** $p < .001$ from age matched control.

Increasing duration of hyperoxia causes failure to thrive and progressively severe reduction in arterial number and extension of muscle. During "recovery" there is compensatory increase on arterial number but a large lung results from growth of a normal number of large alveoli; also despite return to normoxia, extension of muscle progresses and medial hypertrophy is apparent.

Sponsoring member's name and signature

1474 CESSATION OF BENZYL ALCOHOL (BA) HAS NO EFFECT ON MORTALITY BUT INCREASES MORBIDITY.

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The FDA urged the discontinuation of Benzyl Alcohol in May 1982. Studies since have implicated BA in the increased mortality in very low birth weight (VLBW) infants. We reviewed six monthly mortality rates for approximately 35 months in VLBW infants (501 - 1250 gms) from January 1981 to November 1983 (periods I - VI) and did not find such an association. Of significance, was the difference in mortality between period I and all other periods (I:7/10; II:6/22; III:3/15; IV:4/28; V:2/20; VI:2/18; $\chi^2 = 16.97$, $p < .001$ for 501 - 1250 gm), showing a decrease in mortality 12 months preceding the discontinuation of BA. Excluding period I, the mortality among periods II - VI was not significantly different ($\chi^2 = 1.48$, $p > .05$). The difference in mortality prior to and after 6/30/82 was not significant ($\chi^2 = 1.75$, $p < .05$).

However, we did notice an increase in nosocomial infections after the cessation of BA:

	Before	After
No. of infections	5	15 $\chi^2 = 5.90$, $p < .02$
Care days	7433	6191

Conclusion: Cessation of BA did not change mortality in VLBW infants but did increase nosocomial infections in our newborn ICU.