1558 FAILURE OF FUROSEMIDE TO AFFECT THE CLINICAL COURSE OF TRANSIENT TACHYPNEA OF THE NEWBORN. Thomas E. Wiswell, Franklin R. Smith and James S. Rawlings (Spon. by James W. Bass). Department of Pediatrics, Tripler Army Medical Center, Honolulu.

Transient Tachypnea of the Newborn (TTN) prolongs neonatal hospitalization and occasionally results in serious complications. The most widely accepted theory of the etiology of TTN is a delay in the absorption of fetal lung fluid. Furosemide has been shown to affect fluid dynamics in the lung. We hypothesized that a pharmacologically-induced diuresis of body water may reverse the mechanism of TTN and shorten the course of the illness. We designed a controlled, prospective trial of oral furosemide in infants with TTN. 50 consecutive infants presenting with the clinical and radiographic findings of TTN were randomly assigned to two groups. Infants in the treatment group received 2 mg/kg of oral furosemide at the time of diagnosis followed by a 1 mg/kg dose 12 hours later if the tachypnea (RR > 60) persisted. Infants in the control group received an equal volume of placebo. There were no significant differences in the prenatal and perinatal courses, nor in the demographic characteristics between the two groups. Although mean weight loss at discharge was similar in the two groups, the furosemide-treated group lost significantly more weight in the first 24 hours after birth ($p<0.001$). Compared to infants in the control group, the furosemide-treated infants demonstrated no statistically significant difference in the duration and severity of symptoms, nor in the duration of hospitalization. We conclude that oral furosemide, at the doses used in this study, does not alter the clinical course of TTN.

●1559 MYOINOSITOL (INO) EXCESS INDUCES FETAL GROWTH IN HYPOGLYCEMIC RABBITS. Paul Wozniak, Jack Sills, Mikko Hallman, Louis Gluck. Dept. of Ped. Univ. of Calif. San Diego, La Jolla, CA.

The macrosomia of infants of diabetic mothers (IDM) is believed due to fetal hyperglycemia & hyperinsulinemia. Serum INO, a possible growth factor, is elevated among IDM. Therefore, we studied whether elevated INO is associated with IDM growth stigmata. Pregnant (27-day) rabbits were infused with insulin IV for 48 hrs to maintain blood glucose between 45-90 mg/dl. Group 1 (4 does): continuous INO infusion, 2 gm/kg/day; Group 2 (3 does): no INO. All had free access to water & lab chow. The groups had similar IV total fluid intake/kg maternal serum glucose & insulin/kg/hr. The does were sacrificed on pregnant day 29 and the fetuses studied for growth.

Serum INO was significantly higher in Group 1 fetuses than in Group 2 (<.001), with no significant fetal serum C-peptide differences between groups. Group 1 (INO) fetuses had significantly higher body, liver & brain wt., but lung weights were not different (Table).

	Group 1	Group 2	
Body wt. (gm)	38.96 ± 4.34	33.33 ± 7.38	p<.001
Liver wt. (gm)	2.52 ± 0.35	2.17 ± 0.64	p<.015
Brain wt. (gm)	0.98 ± 0.12	0.89 ± 0.12	p<.006

We have demonstrated that excess INO, found also in IDM, increases body, liver & brain wt. despite low maternal & fetal glucose. This suggests possible importance of hypermyoinositolemia in the pathogenesis of macrosomia in the IDM.

1560 MATURITY VERSUS WEIGHT AS PREDICTORS OF SYSTOLIC BLOOD PRESSURE (SBP) IN LOW BIRTHWEIGHT (LBW) INFANTS DURING THE 1st SIX WEEKS (WKS) OF LIFE. Linda L. Wright, Tom L. Wright (Spon. By Allen Schwartz) Univ. Md. Sch. of Med., Dept. Peds., Balto. and Catholic Univ. Amer., Wash., D.C.

Although research in SBP in newborns suggests that chronological age (CA) and gestational age (GA) may be predictors of SBP, weight (WT) is more commonly used to evaluate SBP in LBW infants. This study was designed to determine the contribution of WT versus CA, GA and postconceptual age (PCA=GA+CA) to SBP in LBW infants (birthweight BW≤1500 gm) over the 1st wks of life. The SBP of 33 symptomatic infants (BW=1071±242 gm, GA=29.1±1.9 wks) was determined daily for the 1st 6 wks of life using a Dinamap monitor. Three maturity effects of SBP were noted: GA at birth, CA during the 1st wk and PCA during wks 2-6. At birth GA, BW and SBP were highly correlated: GA/BW (.75), GA/SBP (.50), BW/SBP (.33). Regression analysis (RA) revealed that at birth GA made an independent contribution (IC) of 16%, GA/WT overlapped to predict 9%, BW made no IC to SBP. SBP during the 1st wk of life was correlated with CA (.49), GA(.41), WT (.20). RA revealed that the independent contributors to SBP during the 1st week of life were CA 24%, GA 7%, WT 0%. The overlap between GA/WT contributed 10% of the variance. During wks 2-6 PCA was the most important predictor of SBP. PCA/SBP were correlated (.40); PCA made an IC of 5% of the variance in SBP and combined to predict an additional 11% of the variance during wks 2-6 (multiple R=.40). This study suggests that the common practice of using WT to evaluate SBP in LBW infants ignores the majority of systematic variance available from each of the three operations of maturity: GA, CA and PCA.

1561 POSTCONCEPTUAL AGE (PCA) AS A BASIS FOR SYSTOLIC BLOOD PRESSURE (SBP) NORMS IN LOW BIRTHWEIGHT (LBW) INFANTS. Linda L. Wright, Thomas L. Wright (Spon. by Allen Schwartz). Univ. MD Sch. Med., Dept. Peds., Balto., and Catholic Univ. of America, Washington, D.C.

Chronological age (CA) and gestational age (GA) are often used to predict newborn SBP. The purpose of this study was to test the contribution of PCA (PCA=CA+GA) to SBP and to use PCA as a basis for SBP norms in LBW infants. 33 LBW infants (BW=1071±242, GA=29.1±1.9 wks) were studied with daily Dinamap SBP determinations to a CA=6-8 wks. Infants were grouped by GA: (1)25-27 wks (n=8); (2)28-29 wks (n=10); (3)30-32 wks (n=15). At birth SBP was: Grp 1 =50.7±7.0, Grp 2=54.0±8.0, Grp 3=56.6±8.0. SBP increased significantly in each grp during wk 1 (CA effect). SBP rose differentially in the 3 grps after wk 1 of life; PCA predicted the time required to reach term newborn SBP levels. The SBP of grp 3 plateaued after wk 1 at 68.1±6.3; the SBP of grp 2 rose wks 1-2 ($p<.001$) and wks 2-5 ($p<.03$), plateauing after wk 5 at 69.8±6.4. Grp 1 SBP rose wks 1-2 ($p<.06$) and wks 2-7 ($p<.01$); SBP at wk 7= 68.8±7.3. The mean PCA of all GA grps upon achieving the SBP plateau was 32-33 wks. Achievement of term SBP levels at a similar PCA and differing CA supports the construct validity of PCA as a predictor of SBP. SBP norms by PCA with the Dinamap method (after the wk 1 CA effect) are:

PCA	26	27	28	29	30	31	32	33	34	35	36	37
(n)	1	3	8	14	17	22	22	18	13	7	3	3
SBP	50.0	54.3	61.5	65.1	65.2	66.7	67.4	68.7	67.2	69.9	71.0	72.0
SD		3.5	10.0	6.5	5.0	6.6	7.2	5.6	3.0	3.1	4.0	3.5

These data document an initial CA effect in all GA groups and the continued rise in SBP to a PCA of 32-33 weeks.

†1562 PERINATAL ASPHYXIA AND POSTNATAL CHANGES IN SERUM TOTAL AND IONIZED CALCIUM. Nam Dong, Paul Y.K. Wu, Bijan Siassi, Lota Viray. Univ. of So. Calif. Sch. of Med., LAC-USC Med. Ctr., Dept. of Peds., Los Angeles.

Calcium (Ca) supplementation is used frequently in neonates in the early postnatal days or following asphyxia and resuscitation. The rationale for this supplementation has been based on the laboratory finding of "low" serum total Ca concentrations. Recent studies have shown that total Ca is poorly correlated to ionized Ca^{++} in the serum. Serum total and ionized Ca^{++} with concurrent blood pH were measured sequentially, with an ICA 1 ionized Ca^{++} Analyzer (Radiometer), in 10 term infants (BW=3381±772g, GA=40± 1.5 wks) at 0, 4, 24 and 48 hours postnatally. All infants had fetal distress with moderate to severe variable or late decelerations in their fetal heart rate tracings. Eight infants required emergency Caesarian Section, and 2 were forceps assisted vaginal deliveries. Apgar scores were 1.7±0.9 and 7.2±1.5 at 1 and 5 minutes respectively. Serum total creatine phosphokinase (CPK) was elevated at 24 and 48 hours and the MB fraction of the CPK isoenzymes was the fraction most elevated. Serum total Ca was 11 ±1.1 at birth and fell to 8.7±0.8 and 9.3±1.8 mg/dl at 24 and 48 hours respectively. In contrast, the actual ionized Ca^{++} remained unchanged, being 1.2±0.4, 1.2±0.05, 1.2±0.08 and 1.2±0.05 mM/dl at 1, 4, 24 and 48 hours respectively. In view of the reported complications of excess ionized Ca^{++} producing vasospasm, ischemia, angina, cardiac infarction in adults, and the hazards of intravenous Ca administration in neonates, Ca supplementation should be used with caution. Data from this study suggest that asphyxiated neonates do not require routine Ca supplementation.

1563 FACTORS AFFECTING UNBOUND BILIRUBIN LEVELS IN INFANTS Yoshitada Yamauchi, Audrey K. Brown, Jed Turk, Barbara Delivoria & Gerard J. Boyle. SUNY-Downstate Med. Cntr. Dept. of Pediatrics, Brooklyn, NY.

Unbound bilirubin(UB) was measured in the blood of 79 infants using a micro automated modified peroxidase method employing the UB Analyzer developed by Nakamura. This simple micromethod was found to correlate with the manual method and showed very good reproducibility(Coeff.var.4%). Several factors influencing the level of unbound bilirubin were examined, including gestational age, birth wt., sick vs. well, post-natal age, total bilirubin(TB), total protein(TP), and phototherapy. Direct correlation was found between TB and UB ($r=0.69, p=.0001, n(sample)=145$) and negative correlation with total protein vs UB($r=-0.24, p=0.001, n=152$). The ratio TB/TP vs UB correlated directly($r=0.88, p=0.0001, n=145$). Unbound bilirubin was higher in the first 168 hrs of life than in infants >168 hrs of life. At the same total bilirubin level, there was a dramatic difference in the amount of unbound bilirubin in blood of infants of BW<1000 g than in infants ≥1000 g. There was a sequential decrease in UB with increasing BW. Sick infants, had higher UB than did well infants in the same weight group. At a given serum bilirubin level, unbound bilirubin was higher before phototherapy than during or after phototherapy. It is clear that several factors in addition to total bilirubin determine the concentration of unbound bilirubin in the blood of newborn infants. The UB Analyzer affords a simple reproducible micromethod for measurement of UB in the clinical setting. The ratio TB/TP corresponded better with UB than did TB alone and could be useful as a screen for UB.