1338 A COMPARISON OF SERUM ENZYMES IN VENOUS AND ARTERIAL CORD BLOOD AT BIRTH. Marvin Leventer, Mehmet Y. Dincsoy, Soo Jae Kim, Foazia Siddiq (Sponsored by P.J. Collipp). Health Sciences Center, SUNY at Stony Brook, Nassau County Medical Center, Department of Pediatrics, East Meadow, NY.

Serum enzymes in the human fetus may originate from mother, placenta or the fetus. We studied paired samples of umbilical cord artery (A) and venous (V) serum enzyme activities, namely, creatine phosphokinase (CPK), lactate dehydrogenase (LDH), aspartate aminotransferase (AST), amylase and alkaline phosphatase (Alk-P). The study group consisted of a total of 30 infants with a birth weight (mean±SD) of 3040±501 gm, and gestational age of 38.6±2.4 wks. The following is a comparison between venous and arterial serum levels of the remaining enzymes:

	CPK	AST	LDH	AMYLASE	Alk-P
Venous (U/L)	269±97 *	33±16 *	273±100 *	35±13 *	373±154
Arterial (U/L)	311±115	43±22	313±124	42±16	379±126
ΔV-A +/-(%)	2/15(12)†	2/16(11)†	6/20(23)†	8/22(27)†	8/20(29)†

Paired t: *$p < 0.05$; Wilcoxon signed rank test: †$p < 0.05$

Serum CPK, LDH and amylase were higher in the serum of A than V and the direction of changes were significantly upward from V to A. The negative V-A gradient of these enzymes in cord serum represents fetal contribution and/or placental uptake during each passage of feto-placental circulatory cycle. Intrapartum fetal and placental disorders are expected to produce more acute changes which may be instrumental for further understanding of this issue. Supported in part by grant MOD-226.

1339 BLOOD GLUCOSE (BG) CONCENTRATIONS AND CRANIAL ULTRASOUND (US) ABNORMALITIES IN LOW BIRTH WEIGHT (LBW) INFANTS. Mehmet Y. Dincsoy, Foazia Siddiq, Marvin Leventer, Kathleen McKeever, Susan Tuck. (Spon. by Platon J. Collipp). Health Sciences Center, SUNY at Stony Brook, Nassau County Medical Center, Department of Pediatrics, East Meadow, NY.

Hyperglycemia by producing hyperosmolar state and hypoglycemia by an induction of catecholamine release both have been claimed to play a role in the causation of subependymal-intraventricular hemorrhage (SE-IVH). Theoretically, it is expected that BG levels within the first day of life will have an influence on the incidence of SE-IVH in LBW infants. We studied the relationship between SE-IVH and BG in 58 LBW infants (≤2000 gm) by US study of the head routinely at least once within the first 5 days of life. The mean, highest and lowest BG levels were determined during the first 24 hours of life. The infants studied had a birth weight (mean±SD) of 1475±561 gm, 1 and 5 minutes Apgar scores of 5.4±2.3 and 7.2±2.1 respectively. The following is the comparison of the infants with normal and abnormal cranial US as they relate to BG status during the first 24 hours of life:

	Blood Glucose (mg/dl)			Highest BG		Lowest BG	
Groups	Mean	Highest	Lowest	>125/≤125		≤30/>30 mg/dl	
Abnormal US	76±17	112±26	44±16	7/16	(30%) *	5/18	(22%)
Normal US	69±20	109±30	45±15	6/29	(17%)	6/29	(17%)

*$\chi^2 = 2.61$, p=ns

Since there is no significant difference between the groups with regard to glucose homeostasis, the effect of abnormal BG concentrations on the incidence of SE-IVH seems to be absent or questionable and needs to be analyzed in a larger sample.

1340 USE OF DEXAMETHASONE (DXM) IN SEVERE NEONATAL RESPIRATORY DISEASE SECONDARY TO PERSISTENT PULMONARY HYPERTENSION (PPHN). Roger G. Faix and Steven M. Donn, Dept. of Pediatrics, Univ. of Michigan, Ann Arbor, MI. (Spon. by G.W. Goldstein).

Eight newborn infants, gestational ages 32-42 wks (mean 37 ± 3.6) and birthweights 1870-4160 gms (mean 2899 ± 687) were treated with DXM, 1.0 mg/kg IV every 12 hrs for 4 doses for severe respiratory disease secondary to PPHN. All were receiving IPPV and could not be weaned below an FiO_2 of 0.65; 7 of 8 were receiving $FiO_2 \geq 0.85$. Mean age at DXM therapy was 9.8 days (range 6-17) and mean prior duration of FiO_2 1.0 was 123 hrs (range 56-189). Parameters monitored before, during, and after therapy included mean airway pressure (PAW), ventilator rate (IMV), FiO_2, and alveolar-arterial oxygen gradient (A-a DO_2). Changes in these parameters from initiation to completion of therapy were:

	Δ PAW	Δ IMV	Δ FiO_2	Δ A-a DO_2
mean ± SD	3.6±2.1	14.4±9.2	0.30±0.13	221±77
range	(0.2-5.8)	(0-29)	(0.18-0.59)	(145-383)

The mean time until FiO_2 could be reduced to ≤ 0.5 was 60.7 hrs (range 33-123). The mean time until IMV could be halved was 84.9 hrs (range 28-150). All infants were receiving systemic antibiotics when therapy began; no significant new infections occurred. Transient elevations in serum sodium (2), blood pressure (2), and serum glucose (2) were noted but were not clinically significant.

Our observations suggest DXM is beneficial in the treatment of severe respiratory disease in newborns requiring intensive ventilatory management beyond 5 days of age. A controlled clinical trial will be necessary to demonstrate long-term safety and efficacy.

1341 EFFECTIVENESS OF EARLY ARGININE (ARG) THERAPY IN ARGININOSUCCINIC ACIDURIA (ASA). Steven M. Donn and Jess G. Thoene, Dept. of Pediatrics, University of Michigan Hospitals, Ann Arbor, MI.

The effectiveness of ARG therapy in preventing neonatal hyperammonemia is illustrated by the outcomes of two preterm siblings with ASA. The first case was referred at 4 days of age with lethargy, apnea, coma, and a plasma ammonia concentration of 1992 µg/dl. She received hemodialysis, lowering ammonia to 133 µg/dl, but a rapid rebound to 843 µg/dl prompted institution of ARG therapy, 4 mM/kg/d as 5% ARG-HCl. This lowered ammonia to normal concentrations for the remainder of the hospitalization, at a plasma ARG concentration of 360 µM. At 20 months she is profoundly retarded, cannot sit or stand, and functions at a 3 month level. The second case was diagnosed antenatally by the presence of ASA in amniotic fluid. A male child, delivered at 32 weeks, began receiving ARG therapy at 32 hours of age when the plasma ammonia had increased to 196 µg/dl. ARG, at the same dose, produced a plasma ARG concentration of 184 µM and successfully prevented hyperammonemia. At four months of age his neurologic examination is normal and his development is age-appropriate. Both children have been maintained on a protein-restricted diet supplemented with oral arginine and ornithine acetate, providing 2 mM/kg/d of each.

Treatment of inborn errors of the urea cycle has advanced greatly in the past decade with the advent of alternative means of waste nitrogen disposal. Early diagnosis and treatment of ASA with ARG appears to be a highly effective therapy of an otherwise severe and often lethal inborn error of metabolism.

1342 MORBIDITY AND MORTALITY OF MULTIFETAL GESTATION LESS THAN 1500 GRAMS. Steven M. Donn and Rose M. Viscardi (Spon. by G.W. Goldstein), Dept. of Pediatrics, University of Michigan Hospitals, Ann Arbor, MI.

The clinical courses and outcomes of 61 twins and 9 triplets with birthweights less than 1500 grams (multifetal group, MFG) and delivered at Women's Hospital between Jan. 1980 and June 1983 were compared to all 174 inborn singletons less than 1500 grams (singleton group, SG) delivered during the same interval.

MFG infants were significantly smaller (birthweights 976 ± 277 g v. 1111 ± 238 g for SG, $p<0.001$) and more premature (gestational ages 28.2 ± 2 wks v. 29.3 ± 3 wks for SG, $p<0.01$). Mortality for the MFG was 39%, compared to only 17% in the SG, $p < 0.001$.

Analysis of obstetric factors between MFG and SG showed premature onset of labor ($p < 0.001$) and cesarean section (CS) following labor ($p < 0.001$) to be more common in the MFG, while CS without labor was more common in the SG ($p = 0.002$).

The incidence of respiratory distress (RD), IPPV and $FiO_2 \geq 0.4$, was higher in the MFG (83%) than in the SG (69%), $p<0.05$. The overall incidence and severity of intraventricular hemorrhage (IVH) was similar in both groups, though factors associated with IVH differed. Among the MFG, IVH was more commonly seen only in infants with RD. For the SG, IVH was more common with birthweight <1000 g ($p = 0.02$), 5 minute Apgar ≤ 5 ($p<0.03$), RD ($p<0.0001$), and death ($p<0.0001$). Postnatal phenobarbital therapy reduced the incidence of IVH in the SG from 37% to 17%, but had no apparent effect in the MFG.

We conclude that MFG infants tend to be more premature and smaller than SG infants, and have an increased risk of mortality which does not appear related to IVH.

1343 PREDICTIVE FEATURES OF MORTALITY AND MORBIDITY DUE TO GROUP B STREPTOCOCCAL (GBS) MENINGITIS. Morven S. Edwards, Marcia A. Rench, and Carol J. Baker, Baylor College of Medicine, Department of Pediatrics, Houston.

Clinical and laboratory features at admission for GBS meningitis have not been assessed as possible predictors of mortality or major neurological sequelae. From 1974 through 1979, 61 patients were admitted with this diagnosis. Infection was rapidly fatal in 13 (21%) and 38 of 48 (79%) survivors were evaluated at a mean of 5.8 years later (range 3.3 to 8.9). Of these 51 infants, 21 (41%) ultimately died or had major neurological sequelae, while 30 (59%) were normal or had minor residuae. Analysis of admission parameters revealed a significant risk of death or major morbidity in patients who had coma or semicoma, decreased peripheral perfusion, total WBC<5,000/mm³, absolute PMN<1,000/mm³, serum HCO_3<15 mg/dl and CSF protein>300 mg/dl ($p \leq 0.03, \chi^2$ analysis). Features not significantly associated with poor outcome included seizures prior to admission, duration of symptoms ≥ 24 hr. before diagnosis, ratio of immature to total PMN≥0.2, WBC<100/mm³ in CSF, CSF gram stain, and CSF glucose < 10 mg/dl. Among the 14 infants with early-onset meningitis, the mean birth weight of 5 with a fatal outcome (2.056 kg) was significantly less than that of 9 survivors (3.277 kg), ($t=3.3$, $p<0.01$). The serious morbidity and mortality declined from 80% (1974) to 25% (1979). Application of these data may allow the prospective identification of patients at high risk for major sequelae associated with GBS meningitis. Furthermore, cautious optimism may be communicated to parents when infants do not have features predictive of major morbidity.