●1576 RELATIONSHIPS BETWEEN FETAL HEART RATE MONITORING(FHR) AND CREATINE PHOSPHOKINASE-BB (CK-BB), Rochelle C. Feldman, W. Donald Shields, Khalil M.A. Tabsh, UCLA School of Medicine, Center for Health Sciences, Dept. Pediatrics and Ob-Gyn, Los Angeles, CA 90024 (SPON. by Dale L. Phelps).

Significant elevations in CK-BB (>20) correlate well with moderate degrees of perinatal asphyxia and are particularly valuable in assessing the degree of associated brain damage. FHR has been utilized in the obstetrical community to predict antenatal fetal hypoxia. We prospectively evaluated FHR in 34 intrapartum patients and measured cord blood CK-BB at time of delivery. A scoring system (0-8) for FHR was devised as follows:

	SCORES		
Parameters	0	1	2
Baseline heart rate (BPM)	<100	100-120	120-160
	>180	160-180	
Beat-to-beat variability	<2	3-6	>6
Acceleration	None		Present
Deceleration	Severe	Mild	None
	variable	variable	Early
	Mild/severe late		

A linear regression analysis of CK-BB vs FHR scores showed an r = -0.34. However, significantly more babies with low FHR scores (1-3) had CK-BB >20 when compared with babies whose FHR scores were ≥7 (Fischer's exact test, p=0.017). Severe intrapartum alterations in fetal heart rate patterns are associated with significant elevations in CK-BB. We conclude that ominous FHR patterns are associated with asphyxial damage to the central nervous system.

● 1577 CEREBRAL METABOLIC RESPONSES TO ASPHYXIA AND RECOVERY IN NEWBORN DOGS. J Hellmann, EE Nardis and RC Vannucci. Penn State Univ Coll of Med, M. S. Hershey Med Ctr, Dept of Peds, Hershey, PA.

We used the arterio-venous (A-V) difference method to assess metabolic restoration of the brain following asphyxia in newborn dogs. Brain metabolites and energy reserves were analyzed (after *in situ* freezing) during and following the asphyxia produced by respiratory arrest for 8 min. pHa 6.9, $paCO_2$ 96, paO_2 10.

Blood Metabolites (mMol/L)	Control n=23	Asphyxia n=14	10 min n=12	30 min n=7	120 min n=4
glucose	8.64	7.94	10.24	9.88	11.24
$A\text{-}V_{glucose}$	0.74	*2.73	0.57	*1.10	*1.25
lactate	1.36	*7.85	*7.00	*5.55	*6.14
$A\text{-}V_{lactate}$	0.01	-0.02	0.05	*0.36	*0.48
Brain Metabolites	n=4	n=4	n=4	n=3	n=2
glucose (mMol/kg)	3.26	*0.73	2.81	3.39	4.28
pyruvate	0.073	0.051	0.116	0.072	0.09
lactate	1.72	*9.82	*7.09	*5.61	*4.04
ATP	2.33	*1.45	2.44	2.51	2.43
P~Creatine (P~Cr)	2.99	*0.34	2.71	2.85	3.57

*p<0.05 vs. control values

These findings indicate that during asphyxia anerobic metabolism exceeds glucose availability leading to depletion of ATP and P~Cr. During reoxygenation the increased pyruvate at 10 min, positive $A\text{-}V_{lact}$ and rebound in brain glucose indicate that lactate becomes the preferred substrate for oxidative processes during recovery.

1578 DOCUMENTATION AND PROGRESSION OF INTRACRANIAL HEMORRHAGE (ICH) BY 2-D ECHOENCEPHALOGRAM. Richard W. Henderson, Morton L. Cohen, Larry S. Johnsgard, Michael L. Segall, and Alan E. Shumacher, (Spon by Stanley E. Kirkpatrick) Children's Hospital & Health Center, San Diego, CA.

115 infants weighing 0.45 - 2.5 kilograms were screened for ICH using the Mark III A.T.L. 90° scanner with a 5 MHz transducer. Head scans were obtained on day 1, 3-5, 14, and more frequently as indicated. ICH was divided into 3 classes: Germinal Matrix (GM), Germinal Matrix with Ventricular Extension (VE) and Germinal Matrix with VE and/or Parenchyma Extension (PE). All surviving infants were followed to resolution or hydrocephalus. VE was treated with serial taps and if progressive hydrocephalus developed, ventricular peritoneal shunts were placed. 38 infants were identified as having ICH, 21 (55%) survived. 11/21 (52%) progressed to hydrocephalus, 5/11 (45%) of which required a V-P shunt. All ICH's began as GM bleeds, 14/38 (37%) remained so with 11/14 (80%) surviving. 13/38 (34%) progressed to VE; 8/13 (62%) surviving. 11/38 progressed to PE, 2/11 (18%) surviving. Shunt obstruction occurred in one infant requiring replacement. Mean age for documentation of initial bleed was 3 days post-birth, progression occurring within 24 - 48 hours. 12/38 (32%) were detected in the first day, 9/12 (75%) expired. Resolution of GM hemorrhage varied over 1 - 4 weeks, depending on size, and only 1/11 (9%) showed transient ventricular dilatation. Resolution of VE hemorrhage varied from 2-8 weeks. 6/13 scans (46%) revealed acute dilatation with 3/13 (23%) progressing to V-P shunts. 9/11 infants with PE expired (82%). Both survivors required V-P shunts.

1579 CEREBROSPINAL FLUID ACID-BASE REGULATION IN NEWBORNS. Marcus C. Hermansen, Patricia H. Ellison (spon. Frederick M. Blodgett), Med. Coll. of Wisconsin, Dept. of Peds, Milwaukee, Wis.

Cerebrospinal fluid (CSF) acid-base regulation is an important determinant of cerebral blood flow, and ventilatory drive. We report the CSF acid-base values in 14 newborns with 16 lumbar punctures (mean gest. age 33.8 wks, range 26-44). All had stable respiratory conditions and blood pH≥7.30 and ≤7.50. Eleven samples were from patients with normal cell counts, glucose, protein, and negative cultures; 5 were from patients with intraventricular hemorrhage (IVH) documented by CT scan. pH and pCO_2 determinations were made on the CSF and blood samples, with $[HCO_3^-]$ subsequently calculated. The results follow:

	Capillary Blood			CSF			Gradient		
	pH	pCO2	HCO3	pH	pCO2	HCO3	pH	pCO2	HCO3
$\bar{X}$	7.402	33.6	20.6	7.366	40.3*	21.8	-0.036	6.7	1.2
S.D.	.058	6.6	4.0	.069	6.7	3.3	.037	4.8	1.8
Range	7.310-	26.1-	16.9-	7.255-	32.8-	16.3-	-0.116-	3.2-	-4.2-
	7.500	50.4	32.4	7.476	56.1	28.1	+0.023	13.3	4.0

*p<0.01 (paired t-test)

The samples from patients with IVH did not differ (p>0.05) from those without IVH (t-tests). There was no trend for any blood-CSF acid-base gradient to vary with gestational age (Kendall's tau rank correlation methods). These findings of a slightly acidotic CSF due to an increased pCO2 are similar to those from adult studies, suggesting that CSF acid-base regulation is developed by 26 weeks gestation.

1580 HYPERTHERMIA-INDUCED SEIZURES IN THE RAT PUP. DAVID HOLTZMAN, KATHRYN OBANA, JAMES OLSON; STANFORD UNIV. SCH. MED., DIV. CHILD NEUROLOGY, STANFORD, CA.

Hyperthermia-induced seizures were studied in rat pups as a model for febrile convulsions in young children. Sprague-Dawley albino pups were warmed by an infra-red lamp held over a copper-covered lucite chamber. Rectal or rectal plus brain temperatures were monitored with a thermistor or with copper-constantin thermocouples. Two day old pups developed multifocal clonic seizures at 37°C (rectal). Five day old pups showed generalized seizures at about 40.5°C and seven day old pups at 43°C. All these pups survived a brief post-ictal depression. At ten days, only half the pups survived seizures at 44-45°C. At older ages seizure-like activity occurred above 45°C and all animals died. The threshold temperatures for seizures were independent of the rate of temperature rise. Differences between brain and rectal temperatures in immature rats warmed rapidly caused the maturational increase in seizure thresholds, measured by brain temperature, to be less between 2-5 days and more between 5-7 and 7-10 days than measured by the rectal probe. Electroencephalograms recorded in six and ten day old pups confirmed electrocortical seizures at higher temperatures.

Thus, in the albino rat pup, hyperthermia produces seizures with increasing temperature threshold between 2-10 days of age. This result in an animal model suggests that elevated body (brain) temperature alone may cause or contribute to epileptogenesis in the young child. [Supported by grants to DH from NINCDS (NS16256) and the Cerebral Palsy Foundation.]

1581 Blood-brain barrier changes in acute hepatic encephalopathy. Marc Horowitz, Daniel Schafer, Peter Molnar, Ronald Blasberg, and Joseph Fenstermacher, NIH, Bethesda, MD 20205

Blood-to-brain transport constants (k) for ^{14}C-α-aminoisobutyric acid (AIB), a marker of brain capillary permeability, were measured in rabbits with acute hepatic encephalopathy induced by the hepatotoxin, galactosamine (GAL). Tissue ^{14}C-activity was assayed on 20 micron thick coronal sections of frozen rabbit brains by quantitative autoradiography; adjacent sections were prepared for histological examination. Experiments which were performed 2 and 10 hours after the administration of GLA (times when the rabbits were neurologically normal) showed no changes in the k values of AIB from the untreated controls. Eighteen hours after GAL administration, the rabbits were comatose and their brains were edematous; in addition, five-to ten-fold increases in blood-brain barrier (BBB) permeability to AIB were found in gray matter (no permeability changes were detected in white matter). These observations suggest that the permeability of the BBB not only to AIB but also to other solutes (notably, Na and Cl) and to water has increased in gray matter and that the role of the BBB as a regulatory of brain volume has been compromised by these changes, thereby leading to cerebral edema. A similar pathogenesis may be involved in other diseases in which hepatic dysfunction frequently leads to acute cerebral edema, elevated intracranial pressure, and death (for example, Reye's syndrome).