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**CEREBROSPINAL FLUID/BLOOD GLUCOSE RATIOS IN PREMATURE AND FULL-TERM INFANTS.** Michele Monaco, Pamela Kopen, Emily Bloom, Arthur Carter & Robert Vannucci (Spon: N.M.Nelson)

Penn State Univ-M S Hershey Med Ctr, Dept Ped, Hershey Pa. Low CSF glucose concentrations occur in inflammatory & malignant conditions which affect the CNS & during hypoglycemia. However, alterations in CSF glucose in newborn infants may be attributed to "physiologically" low blood glucose levels. Knowledge of the CSF/blood glucose ratio may aid in minimizing diagnostic errors, but this relationship has not been fully explored. Charts of 1100 neonates admitted to the HMC newborn ICU were reviewed; of these, 219 underwent 240 LPs with near simultaneous collection of blood for glucose determination. Spinal fluids were analyzed without regard to protein content, but specimens with RBC and WBC counts of  $> 5,000$  &  $50$  cells/mm<sup>3</sup>, respectively, & with bacteriologically positive cultures were excluded. Results were tabulated according to post-conceptual age in weeks (mean  $\pm$  S.D.):

	< 31	31-34	35-38	> 38
#CSF specimens	47	90	55	48
Blood glucose	123 $\pm$ 67	107 $\pm$ 95	87 $\pm$ 51	98 $\pm$ 58
CSF glucose	77 $\pm$ 32	62 $\pm$ 35	60 $\pm$ 25	67 $\pm$ 33
CSF/blood glucose	0.77 $\pm$ 0.46	0.75 $\pm$ 0.38	0.80 $\pm$ 0.34	0.75 $\pm$ 0.29

No significant differences in CSF/blood glucose ratios were noted among the age groups. In infants (n=83) with blood glucose between 40 & 80 mg/dl, mean CSF/blood glucose ratio was 0.81 $\pm$ 0.23. Ratios were higher (1.39) during hypoglycemia & lower (0.61) during hyperglycemia. Thus, CSF/blood glucose ratios below 0.35 ( $\pm$  2 S.D.) concurrent with normal or low blood glucose levels should be considered pathological & warrant further investigation.

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**HYPOLYCORRHACHIA ASSOCIATED WITH INTERCRANIAL HEMORRHAGE (ICH) IN THE NEWBORN.** Robert M. Nelson, Richard L. Bucciarelli, Jon W. Nagel, Ernest F.

Beale, Donald V. Eitzman, University of Florida College of Medicine, Shands Teaching Hospital, Department of Pediatrics, Gainesville.

During 1977 four newborns who developed severe hypoglycorrhachia associated with ICH were seen at the Regional Neonatal Intensive Care Unit of the University of Florida. These infants represented 20% of all infants diagnosed as having ICH in our unit during the past year. CSF cultures were negative in each case. All hemorrhages were confirmed by CAT scan. As noted in the Table the hypoglycorrhachia occurred from 3-14 days after there was clinical evidence of the bleed. In each case the hypoglycorrhachia persisted for weeks. Two babies with frequent taps had 0 mg% CSF glucose for at least three days which was associated with the peak WBC and protein elevation. We feel the decreased CSF glucose in these infants was secondary to the ICH. Similar findings have also been noted in adults after ICH. The association of hypoglycorrhachia and ICH should be taken into account when caring for high risk infants.

	GESTATIONAL BIRTH		INTERVAL FROM BLEED	GLUCOSE	
	AGE	WEIGHT		CSF	BLOOD
CASE 1	26 weeks	860 grams	10 days	0 mg%	90 mg%
CASE 2	40 weeks	4420 grams	8 days	18 mg%	61 mg%
CASE 3	36 weeks	2440 grams	14 days	0 mg%	95 mg%
CASE 4	40 weeks	4000 grams	3 days	24 mg%	90 mg%

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**BRAIN THIAMINE DIPHOSPHATE-ATP PHOSPHOKINASE ACTIVITY** Jerome V. Murphy, Bernard J. D'Souza, The Med. Coll. of WI, Milw. Children's Hosp., Dept. of Neurology, Milw.

That thiamine triphosphate (TTP) has a functional role in the nervous system is indicated by: (1) its deficiency in the brain of patients with Leigh's disease, (2) release of TTP from nerve membrane by pharmacologic or electric stimulation, and (3) the rapid incorporation of <sup>32</sup>P into TTP by brain slices or subcellular nerve particles. Although thiamine triphosphate has been well-characterized, the enzyme which synthesizes TTP in brain has not previously been described.

We have been able to demonstrate TTP synthesis by incubating ATP, thiamine diphosphate, MgCl<sub>2</sub>, TEA (pH 7.4), sucrose and microsomes at 37.5°. Controls (no synthesis) are identical save that the microsomes are heat-denatured (5' at 100°). TTP is separated by electrophoresis on cellulose acetate, and identified by applying purified TTP on an adjacent strip.

Utilizing this method, TTP is synthesized at a linear rate for at least 3 hours. The addition of incremental amounts of microsomal protein is associated with an increased rate of TTP synthesis; i.e. 43.5 nM TTP/mg protein/hr when 0.456 mg of protein is used, and 90 nM/mg protein/hr when 3.08 mg are used. The effect of the inhibitor associated with Leigh's disease on this enzyme(s) will be studied.

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**NON-SURGICAL TREATMENT OF ACQUIRED HYDROCEPHALUS: EVALUATION OF SERIAL LUMBAR PUNCTURE.** L.A. PAPILE, H. KOFFLER, R. BURSTEIN, B. KOOPS. (Sponsored by ROBERT GREENBERG), Department of Pediatrics, UNM School of Medicine, Albuquerque, New Mexico

Serial lumbar puncture (LP) as a method of treating hydrocephalus was evaluated in ten infants of birthweights  $\leq 1500$  grams, who had a cerebral intraventricular hemorrhage (CVH) and ventricular dilatation identified by computed tomography (CT). Each of the infants had an initial CT scan within the first ten days of life. Follow-up CT scans were done at weekly intervals. If progressive ventricular dilatation was observed, daily LP was attempted. Ventricular size remained stable or decreased throughout the tapping period and remained so after the cessation of tapping in eight of the ten infants. Follow-up CT scans at six months of age on eight of the ten infants showed no increase in ventricular size and an increase in the thickness of the cerebral mantle. The two infants from whom cerebrospinal fluid could not be obtained had progressive ventricular dilatation and required the placement of a ventriculo-peritoneal shunt.

This approach to early intervention of progressive ventricular dilatation secondary to CVH by means of serial LP may arrest the development of hydrocephalus and eliminate the need for a shunt.

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**AMNIOTIC FLUID INFECTIONS, HYPERBILIRUBINEMIA & BRAIN DAMAGE.** Richard L. Naeye (Spon. by Nicholas M. Nelson Pennsylvania State Univ., Dept. Pathology, Hershey, Pa.)

The study analyzed acute amniotic fluid infections as a possible cause of brain damage using data from a prospective study of 53,518 pregnancies in which the offspring were followed to eight years of age. Acute inflammation of the subchorionic plate of the placenta, a marker for amniotic fluid infections was associated with increasing neurotoxicity as serum bilirubin levels rose in the neonatal period.

Peak Serum Bilirubin	IQ VALUES <80 AT 4 YEARS OF AGE		
	Infection Absent	Infection Present	
0-8 mg%	11.7	12.7	P > .1
9-12	14.4	17.5	P < .1
13-16	13.1	20.5	P < .02
Over 16	16.1	27.4%	P < .02

The frequency of the retardation increased as the chorionitis became more severe. Significant neurologic abnormalities began at peak neonatal bilirubin levels of 13-16 mg/dl in the infected and at 17 mg/dl in the noninfected. 24 different medical, social and demographic factors that can affect psychomotor development were found not responsible for the impairment associated with the chorionitis and hyperbilirubinemia. The proportion of cases who were infected increased as neonatal bilirubin levels increased so the brain damage may have been due either to the infections or an interaction of the infections with hyperbilirubinemia.

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**CEREBRAL INTRAVENTRICULAR HEMORRHAGE: A MULTIVARIANT MODEL.** L.A. Papile, B. Skipper. (Sponsored by Robert Greenberg), Departments of Pediatrics and Family and Community Medicine, UNM School of Medicine, Albuquerque, New Mexico.

Currently available information on the perinatal and neonatal factors associated with cerebral intraventricular hemorrhage (CVH) in the preterm infant is based only on autopsy studies. In an attempt to identify which factors are associated with CVH in preterm infants, we have developed a multivariate model derived from 100 consecutive infants with birthweights  $\leq 1500$  grams who were admitted to the newborn intensive care unit. All 100 infants had a computed tomography (CT) brain scan performed within the first seven postnatal days to determine which of the infants had CVH.

Fifty perinatal and/or neonatal factors were analyzed. With the use of discriminant analysis, we have been able to determine the six most important factors associated with CVH. These factors are: sex (male), birthweight (900-1200 grams), one minute APGAR score (<4), Idiopathic Respiratory Distress Syndrome (IRDS), multiple gestation, and neonatal transport.

Application of this mathematical model to infants with birthweights  $\leq 1500$  grams gives the probability of CVH in an individual infant and identifies those infants who are at greatest risk for CVH.