●1705 PERINATAL HYPOXIA-ISCHEMIA CAUSES SEVERE BUT REVERSIBLE DEPRESSION OF BRAIN 3-H-DOPAMINE UPTAKE. F. Silverstein, K. Buchanan, and M.V. Johnston. U. of Michigan, Sect. of Pediatric Neurology, Ann Arbor.

Hypoxia-ischemia in brain stimulates catecholamine release by increasing neuronal activity and/or by disrupting presynaptic nerve terminals. Neurotransmitter (NT) uptake by synaptosomes is energy-dependent and requires intact membrane systems. To examine the relationship between NT release and neuronal damage, we compared changes in dopamine (D) turnover and synaptosomal 3H-D uptake in hypoxic-ischemic rat striatum. In 7 day old rat pups, unilateral carotid artery ligation (UCL) & exposure to 2 hours of 8% O2, leads to striatal D depletion and homovanillic acid (HVA) accumulation on the side of UCL: HVA/D= 2.27±.82 vs. HVA/D=.13±.03 on the opposite side (n=20, p<.001, t-test). In synaptosomes prepared from striatum of 22 pups treated in similar fashion,on the side of UCL there was a 76% reduction in 3H-D uptake (.12±.03 pmol/mg tissue vs. .51±.04 in 20 untreated controls, (p<.001, t-test). Uptake was reduced 47% on the opposite side (.27±.05 pmol/mg), similar to values found in pups subjected to hypoxia alone (.31±.06). However, if pups were sacrificed 24 hours after UCL synaptosomes showed normal activity(.53±.04 pmol on side of UCL and .55±.03 on the opposite side). The results suggest that immature D nerve terminals exposed to hypoxia-ischemia are initially disrupted functionally but do recover. Understanding the metabolic processes involved in the initial depression and subsequent recovery is important for devising strategies to protect the brain from hypoxic-ischemic injury.

1706 MENINGITIS: A DEVASTATING COMPLICATION OF SERIAL LUMBAR PUNCTION (LP) THERAPY FOR POSTHEMORRHAGIC HYDROCEPHALUS (PHH). Kathleen M. Smith, Ruth B. Deddish, Edward S. Ogata, Northwestern University Medical Sch. Depts of Pediatrics, OB/Gyn, Chicago, IL.

Serial LP has been advocated as a safe therapy for relieving intracranial pressure in prematures with PHH. During two years, 26 of 122 premature infants with IVH developed PHH. One infant had grade II, 14 grade III (54%), and 11 grade IV (45%) IVH. Serial LP (daily or qod) was initiated for progressive ventriculomegaly. Twenty-two infants underwent 3-33 LP while 4 infants required immediate neurosurgery. Five of the 22 infants (23%) developed meningitis directly related to LP. This far exceeds the incidence of meningitis in our infants without PHH. Organisms were Staphylococcus epidermitis (2), Enterobacter aerogenes, Klebsiella pneumoniae, and Candida albicans. We could not identify specific risk factors responsible for LP associated meningitis. Meningitis and noninfected groups were of similar birthweight (1088 vs 1200 g), GA (29 wks), and had similar clinical problems. The use of central venous catheters and the concurrent administration of antibiotics was similar. The number of LP and the incidence of difficult (multiple attempts 12-15%) and traumatic (3-7%) LP did not differ. One of the 5 infants with meningitis died while the other 4 required neurosurgery for progressing PHH. Of the 16 noninfected infants, 4 ultimately required neurosurgery and 1 died of complications of this procedure. LP meningitis greatly worsened PHH outcome. The risk of LP therapy must be carefully considered before treating PHH.

1707 PERIVENTRICULAR LEUKOMALACIA: INCIDENCE AND SIGNIFICANCE IN PREMATURE INFANTS. Yolande F. Smith, Michal A. Young, Anita M. Sostek and Edward G. Grant(Spon. by Philip L. Calcagno). Department of Pediatrics, Georgetown University Medical Center, Washington, D.C.

Portable cranial sonography was done on 389 infants with birth weights of 1750g or less admitted to Georgetown University Hospital's Intensive Care Nursery during the 3-year period May 1981 to April 1984. Periventricular leukomalacia(PVL) was diagnosed in 28 infants (7.2%) with birthweights of 680-1730g with appropriate gestational ages 26-33 weeks. The lesions were bilateral in 89% and widespread along the ventricular borders in 86%. Survival in the PVL group was 75%, statistically not significant from 83% on infants without PVL.

Twenty of the 21 PVL survivors have been evaluated quarterly using the Bayley Scales of Motor (PDI) and Mental(MDI) Development and standard neurological criteria. All infants demonstrated generalized hypertonia/spasticity on follow-up to 6-35 months (mean 17.6 months). Ninety percent had severe motor deficits, and 75% had significant delay in mental scores (MDI). Cortical blindness was present in 25%. The infants with significantly abnormal MDI and PDI (scores<50) all had bilateral widespread PVL, in contrast to the infants within the normal range who demonstrated focal and/or unilateral lesions.

Conclusions: 1)PVL occurs in 7.2% of premature infants weighing 1750g or less at birth; 2) significant neuro-developmental handicaps are present in infants with bilateral widespread PVL; and 3) infants with focal and/or unilateral lesions have a better chance for reasonable developmental outcome.

†1708 NEUROLOGICAL DISEASE IN INFANTS WITH AIDS. Madhav Suri, Shaleish Asaiker, Asha Gupta, Johanna Goldfarb, Aditya Kaul and Ram Kairam. New York Medical College, Westchester County Medical Center, Department of Pediatrics, Valhalla, NY 10595

We followed serially the neurological examination of three infants with AIDS. All three children, first seen 6-8 months of age came from families with high risk for AIDS: Haitian (1/3) and I.V. drug user (2/3), all had failure to thrive, hepatosplenomegaly, lymphadenopathy and recurrent infections.

All exhibited a marked arrest of development, progressive corticospinal tract signs and impaired brain growth (microcephaly). Developmental delay was most striking in the areas of language acquisition and gross motor skills while fine motor skills were relatively intact. The corticospinal tract signs were progressive, presenting as ankle clonus and increased deep tendon reflexes and progressing to spastic rigidity. One child had basal ganglion calcification on CT scan. One child died of pneumococcal meningitis at 13 months of age.

Repeated cerebral spinal fluid cultures for bacterial, fungal and viral pathogens were negative.

We postulate that the neurological disease in infants with AIDS is caused by intrauterine infection with an agent that interrupts neurological maturation during the vulnerable period. Search for HTLV-III in neural tissue or spinal fluid may help elucidate the etiology.

1709 COMPUTERIZED CRANIAL TOMOGRAPHY IN PATIENTS WITH SOTOS' SYNDROME. Roberto Talamantes, Geraldine W. Wilson, Alan K. Percy, William D. Williamson, Frank Greenberg. Baylor College of Medicine, Texas Children's Hospital, Department of Pediatrics, Houston.

Detailed assessment of patients with Sotos' Syndrome using current neuroradiographic procedures has not been widely reported. Twenty-one patients meeting the characteristics of this syndrome (Sotos, J. AJDC, Vol. 131,1977) were studied. Seventeen had computerized cranial tomography, of which five were normal. Of the remaining twelve, nine had ventricular dilatation, two had cerebral atrophy, and two had agenesis of the corpus callosum. In eight of the patients the dilatation was mild, but one had significant dilatation requiring a V.P. shunt.

Several clinical findings not described previously were noted: two patients had atrial septal defects, three had tracheomalacia, and two had strabismus. In addition one had hypothyroidism, a finding previously described.

This is the first study to present the findings of computerized cranial tomography from a large number of patients with Sotos' Syndrome. The role of these findings in the pathogenesis is unclear, but the significant incidence of ventricular dilatation, cerebral atrophy, and agenesis of the corpus callosum makes it evident that C.T. scan of the head is an important part of the initial evaluation of patients with Sotos' Syndrome. Consideration must also be given to concurrent or associated disorders, such as cardiac anomalies, tracheomalacia, strabismus and hypothyroidism.

1710 RAPID CORRECTION OF CHRONIC HYPONATREMIA (CHR HYPO-Na^+) ELEVATES DEPRESSED BRAIN (Br) AMINO ACID LEVELS [AA]) IN MICE: POSSIBLE RELATION TO CENTRAL PONTINE MYELINOLYSIS (CPM) Jean Holowach Thurston, Richard E. Hauhart. Washington U., Dept. of Ped.; Children's Hospital, St. Louis.

Effects of Chr Hypo-Na^+ or rapid correction of Chr Hypo-Na^+ on Br metabolism are unknown. Acute Hypo-Na^+ (4h) reduced Br [AA] in mice (J Neurochem 24:953 (1975)). If similar findings occur in Chr Hypo-Na^+, elevation of plasma (Pl) [Na^+] might elevate Br [AA] (J Neurochem 40:240 (1983)). To test the hypothesis 20-d-old mice were made Hypo-Na^+ (4-d Pitressin + 2.5% dextrose); then treated with 1 M and 0.9% NaCl for 9h. Pl [Na^+] (meq/l) and Br [AA] (mmol/kg) (mean ± SE) are given below.

Measurement	Control(N=3)	Chr Hypo-Na^+(N=5)	Rapid Na^+ Rx (N=5)
Pl Na^+	145 ± 1	104 ± 4	139 ± 3
Br glutamate	8.19 ± 0.11	5.22 ± 0.47	9.38 ± 0.17
Br aspartate	2.56 ± 0.17	1.31 ± 0.02	3.68 ± 0.23
Br glycine	1.57 ± 0.13	0.71 ± 0.04	1.15 ± 0.03
Br GABA	2.03 ± 0.06	1.56 ± 0.07	1.87 ± 0.08
Br taurine	14.78 ± 0.66	3.48 ± 0.87	5.20 ± 0.49

Chr Hypo-Na^+ decreased Br [AA] 23% to 74% ($P<0.005$). Rapid elevation of Pl [Na^+] increased Br glutamate and aspartate levels above normal (14% and 44%, respectively (resp.), $P<0.014$); glycine and taurine levels were still reduced (27% and 65%, resp., $P<0.006$). Osmotic disequilibrium or injury to endothelial cells could produce Br edema. Increased levels of glutamate and aspartate (neuroexcitatory) and decreased levels of taurine and glycine (neuroinhibitory) could relate to the hyperactivity and/or seizures and the neuropathology seen in experimental CPM.