1660 OUTCOME IN CHILDREN WITH PRENATALLY DIAGNOSED CONGENITAL HYDROCEPHALUS. Charles, C. Duncan, Frank A. Chervenak, Laura R. Man, David T. Scott, Richard A.Ehrenkranz (Spon, by A. Pearson) Yale Univ Sch Med, Dept of Neurosurg, between the second structure of the second structure of the second prosed congenital hydrocephalus, defined as excessively increasing ventricular size and biparietal diameter on at least two sequential examinations, were available for outcome evaluation. Others in whom this diagnosis was made either did not survive gestation or the init-in neonatal period due to interruption of the preg-nancy or intrapartum demise. GA at diagnosis varied from 26 to 38 wks with a mean of 31 wks. Age at delivery varied from 32 to 40 wks with a mean of 36 wks. Four of these children had myelomeningocoele, 6 had hydrocephalus without other anomalies, three had intracranial cysts, and one had an encephalocoele. All of them underwent ventriculo-peritoneal shunting. Five had shunt related infections. Six had Bayley mental or Stanford-Binet scores >80 (43%), 2 had scores between 65 and 80 (14%), and 6 had scores <65 (43%). The poorer outcome of this group in com-parison to other series of such hydrocephalus may relate both to the early stage at which the diagnosis was made such that children with more severe con-ditions have survived and the high rate of infection. OUTCOME IN CHILDREN WITH PRENATALLY

•1670 BIOGENIC AMINE METABOLISM IN RETT'S SYNDROME, Huda Y. EI-Hibri, Alan K. Percy, Ian J. Butler, Baylor College of Medicine, Texas Children's Hospital, Dept. Pediatrics & Neurology, Houston, Texas. Rett's Syndrome is a progressive disorder in females charac-trized by autistic behavior, dementia, ataxia, loss of purpose-ful use of the hands and seizures. Extensive laboratory investi-gations are usually normal with the exception of electroenceph-alography. To date no biochemical abnormalities have been reported. In an attempt to find a lead to the pathogenesis of this syndrome we assayed biogenic amine metabolites in the cerebrospinal fluid (CSF) of six patients, age 2-15 years, with Rett's Syndrome. The following metabolites were assayed and compared to age matched controls: 3-methoxy-4-hydroxyphenyl-ethyleneglycol (MHPG), homovanillic acid (HVA) and 5-hydroxy-indole acetic acid (5-HIAA). Results are as follows: Mean ± s.D. (ng/ml).

	MHPG	HVA	5-HIAA
Patients	7.81±1.49	64.70±26.5	23.83±6.25
Controls	12.35±0.64	110.23±25.83	32.07±8.26
p-value	0.0005	0.0008	0.004

Thus, norepinephrine, dopamine, and serotonin metabolites are significantly reduced in the CSF of patients with Rett's Syn-drome. This may provide new insight into the pathogenesis and future therapeutic intervention in these patients.

1671 DIAGNOSIS OF CEREBRAL ATROPHY IN INFANTS BY THE NEARby <u>Asma Q. Fischer</u>, Medical College of Georgia, Dept. of Neurology, Augusta, Georgia Cerebral atrophy in infants has been difficult to assess by cranial

sonogram (CS) because the subarachnoid space has been a "blind spot" by ultrasound, yet CS is an ideal imaging method for infants spot as it is portable, non-radiative and unlike computed cranial tomography (CCT), requires no intravenous contrast or sedation. We present a new method of evaluating the subarachnoid space by CS in infants and correlate it with the presence of cerebral atrophy. Material & Methods: Realtime ultrasound equipment with a modified transducer was used for imaging the "nearfield". The nearfield was defined as the first 2-3 cm from the scalp into the brain. The subarachnoid space which can be detected within the nearfield was assessed in 25 consecutive infants who were sonogrammed for various neurologic indications. The results were correlated with CCT findings of cerebral atrophy. Results: On nearfield sonography, 3 of the 25 patients had enlargement of the subarachnoid space. All 3 patients had cerebral atrophy on CCT. 22 of the 25 patients had no enlargement of the subarachnoid space by nearfield sonography. None of the infants who had CCT showed evidence of cerebral atro-phy. Enlargement of the subarachnoid space by nearfield sonography was therefore found to be most sensitive and specific for cerebral atrophy when compared to CCT. Summary: Nearfield sonography provides a simple tool for assessing the subarachnoid space and there-fore the presence of cerebral atrophy. It opens the way for a non-invasive method of evaluating the degree of cerebral atrophy and its correlation with the clinical state and prognosis. Larger series using this technique seems appropriate.

NEONATAL NEUROSONOGRAPHIC PREDICTORS OF CEREBRAL †**1672** PALSY IN PRETERM INFANTS.

 IU/2 FALST IN FREIER INFANIS.
 L. Graziani, M. Pasto, C. Stanley, F. Pidcock,
 H. Desai, S. Desai, B. Goldberg, Thomas Jefferson University Hospital, Departments of Pediatrics and Radiology, Philadelphia, PA. In a prospective study, preterm infants of less than 33 weeks gestation were routinely evaluated by cranial ultrasonography at frequent intervals during their neonatal course. Thirteen survigers form of correlated paler at 12 to the second vors were found to have a spastic form of cerebral palsy at 12 to 36 months of age; two of the infants had a hemiparesis, 1 a mono-paresis and 10 a diparesis. During the neonatal course of each paresis and 10 a diparesis. During the neonatal course of each handicapped infant, either or both of the following neurosono-graphic abnormalities were present: 1) frontally located peri-ventricular porencephaly secondary to a grade IV intracranial hemorrhage (ICH), 2) extensive periventricular echodensity with subsequent cyst formation extending frontally. Ultrasound abnor-malities consisting of ICH, or periventricular echodensity but malliles consisting of 100, or periventricular echodensity but without frontal porencephaly or persisting periventricular cysts were noted during the neonatal course of 49 other preterm infants who did not have cerebral palsy at age 12 to 36 months. The pathogenesis of the periventricular echodensities and subsequent pathogenesis of the periventricular echodensities and subsequent cyst formation is unknown but the ultrasound studies are consis-tent with hemorrhagic infarction and ischemic lesions involving pyramidal tracts as they enter the internal capsule. We conclude that spastic forms of cerebral palsy are likely to develop in preterm infants if neonatal ultrasound studies demonstrate persisting destructive lesions in frontally located periventicular heat structures.

periventricular brain structures.

•1673		VIVO ABOLISM		TION TH	OF THER	BRAI	N C IC	LUMBAR
Younkin, E	Eileen	Donlon,	Britt	ton Ch	ance,	and	Maria	Delivoria-
Papadopoulos Physiology, B	ioche	<ol> <li>of PA.</li> </ol>	. School	of Me	edicine, Phila., P	, Dep	ts. of	Pediatrics,

Sequential cerebral spectra of phosphorus-containing compounds, ATP, phosphocreatine (Pcr), inorganic phosphorus-containing compounds, ATP, phosphocreatine (Pcr), inorganic phosphorus-containing compounds, ATP, phosphocreatine (Pcr), inorganic phosphate (Pi), phosphodiester (PD), and phosphomonoester (PME), by 31-P nuclear magnetic resonance (NMR) were obtained in a severely asphysiated premature infant with hydrocephalus and seizures. Between days 2-20, 10 therapeutic LP's were done and NMR cerebral spectra were obtained pre (a) and post (p) LP. Pcr/Pi (normal  $\bar{x} = 1.01$ ), a measure of bioenergetic reserve, was low at 48h; Left (L) = 0.50, Right (R) = 0.60. After the initial LP, Pcr/Pi rose to L=1.4 and R=0.6. After subsequent LP's, a sharp decrease in Pcr/Pi was followed by a rebound (a LP, L=1.50, R=1.03; p LP, L=0.87, R=0.62; 1½ hrs p LP, L=1.52, R=1.66). Pcr/Pi increased by day 8 (L=1.52, R=1.60) then stabilized during the next month (L=1.0, R=0.8). Tissue pH did not change significantly (day 5: a LP=7.07, p LP=7.02, 1½ hrs p LP=7.08; day 8: a LP=7.12, p LP=7.02, 1½ hrs p LP=1.12). PME/ $\beta$ -ATP, a precursor in membrane biosynthesis, declined (day 2, L=3.15, R=2.53; day 46, L=1.46, R=1.17) but changed inconsistently post LP. Acute changes in Pcr/Pi and PME/ $\beta$ -ATP represent changes in local cerebral Sequential cerebral spectra of phosphorus-containing compounds, in Pcr/Pi and PME/ $\beta$ -ATP represent changes in local cerebral metabolism. Removal of 5 cc of CSF may result in acute intracerebral tissue shifts, acute hydrostatic changes with probable effects on CSF and cerebral blood flow, and hence provision of metabolic substrate to brain tissue. These data suggest that acute transient changes in Pcr/Pi and  $\mathsf{PME}/\beta$  -ATP, representing changes in local cerebral metabolism, reflect general changes in cerebral metabolic or seizure activity.

CREBRAL INFARCTION IN UTERO PRESENTING AS NEONATAL SEIZURES Erik A Hagen (Spon by Willa H. Drummond) Univ of Florida College of Medicine, Gainesville Nonatal cerebral infarction (Cl) is usually associated with risk factors such as birth trauma, asphyxia or coagulopathy, but risk factors. We have seen 2 cases of left middle cerebral arge in infants with "moderate" risk factors (meconium staining in case #1; variable/late decelerations, vacuum extraction, nuchal ord x l in case #2). In both infants, CT scans within 24 hours of age showed Cl in the left MCA distribution, and, in both cases, these changes appeared to be subacute (i.e: infarction atrophy, ipsilateral optic nerve atrophy and absent nasal retinal atrophy, ipsilateral optic nerve atrophy and absent nasal retinal atrophy, spasiticity and decreasing head growth. In case #2, the neurologic residua. On CT there is minimal residual evidence of Cl with "islands" of normal brain tissue in the previous area of capehalomalacia. This infant is seizure free off medication. These cases are similar in presentation but remarkably different in outcome. CI can result in devastating permanent sequiae, but outcome may also be entirely normal. Prognosis in CI may be more occurance of the lesion itself.