1600 DEVELOPMENT OF NORMS FOR MEASUREMENT OF INTRACRANIAL STRUCTURES IN HIGH-RISK INFANTS: PRELIMINARY FINDINGS.

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25 real-time ultrasonic sector scans of the head were obtained through anterior fontanelles of high-risk infants who were judged to have no intracranial pathology by clinical and usual sonographic criteria. Thicknesses of frontal mantle (FR.M) and occipital mantle (OC.M) were measured in sagittal planes. Distances between the inner tables of the calvaria were measured in coronal sections at the level of the caudate nuclei (BC) and of the atria of the lateral ventricles (BA). Distances between outer walls of lateral ventricles were measured at the same levels (VC and VA). We recorded birthweight, present weight, length of gestation, age post-conception and head circumference at each examination.

Infants in this study ranged from 29-48 weeks post-conception. They weighed 800 to 3140 g. All four measures of brain size or thickness (OC.M, FR.M, BA, BC) increased significantly with age and with body weight ($P<0.01$ in 6/8 and <0.05 in 2/8). VA also increased significantly with age and weight but not so with VC.

All of the following ratios remained stable as weight and age increased: VC/BC=0.31±0.07 (m±S.D.); VA/BA=0.54±0.09; OC.M/FR.M =0.86±0.13. The ratios were normally distributed in the population.

We are expanding this series in search of quantitative measures of normal brain structures that may be used to identify ventricular enlargement early in high-risk infants.

1601 NEUROPHYSIOLOGICAL, PSYCHOLOGICAL, AND NUTRITIONAL INVESTIGATIONS DURING DISCONTINUATION OF THE PHENYLALANINE-RESTRICTED DIET IN CHILDREN WITH CLASSICAL PHENYLKETONURIA (PKU).

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Children with classical PKU had been maintained on a low-phenylalanine diet from the first few weeks of life until their 5th birthday when the phenylalanine-restricted diet was stepwise changed to a regular diet. Physical and neurological examinations, nutritional evaluations, anthropometric measurements, and psychological assessments were done at 4, 4½, 5, and 6 years of age. In addition, neurophysiological investigations including somatosensory, auditory and visual evoked responses were obtained at 4 years 10 months, 5 years, 5 years 2 months, and 5 years 6 months.

Analysis of relationships among the study variables revealed that children who had adequate dietary control and who were of average or above average intelligence displayed normal somatosensory evoked potentials, whereas those children with less adequate dietary control and lower IQ scores had atypical somatosensory evoked potential waveforms. Yet, these multiple assessments did not uncover any significant changes when pre-diet termination data were compared with post-diet termination results.

1602 ASSOCIATION OF HYPERTENSION AND FLUIDS WITH CLINICAL MANIFESTATION OF SEVERE INTRACRANIAL HEMORRHAGE(ICH) IN VERY LOW BIRTH WEIGHT INFANTS:

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Fluid overload in VLBW infants and induced hypertension in animals have been implicated in the etiology of ICH. We compared 51 clinical and biochemical variables from 34 VLBW infants with autopsy proved severe ICH, and 38 randomly selected control infants (C). No differences were found between the groups in:4 prenatal stress factors, mode of presentation and delivery, admission blood gases and diagnosis. ICH group had 2/3 symptoms of severe ICH by 29.8±20 (SD) hours. C infants had no symptoms, normal CSF and are developing normally. B.Wt and G.A. were, ICH:0.93kg and 28Wks: C group 1.2kg and 30 Wks ($p<0.05$). Other results, ($\bar{x}$± SE values) are in the Table. ICH infants were smaller, 1.5 Wks less mature, more

	Apgar 1'	Apgar 5'	Adm Temp.	BP Adm.	BP(Torr) Day 1	BP(Torr) Day 2	Fluids ml/kg/d Day 1	Fluids ml/kg/d Day 2
ICH	3.7±0.4 *	5.3±0.5 *	94.6±2 *	35.1±1.8	36.5±5.2	52±5 *	115±10	149±10 *
C	5.2±0.3	7.1±3.5	96.6±1.4	40±2.2	37.9±1.6	40±2	100±6	120±7

asphyxiated and colder on admission. (*$p<.05$) They received slightly more fluid (N.S) on day 1. On day 2 however, they became more symptomatic, had elevated mean BP, and had received about 30ml/Kg. more fluids (*$p<.05$). We feel that in VLBW infants, asphyxia sets up a stage for ICH. Vigorous resuscitation, and rapid volume expansion trigger a hypertensive response precipitating the final episode of massive ICH.

1603 CEREBRAL PERFUSION PRESSURE(CPP) IN BIRTH ASPHYXIA(BA) AND ITS RELATION TO THE QUALITY OF OUTCOME.

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In 10 normal infants and 14 BA infants ICP changes were studied by a noninvasive technique (Iadd Monitor). Systemic BP, mean ICP and CPP (Mean BP-ICP) were calculated during acute phase (3 days) and recovery (6 days) in BA infants. 4 infants died and had evidence of brain damage and 2 of the 10 survivors had cerebral palsy (CP) at 3 years. 8 remaining survivors were normal (26-46 mos) Clinical, ICP and CPP values from normals. (Gr.I) BA with normal long term outcome (Gr.II), BA with brain damage (Gr.III) are shown below: (values mean + SE) *$p<0.05$, **$p<0.025$, + $p<0.001$. CPP during acute phase

	B.Wt.	GA	Apgar 5 Min.	Ad.pH	ICP Torr	Admin. BP Torr	Least CPP Torr
Gr.I n=2	3.3±0.5	39±0.1	9±1		8.2±0.8	51±1.8	42±1.7
Gr.II n=8	2.9±0.3	38± .3	4.4±0.8	7.18±0.05	13.5±1.5 *	44±2.5 *	31.3±2.2 **
Gr.III n=6	3.4±0.5	39±1.5	3±1	7.06±0.04	22.8±4.7 **	38.3±2.2 **	13.5±1.5 +

was low in Gr.II and improved during recovery. In Gr.III profound falls of CPP were due to elevation of ICP (cerebral edema) and fall of BP. In 12 infants who had accurate CPP measurements, 6 had CPP <30 Torr (mean-3 SD). 5 of these had brain damage and the other has amblyopia. None of 6 with >30 Torr CPP had brain damage. We conclude: A) Very low CPP in BA infants is associated with brain damage. ($p=0.015$, Fishers Ex.Test), B) CPP should be utilized in the management and to prognosticate the outcome.

1604 PROSTAGLANDIN LEVELS IN THE CEREBROSPINAL FLUID OF INFANTS AND CHILDREN WITH FEVER.

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It has been proposed that central nervous system (CNS) prostaglandins (PG) may be mediators of fever. In some animal models, cerebrospinal fluid (CSF) PGE is elevated in pyrogen-induced fever; and the intraventricular injection of PGE results in a febrile response. To investigate the role of CNS Prostaglandins in fever, PGE and $PGF_{2\alpha}$ concentrations were measured by radioimmunoassay in the CSF of 16 pediatric patients who were febrile (average temp. 39.0°C). They were free of CNS disease and were on no prostaglandin synthetase inhibiting drugs. Their values were compared to those of 19 patients who were neither febrile or on prostaglandin synthetase inhibitors. The following results (mean ± SEM) were obtained:

	Febrile	Afebrile
PGE (pg/ml)	47 ± 15	39 ± 19
$PGF_{2\alpha}$ (pg/ml)	169 ± 26	133 ± 37

There were no significant differences in the CSF levels of either PGE or $PGF_{2\alpha}$ between the febrile and the afebrile groups. These observations do not support the hypothesis that CNS prostaglandins mediate fever.

1605 PROSTAGLANDIN LEVELS IN THE CEREBROSPINAL FLUID (CSF) OF CHILDREN WITH MENINGITIS.

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Prostaglandins (PG) are synthesized in the Central Nervous System (CNS) during inflammatory processes. In order to study the relationship between meningitis and CNS PG production, PGE and $PGF_{2\alpha}$ concentrations were measured by radioimmunoassay in the initial CSF sample (day 1) of 7 children with bacterial and 4 with aseptic meningitis. Follow-up measurements were also obtained at 3 and 12 days in the bacterial meningitis group. The following results (mean ± SEM) were obtained.

	Aseptic Meningitis (Day 1)	Bacterial Meningitis (Day 1)	(Day 3)	(Day 12)
PGE (pg/ml)	173 ± 41	1683 ± 229	257 ± 116	80 ± 37
$PGF_{2\alpha}$ (pg/ml)	473 ± 145	1333 ± 232	530 ± 160	125 ± 33

The children with bacterial meningitis had significantly higher levels of both PGE ($p < 0.001$) and $F_{2\alpha}$ ($p < 0.03$) than did those with aseptic meningitis. Moreover, the PG levels paralleled the clinical course of the bacterial meningitis. In one patient (not included in table) who failed to respond to therapy, the initially high levels (pg/ml) of PGE (1950) and $PGF_{2\alpha}$ (2100) persisted at 3 days (PGE 2250, $PGF_{2\alpha}$ 2200) and 12 days (PGE 2300, $PGF_{2\alpha}$ 2480).

Thus, CSF PG measurements may be helpful in distinguishing between bacterial and aseptic meningitis, and may be useful in monitoring the clinical course of bacterial meningitis.