1558 EFFECT OF ELEVATED INTRACRANIAL PRESSURE ON DISTRIBUTION OF CARDIAC OUTPUT IN YOUNG LAMBS. Joanne E. Backofen, Raymond C. Koehler, M. Douglas Jones, Jr., Mark C. Rogers and Richard J. Traystman. Johns Hopkins Med. Inst., Johns Hopkins Hospital, Depts. of Anesthesiology/Critical Care Medicine and Pediatrics, Baltimore, MD 21205

We tested the hypothesis that elevation of intracranial pressure (ICP) produces redistribution of cardiac output. Nine lambs (3 months old) were anesthetized with pentobarbital and pancuronium, and ventilated. ICP was increased by infusing mock CSF into the right lateral ventricle. Regional blood flow was measured with the radiolabelled microsphere technique at two control levels separated by 1.5 hr, and at two different elevations of ICP, each maintained for 10 minutes. Elevation of ICP to 53±5 (SE) mmHg at a mean arterial pressure (MABP) of 102±6 mmHg (cerebral perfusion pressure (CPP)=50±3) resulted in a 7±3% decline of cerebral blood flow (CBF). At this level of ICP, there was no reproducible pattern of change in regional blood flow to intestines, kidney, spleen, liver, pancreas, adrenals, skin or skeletal muscle. Severe elevation of ICP to 93±4 mmHg, produced an increase in MABP from 108±6 to 118±7 mmHg, and a 30±6% fall in CBF (CPP = 24±5). Hepatic arterial flow decreased by 35±14%. Skeletal muscle flow increased by 85±34%. There was no consistent blood flow change in other organs studied. However, in lambs in which cerebral O_2 consumption fell by more than 10% (n=4), renal and skin flow decreased by 14 ±4% and 16±1%, respectively. These data indicate that severe elevations of ICP sufficient to cause cerebral ischemia result in differential distribution of cardiac output.

1559 DEPAKENE (VPA) INDUCED HYPERAMMONEMIA (HA) Mark L. Batshaw, David Valle, and Saul W. Brusilow, Johns Hopkins Med, Inst., Balto., MD.

A 16 year old brain damaged girl with 10% activity of carbamyl phosphate synthetase (CPS) maintained NH4 levels of 20-55uM, on ketoacid therapy for 4 years. VPA (20 mg/kg/d) was then started to control akinetic seizures. Plasma NH4 rose from 30 to 226μM in one week. VPA was stopped and reinstituted on 3 other occasions with a similar HA response. During VPA therapy SGOT was 13-27 IU/1, SGPT 9-24 IU/1, bil. 0.4mg/d1. Plasma citrulline fell from 15 ± 1 to 9 ± 1μM (SEM) $p < .005$ on VPA. Plasma glycine remained normal. Plasma NH4 was also measured in 28 epileptic children on VPA and in 29 patients on other anticonvulsants. Plasma NH4 was higher in the VPA group, 33.6 ± 1.9 vs 23.6 ± 1.5, $p < .001$. Mean plasma VPA level was 11.5 mg/dl, SGOT 23 and SGPT 13. There was no correlation between plasma concentrations of NH4 and VPA. Two adults and one child received VPA (20mg/kg/d) for a period of one week proceeded and followed by 1 week control periods. Plasma NH4 levels before, during and after VPA were: 32.3±3.7, 50.4±4.7, 26.9±3.9 $p < .001$. Plasma VPA levels were therapeutic and SGOT 18. Thus, VPA therapy resulted in a modest but significant increase in plasma NH4 without evidence of liver damage. It caused symptomatic HA in a patient with impaired waste nitrogen excretion. VPA induced HA may be a result of inhibition of activity of CPS or acetylglutamate synthetase as is thought to occur in certain organic acidemias. These data suggest that NH4+ levels should be monitored in patients receiving VPA.

● **1560** BRAINSTEM AUDITORY EVOKED RESPONSES (BAER) IN INFANTS WITH CONGENITAL ALVEOLAR HYPOVENTILATION SYNDROME (CAHS). R. Beckerman, J. Meltzer, D. Dunn, A. Sola, M. Ellis, (Spon. by J. Lewy), Departments of Pediatrics and Otolaryngology, Tulane Univ. School of Medicine and Newborn Division, Baptist Hospital, New Orleans, Louisiana.

BAER may be useful in localizing nonstructural defects in the brainstem. Two infants with CAHS were evaluated with tests of ventilation {$\dot{V}E$ awake(AW)/$PaCO_2$, $\dot{V}E$ asleep(AS)/$PaCO_2$}, routine neurological studies and BAER in order to assess the correlation between BAER and loss of central control of ventilation. BAER were also measured in normal infants (CON) and in an infant with prolonged sleep apnea (PSA) but without alveolar hypoventilation.

Dx	Age (Mos)	Neurological Sx	Cat Scan	$\dot{V}E(AW)$*** / $PaCO_2$	$\dot{V}E(AS)$*** / $PaCO_2$	BAER
CAHS	15	Hypotonia +Gag	Mild hydroceph.	202* / 48	54** / 60	Abn. waves I→III
CAHS	3	Hypotonia +Gag	WNL	153** / 41	51** / 61	Abn. waves I→III
PSA	18	None	WNL	---	245* / 39	Abn. waves I→III

*Normal $\dot{V}E$ **Abn. $\dot{V}E$ ***$\dot{V}E$ in ml/min/kg $PaCO_2$=end tidal CO_2

All BAER values in the CON subjects were normal. All three patients have exhibited depressed ventilatory response to CO_2 during quiet sleep and prolongation of intrapeak latencies between waves I→III suggesting significant conduction delays in the lower brainstem. These studies suggest that BAER may be helpful in localizing defects in brainstem function implied by loss of metabolic control of ventilation during sleep.

1561 PSYCHIATRIC SEQUELAE OF LOCALIZED CORTICAL INJURY IN CHILDREN. Polly E. Bijur, David Shaffer, Oliver D. Chadwick, (Spon. by David Rush). Columbia Univ. and N.Y. State Psychiatric Institute. Department of Child Psychiatry, New York.

To test the hypothesis that localized cortical damage is associated with specific forms of psychiatric disorder, 54 children with unilateral compound depressed head fractures hospitalized in Great Britain were examined and their parents and teachers interviewed. Children were categorized into four groups by site of dural tear as determined from surgical records. Psychiatric disorder was measured by four symptom scales derived from psychiatrist, parents' and teachers' questionnaires.

There were no significant associations between hyperactivity, conduct disorder and other emotional disorders and site of injury or hemisphere. Children with right frontal and left posterior injuries had significantly more severe depressive symptoms than those injured at other sites ($F=3.65$ $p=.02$). The contrast between the right frontal-leftposterior and left frontal-right posterior groups remained significant after the effects of social disadvantage, age at injury, age at examination and parental history of psychiatric disorder were controlled in a multiple regression analysis.

The general lack of association between locus of injury and form of psychiatric disorder suggests that the effect of localized damage is not marked. The depression results, however, are supported by neurophysiological findings and indicate that damage to a specific site can result in a specific type of disorder.

1562 A COMPARISON OF PRETERM AND FULL-TERM INFANTS USING THE PRECHTL NEUROLOGICAL EXAMINATION. M. Jayne Brennan and Charles R. Bauer (Spon. by E.Bancalari), Univ. of Miami, Dept. of Peds., Miami, Florida

The Prechtl Neurological Examination of the Newborn was administered to 50 infants at approximately 40 weeks (± 1 week) gestational age. These infants were randomly selected from the normal nursery and special care units. Twenty five were healthy, full-term infants ($\bar{x}$ B.W. = 2740 grams) ($\bar{x}$ G.A. = 39.4 weeks). The remaining 25 infants were born prematurely with multiple perinatal complications including respiratory distress, hyperbilirubinemia, apnea etc. ($\bar{x}$ B.W. = 1652 grams) ($\bar{x}$ G.A. = 33.5 weeks). The length of hospitalization in this group varied ($\bar{x}$ = 30 days). None of these infants suffered from severe central nervous system abnormalities, chromosomal anomalies, nor major metabolic disturbances. The Prechtl Examination was performed by the same examiner in all infants at approximately the same time post-feeding. Infants were in the quiet-alert state at the initiation of the examination. A scoring system based on the 55 items comprising the Prechtl Exam was established. A student t test for independent samples was performed on the total score received by each infant. The preterm infants were found to have a significantly better overall performance on this assessment than did the full-term infants ($p < .05$). These results suggest that sick premature infants have accelerated maturation which may result from intense stimulation they received during a extended stay in a Special Care Nursery. The long-term predictability of this examination has yet to be determined.

1563 CEREBRAL AUTOREGULATION IN NEONATAL PUPPIES. D.J. Camp, U. Kotagal, L.I. Kleinman, U. Cincinnati College of Medicine, Department of Pediatrics

In an attempt to document autoregulation of cerebral blood flow in the hyperoxic and hypoxic newborn animal, cerebral blood flow was measured in 6 hyperoxic (HE) and 4 hypoxic (H) neonatal puppies aged 2-13 days using the radioactive microsphere reference organ technique. In the HE group arterial oxygen tension was maintained above 250 torr with 100% O_2 throughout all periods. In the H group arterial oxygen tension was maintained at 47.5 torr ± 7.6 SEM by continuous administration of 12% O_2. Following control measurements (period I), puppies were made hypotensive (period II) by withdrawing 15-25 ml/kg of blood in the HE group and 20-32 ml/kg in the H group. In the HE group during period II, mean blood pressure fell 20.5% ± 2.8 (p=0.016) and cardiac output decreased 20.9% ± 5.7 (p = 0.016) while cerebral blood flow was unchanged (31.37 ± 3.76 and 32.69 ± 5.90 ml/100g/min in periods I and II respectively). When hypoxic animals were made hypotensive, mean blood pressure fell 18.6% ± 4.7 and cardiac output decreased by 47.9% ± 10.9 while cerebral blood flow was unchanged (38.66 ± 4.64 and 45.2 ± 4.00 ml/100g/min in period I and II respectively). The fraction of cardiac output to the cerebrum increased from 2.8% ± 0.4 to 3.8% ± 0.8 in the HE animals ($p < 0.05$) and from 2.5% ± 0.5 to 6.3% ± 1.1 in the H group ($p < 0.05$) during hypotension. Thus, in the presence of decreased mean blood pressure and cardiac output in both hyperoxic and hypoxic neonatal puppies, cerebral blood flow is maintained, demonstrating the presence of cerebral autoregulation.