†1717 THE ROLE OF CARDIOPULMONARY RECEPTORS IN NEURAL LOBE AUTOREGULATION. D.A. Wilson, D.F. Hanley and R.J. Traystman (Spon. by: Mark C. Rogers). The Johns Hopkins Medical Institutions, The Johns Hopkins Hospital, Department of Anesthesiology/Critical Care Medicine and Neurology, Baltimore, MD 21205

Cerebral autoregulation (AR) is not influenced by baroreceptor activity. However, neural lobe blood flow (NLQ) may be regulated differently. In this study we examine the relationships between arterial pressure (AP), NLQ and arginine vasopressin (AVP) after bilateral vagotomy (V). Regional cerebral blood flow (rCBF) was measured by the microsphere method in 4 pentobarbital anesthetized, V dogs. Measurements were made at normotension and 3, 5, 10, 15 and 30 minutes after reducing AP to 80 mmHg. AVP was measured by radioimmunoassay. Measurements previously obtained in intact dogs served as control (C). In C dogs, NLQ and AVP increased (591 ± 110 to 1832 ± 176 ml/min/100g and 2 ± 1 to 102 ± 25 pg/ml, respectively) within 3 minutes. Other brain areas showed typical AR. V abolished the NLQ spike (871 ± 176 to 1000 ± 186 ml/min/100g), but left the AVP spike intact (72 ± 5 to 147 ± 37 pg/ml). No other rCBF differences were seen. We conclude that NLQ and neural lobe function are transiently coupled and that this couple affects NLQ AR. The couple does not appear to be metabolically mediated since: 1) AVP remains high during hemorrhage, whereas NLQ returns to C, and 2) V obliterates the NLQ spike leaving the AVP spike intact. Such findings suggest that the afferent pathways affecting the NLQ spike are carried by the vagi and are different from the pathways affecting AVP release.

1718 THE KLUVER-BUCY SYNDROME IN A CHILD: NEURORADIOLOGIC AND PSYCHOMETRIC CORRELATES AND LONGTERM FOLLOWUP. Robert C. Woody, Michael E. Blaw, Amy Talbot, (Spon. by Terry Yamauchi). Department of Neurology and Pediatrics, University of Arkansas for Medical Sciences, Little Rock, and UTHSCD- Southwestern Medical School, Dallas.

The Kluver-Bucy Syndrome, first described in monkeys following bilateral temporal lobectomy, has rarely been described in adults following head trauma, infections, and tumors. No cases have been reported in children. An 8 year old developed diabetic ketoacidosis and stupor. Although medical management of ketoacidosis was uncomplicated, she developed hyperphagia, hypersexuality, hypermetamorphosis, visual agnosia, dull affect, rage alternating with apathy, and a mild right hemiparesis. Cerebrospinal fluid and serum analysis suggested an acute adenovirus infection (titer rise 1:16 to 1:64). CT initially showed striking enhancement of both mesial temporal lobes, the olfactory and orbital-frontal regions, and the thalamic-hypothalamic regions. Repeat CT at three years revealed atrophy and cavitation in these regions. Psychometric testing at three years revealed above average intelligence, severe visual attentive difficulties, a marked deterioration of cognitive control of emotions, and thought processes suggestive of psychosis. Numerous trials of psychopharmacology have failed to alter the girl's behavior. This case resembles adults with Kluver-Bucy Syndrome, and is the first reported case in childhood of acquired limbic dementia with neuroradiographic and psychometric correlation.

1719 THE CO₂ RESPONSE TEST (CO₂RT) IN CHILDREN WITH MENINGOMYELOCELE. Gordon Worley, W. Jerry Oakes, Alexander Spock. Department of Pediatrics and Neurosurgery, Duke University School of Medicine, Durham.

Meningomyelocele is accompanied by the Chiari II malformation of the brainstem which can cause life-threatening symptoms. Since hypercapnic respiratory drive is a medullary function which can be measured, we performed 35 CO₂RT (Δ minute ventilation/kg. $\div \Delta$ P_ACO₂) on 31 patients (ages 1 day to 19 years) with meningomyelocele. Results were classified using published norms. 19 of 31 patients had an abnormal test. The mean age of those with an abnormal test (3.8 years), was less than that of those with a normal test (8.8 years), $p < .01$. Of the 19 patients with an abnormal CO₂RT, 9 had or developed serious signs of brainstem dysfunction (7 had stridor, diminished gag or dysphagia; 2 had apnea with stridor; 1 had apnea alone; 1 had breath holding spells with anoxic seizures). Of the 12 patients with a normal CO₂RT, 1 child had mild dysphagia ($p < .05$). Increased tone and decreased strength of the arms were equally frequent in both groups.

4 patients had 2 CO₂RTs each. 2 improved clinically and had an increase in the CO₂RT; 2 worsened and had a decrease in the CO₂RT. 6 patients had CO₂RTs prior to posterior fossa decompression and laminectomy. The 3 requiring the operation for brainstem dysfunction had an abnormal CO₂RT; the 3 with progressive arm weakness alone had a normal test.

The CO₂RT results correlate with clinical evidence of brainstem function in children with meningomyelocele and the Chiari II malformation.

1720 SAGGITAL SINUS THROMBOSIS POTENTIALLY ACCENTUATES PERINATAL HYPOXIC-ISCHEMIC BRAIN DAMAGE- D. Wurtzel, D. Rosenfeld, R. Vannucci, T. Hegyi, UMDNJ-Rutgers Medical School, St. Peters Medical Center, New Brunswick, N.J. Previous investigations from our NICU have shown that term infants severely asphyxiated at birth often sustain sagittal sinus thrombosis with secondary hemorrhagic infarction of the brain. In these term infants, the thrombotic obstruction to venous outflow was documented by digital IV angiography (Voorhies, et al, 1984). Recently, a 30-week gestation infant was born to a 32 year old Black female with severe sickle-thalassemia. The infant was delivered by emergency C-section for fetal bradycardia. B.W. was 1.060 gm & Apgar scores were 2 & 6. The infant required immediate intubation & subsequent ventilation. Neurological exam revealed a stuporous, flaccid newborn infant with preservation of brainstem and deep tendon reflexes. Seizures were never observed. Head ultrasound & CT scan at 8 hr revealed large, bi-occipital hemorrhagic infarcts. Germinal matrix hemorrhages were also present but there was no IVH. The sagittal sinus was incompletely filled with blood at its posterior aspect, and a Delta sign was present. The CT findings in this infant are equivalent to those observed in term asphyxiated infants in whom sagittal sinus thrombosis occurred. Sequential sonograms showed evolving cystic encephalomalacia of the occipital lobes; repeat CT scan at 1 mo showed large bilateral porencephalic cysts. Presently (2 mo) the infant remains lethargic with little or no spontaneous movements. It is likely that sagittal sinus thrombosis occurred as a consequence of multiple factors; including prematurity, birth asphyxia anemia, congestive heart failure and viral disease. The hemorrhagic infarcts resulting from the sagittal sinus thrombosis produced major and permanent cystic brain damage.

●1721 THE EFFECT OF SEIZURES ON CEREBRAL METABOLITES IN CHILDREN. Donald Younkin, John Maris, Eileen Donlon, Robert Clancy, Barry Lawson, Ronnie Guillet, Britton Chance, Maria Delivoria-Papadopoulos. Univ. of PA., Depts. of Neurology, Pediatrics, Biochemistry, Phila., PA. 19104

The effect of seizures on cerebral metabolites have been evaluated in animals but has not been studied previously in humans. We used 31-P NMR spectroscopy to measure changes in the cerebral phosphocreatine (PCr) to inorganic phosphate (Pi) ratio and to calculate intracellular pH (pHi) in five infants with seizures. Three babies developed seizures after perinatal asphyxia; two infants had the onset of seizures beyond the neonatal period (4 wk of unknown etiology; 11 wk due to intracranial hemorrhage). When studied the infants were clinically stable on anticonvulsants. Metabolic changes were present on initial study and varied with time between most recent seizure and NMR. Major changes were seen in two children with subtle seizures during the study and focal abnormalities on EEG: PCr/Pi $\sim .75$ in the hemisphere with seizures and ~ 1.6 in the contralateral hemisphere. Two patients with focal seizures, but no clinical manifestations during the NMR study, had less severe metabolic abnormalities on the side of EEG abnormality ($\sim .87$ vs ~ 1.18). The fifth child with asphyxia, prolonged status epilepticus and grade 4 IVH had marked bilateral changes (PCr/Pi $\sim .5$) that improved with more aggressive anticonvulsant management (PCr/Pi ~ 1.2). Interhemispheric differences in pHi were not significant ($7.1 \pm .4$ in hemisphere with seizures vs $7.2 \pm .2$) suggesting that CBF was sufficient to remove lactate generated by anaerobic glycolysis. Four patients had serial studies that showed improvement in cerebral metabolite ratios, suggesting that the initial changes were not due to structural damage. Our results suggest that human seizures cause significant cerebral metabolic stress and may cause or exacerbate neurologic damage.

PULMONOLOGY

1722 THE COMPARATIVE EFFECTS OF THEOPHYLLINE AND CAFFEINE THERAPY ON VENTILATORY RESPONSES OF GROWING PRETERM NEONATES TO COMBINED INSPIRATORY AND EXPIRATORY LOADS. S. Abbasi, E.M. Siviari, V.K. Bhutani, W.W. Fox. Univ. Pa. Sch. Med., Dept. Peds., Pa. Hosp., Children's Hosp. of Phila., Pa.

The comparative effects of caffeine therapy (C) and theophylline therapy (T) on ventilatory responses of growing preterm infants with periodic breathing were evaluated. $\bar{X} \pm \text{SEM}$ BW = 1500 ± 88 gm, GA = $31.4 \text{ wks} \pm 1.45$, study age = 34.7 ± 9.4 wks, study weight = 2077 ± 219 gm, T level = 8.4 ± 2.2 mg/dl, C level = 10.0 ± 1.3 mg/dl. An external combined inspiratory and expiratory load ($R = 100 \text{ cm H}_2\text{O/L/sec}$) was applied for 60 sec before and during T (Gr.1, N=8), and C (Gr.2, N=6). Pulmonary variables: tidal volume (V_T), respiratory frequency (f), minute ventilation (MV), inspiratory time/total respiratory time (T_I/T_T), esophageal pressure (P_{es}) and work of breathing (WOB) were obtained before and 60 sec after application of R. There was no significant difference between the two groups in control pulmonary measurements. Control values in Gr.1 prior to T were: $V_T = 7.9 \pm 1.7$ ml/kg, $f = 59.8 \pm 3.2$ breaths/min, $MV = 466 \pm 4$ ml/min/kg, $T_I/T_T = .47 \pm .23$, $P_{es} = 9.3 \pm 1.7$ cm H₂O, $WOB = 43.6 \pm 14.3$ gm cm/kg. 6/8 infants had several apneic pauses and only 4/8 tolerated R for 60 sec prior to T. 4/6 Gr.2 infants had apneic pauses and 5/6 tolerated R prior to C. Application of R resulted in an increase in P_{es}. This response was significantly greater ($p < .05$) during T and C. T and C therapy similarly resulted in improved ventilatory endurance (all infants in Gr.1 and 2 tolerated load for 60 sec with significantly fewer respiratory pauses). There was no significant difference between the T and C in augmenting ventilatory drive and maintaining ventilatory output.