THE ELECTROCONVULSIVE THRESHOLD AND CEREBRAL METABOLISM DURING STARVATION IN THE ADULT RAT. Darryl C. DeVivo, Kenneth Malas, Mary P. Leckie (Intr. by Arthur L. Prensky); Washington Univ. Sch. of Med., St. Louis Children's Hosp., Dept. of Ped., St. Louis.

Brief fasting raises the convulsive threshold in the human. This clinical observation led to the use of a ketogenic diet in epilepsy. To study the mechanism underlying this observation, the electroconvulsive threshold (ECT) of adult rats was determined before fasting and at 24, 48 and 72 hours after food deprivation. The ECT was unchanged at 24 hours, rising at 48 hours ($p < .05$) and 72 hours ($p < .01$). During starvation blood glucose levels fell and blood ketone bodies rose with similar changes, respectively, in brain tissue. Brain sodium levels were slightly higher at 24 and 72 hours and unchanged at 48 hours whereas brain potassium levels gradually rose at 48 hours ($p < .10$) and at 72 hours ($p < .025$). Brain water content was relatively constant. Ouabain sensitive and insensitive ATPase activity remained unchanged. Glucose-6-phosphate levels rose at 48 and 72 hours whereas malate fell progressively throughout fasting. Lactate and pyruvate levels fell initially then rose and the L/P ratio fell steadily from 12.7 to 10.9. The adenylate pool and Pi remained constant but the cerebral energy charge potential and the phosphorylation state rose at 48 and 72 hours ($p < .05$). The cerebral energy reserve remained constant. These observed changes are felt to be the consequence of increasing utilization of ketone bodies for cerebral oxidative metabolism with a secondary depression in glycolysis. The rise in brain potassium may reflect this increased utilization of anions and explain the rise in the electroconvulsive threshold.

NEONATAL VISUAL PATTERN FIXATION. A POSSIBLE PREDICTOR OF FUTURE MENTAL PERFORMANCE. Simón Miranda, Maureen Hack, Avroy Fanaroff, Marshall Klaus. Case Western Reserve Univ. Depts. of Pediatrics and Psychology, Cleveland.

Accurate assessment for subsequent mental performance is now especially needed in the neonatal period. To determine the prognostic value of visual fixation responses in the neonate, 14 infants born at 27-44 weeks gestation with worrisome problems including severe asphyxia (8), systemic herpes (2), suspected microcephaly (2), severe tremors (1), and abnormal skull calcification (1), were tested neurologically and visually at a post conceptional age of 35-46 weeks. The visual tests, based on techniques developed by Fantz, assessed fixation of a single pattern, discrimination between pattern over plain and between two patterned stimuli.

These infants were subsequently followed and evaluated by Stanford-Binet or Bayley tests at an average age of 28 months (range 4-60 months). Prediction of development based on visual testing was accurate for 13 of 14 infants. The neurological exam was accurate for only 7 of 14 infants. Five rated normal by visual testing are normal on follow-up. Of 8 infants abnormal by visual testing, 5 are now retarded (I.Q. or D.Q. < 70) and 3 died (hydrancephaly, microcephaly, severe retardation). One infant rated normal by both visual and neurological exam is retarded.

This preliminary study suggests that neonatal visual testing may be a valuable tool for predicting future mental performance.

INCREASED BRAIN GLUCOSE AND GLYCOGEN AFTER HYDROCORTISONE: MECHANISMS OF ACTION. Jean Holowach-Thurston, Richard E. Hauhart, and Elizabeth M. Jones, Washington Univ. Sch. of Med., St. Louis, Mo.

We have previously reported that in young mice hydrocortisone (100 mg/kg s.c. from 10 to 20 days of age) doubled brain glucose (J. Neurochem. 16:107, 1969). Possible explanations were increased glucose transport from the blood to the brain or decreased glucose utilization by the brain. In this study brain metabolism was estimated from the rate of fall of total available and potential high energy phosphate after decapitation (O.H. Lowry, et al., J. Biol. Chem. 239:18, 1964). 5 litters of mice (treated as above) were either frozen whole in liquid N_2 or decapitated and the heads frozen after 15 and 30 sec. Zero time hydrocortisone mice had significantly elevated brain glucose (56%, $p < 0.001$) and glycogen (16%, $p = 0.004$). Brain ATP, P-creatine and lactate were unchanged. The fall of high energy phosphate during 30 sec. of ischemia was 6.63 mmoles/kg in controls and 6.44 mmoles/kg in hydrocortisone treated animals indicating no change in cerebral metabolic rate. The brain to plasma glucose ratio, an index of glucose transport when the metabolic rate is normal, was 90% higher in the hydrocortisone animals, namely 0.207 vs. 0.109 in controls. Other explanations for the increased glucose and glycogen, such as inhibition of the pentose phosphate pathway and (although less likely) increased gluconeogenesis as in other tissues after hydrocortisone, are currently under investigation.

PROSPECTIVE EVALUATION OF ABNORMALITIES OF THE CRANIOSPINAL AXIS (CSA) IN NEWBORNS. Keith R. Powell, Thomas J. Hougen, and James D. Cherry, UCLA Sch. Med., Dept. Ped., Los Angeles.

A review of the literature revealed 104 cases with dermal defects distributed along the CSA (59% lumbar or lower and 31% occipital). Although at least 41 patients had lesions noted as newborns, only 20 patients were operated on before becoming infected. Ten of the affected children died and 32 had neurologic residua; none of 20 children operated on before becoming infected died or had residua. Adequate incidence and treatment data could not be determined.

Term infants born at the UCLA Hosp. from Sept. 1 - Dec. 31, 1973 were examined for abnormalities of the CSA. The CSA of each infant was palpated and inspected (in good light, parting the scalp hair, and using magnification when necessary) from the bridge of the nose to the anus. Suspicious lesions were photographed, and roentgenographs obtained for presumed sinuses and other selected lesions. One or more suspicious finding was seen in 39 (4.2%) of 834 neonates. These included 16 (1.9%) deep dimples (bottom seen); 14 (1.7%) presumed sinuses (bottom not seen); 6 (.7%) masses; 3 (.35%) skin tags; 2 (.22%) discolorations; and 1 (.12%) abnormal hair. All lesions but one were lumbar or lower. Incidence by sex was equal, and no roentgenographic abnormalities were found.

The results show an alarmingly high incidence of abnormalities of the CSA with a surprising paucity of occipital lesions. The significance of these lesions remains to be defined by careful follow-up and surgery in selected cases.

NEUROLOGIC ASSESSMENT OF HYPERBILIRUBINEMIC INFANTS: William J. Keenan, Richard Butcher, Kathy Kazmaier (intr by James M. Sutherland), Univ. of Cinti, Col. of Med., Dept. of Ped., Cincinnati, Ohio.

An in vivo evaluation of the encephalopathic risk of hyperbilirubinemia would ideally consist of a brief non-invasive repeatable measure that correlated with CNS function, would be sensitive to minimal toxicity and would demonstrate improvement with appropriate therapy. Assessment of non-nutritive suck (NNS) was carried out immediately before and within 1 hour after ten 2-volume exchange transfusions performed for hyperbilirubinemia in 5 infants. NNS was sensitive to acute reduction of serum bilirubin and changes in NNS were demonstrated each time bilirubin levels were lowered. Improvement in NNS correlated with the demonstrated ability of exchange transfusion to reduce bilirubin encephalopathy. Mean ± SEM suck rate was 1.56±.05 per second per burst pre-exchange and 1.77±.03 post-exchange ($t=5.8, p<.001$). With lowered bilirubin levels there was a trend towards fewer abnormal wave forms, less frequent tremor in the interburst interval, and an increase in median burst amplitude but these differences were not significant. The results demonstrate that NNS may be a sensitive measure of the in vivo effects of hyperbilirubinemia and may be useful in monitoring the response to therapy.

WORMIAN BONES: RADIOLOGICAL WARNING OF ABNORMAL NERVOUS SYSTEM. Charles V. Pryles, Abdul J. Khan, Dept. of Ped., S.U.N.Y., Downstate Med. Ctr. and The Jewish Hosp. & Med. Ctr. of Brooklyn, N.Y.

A total of 515 infants and children from inpatient, outpatient, and mental retardation clinics were studied to correlate clinical features with the radiological presence of intrasutural or Wormian bones (WB), which are seen in osteogenesis imperfecta, cretinism, cleidocranial dysostosis and hydrocephalus. The four fold higher incidence of WB in the mentally retarded group than in the rest was significant ($P=0.01$). Ninety-one, or 17% showed WB in their skull roentogenograms. Of these, 96% had manifestations of CNS abnormalities, including 5 cases (6%) with minimal brain dysfunction and 82 (90%) with gross CNS disorders. Three of the remaining four cases had anomalies of other organ systems. The incidence of WB, was unrelated to sex or race, but its decrease with age (68% below 5 years, 24% between 5-10 years and 8% above 10 years) is probably related to closing of sutures. Demonstration of WB in young infants may be a sueful sign in early identification of CNS abnormalities.