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**LITHIUM: PLACENTAL TRANSFER, BREAST MILK EXCRETION, AND SERUM ELECTROLYTE CHANGES.** B. R. Parks, B. H. Douglas, and J. E. Rawson. Departments of

Pediatrics and Anatomy, University of Mississippi Medical Center, Jackson, Mississippi. (Sponsored by B. Batson).  
 Five pregnant rabbits weighing approximately 3 kg each were used to determine lithium disposition following oral administration of lithium to the mothers. All of the animals received 25 mg/kg lithium carbonate orally prior to delivery, and lithium, furosemide (2 mg/kg), or a combination of the two drugs after parturition. This dose of lithium resulted in maternal serum levels of  $0.50 \pm 0.10$  mEq/l. On the day of delivery, maternal values decreased to  $0.38 \pm 0.06$  mEq/l, offspring serum levels were  $0.46 \pm 0.13$  mEq/l, and breast milk concentrations were  $0.13 \pm 0.06$  mEq/l. Maternal serum Na and K were decreased during administration of lithium, furosemide, and the combination as compared to control values. The combination treatment resulted in a change in serum Na from  $147.7 \pm 6.9$  mEq/l to  $129.0 \pm 17.1$  mEq/l and a change in serum K from  $3.89 \pm 0.77$  mEq/l to  $3.33 \pm 0.79$  mEq/l. Lithium alone depressed sodium levels more than furosemide while the converse was observed for potassium concentration.

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**INTRACRANIAL PRESSURE (ICP) MONITORING DURING GENERAL ANESTHESIA:** T. N. K. Raju, C. Torres, E. J. Bennette, E. M. Grundy, D. Vidyasagar (Spons. by I. M. Rosenthal) ALSM. Uni.

Ill. Dpt. Peds and Anes., Chicago, Illinois.  
 A significant rise of ICP during anesthesia has been reported in adults. (R.W. Adams et al. 1972). This is believed to be due to the effects of halothane. We studied ICP changes in 9 infants undergoing surgery. The procedures were: herniorrhaphy in 6, ventriculo peritoneal shunt in 2 and anoplasty in 1. The age ranged from 7 days to 10 months and the weight ranged from 1.5 to 9 kg. ICP was measured via the anterior fontanel (AFP) using a previously described technique (Ped. 59:957, 1977). All received a mixture of halothane, N<sub>2</sub>O<sub>2</sub> and O<sub>2</sub> for anesthesia. The procedures lasted for 41.5 ± 5.2 minutes. The mean ± SE AFP before anesthesia (Pre. Anes.) during awake intubation (Int. Awake) after a brief induction (Int. Indu.) during anesthesia (Du. Anes.) and post extubation (Post. Ext.) are shown in the Table.

	Pre. Anes.	Int. Awake	Int. Indu.	Dr. Anes.	Post. Ext.
AFP in Cm. H <sub>2</sub> O	15.5 ± 1.8	89.7 ± 8.1	33.6 ± 5.2	27.1 ± 2.2	14.3 ± 2.1
(n)	(8)	(5)	(4)	(9)	(6)

The  $\Delta$  rise in AFP during awake intubation was highly significant. ( $p < 0.0001$ ). AFP during the procedure remained higher as compared to pre. anes. levels ( $p < 0.001$ ). Post extubation AFP was normal. In 2 patients undergoing VP shunt there was a gradual fall in AFP during the procedure and in one of these ventricular fluid pressure and AFP were similar. (16.5 & 16.0 Cm. H<sub>2</sub>O). The data indicate: 1. A significant rise in AFP occurs during halothane anesthesia and 2. during awake intubation. 3. The rise in pressure persists during the procedure.

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**LIVER ULTRASTRUCTURE IN NEUROLOGICAL GRADE I (SO-CALLED MILD) REYE'S SYNDROME (RS).** John C. Partin, Jacqueline S. Partin, and William K. Schubert. University of Cincinnati, Children's Hospital Medical Center, Department of Pediatrics, Cincinnati.

Of 106 consecutive cases of RS, 23 were neurological Grade I upon admission. Fourteen cases, including 2 who progressed to coma, received diagnostic liver biopsy for examination by lipid histochemistry, and electron microscopy (EM). Initial clinical chemical measures were SGOT 130-1524 IU (mean 609), SGOT 50-2300 IU (mean 761), blood NH<sub>3</sub> 15-339  $\mu$ g/dl (mean 103), CPK 0-10 Bio-science units (mean 2.3). By light microscopy, all liver specimens were typical of RS, demonstrating heavy panlobular microvesicular neutral fat. There was substantial variation in organellar damage from patient to patient; all demonstrated universal mitochondrial matrix expansion and pleomorphism in osmium-fixed liver, but matrix dense bodies were not universally absent, being present in most mitochondria of least damaged livers and absent in 2 livers demonstrating the most severe EM changes. Peroxisomes and smooth endoplasmic reticulum (ER) were greatly increased; glycogen was reduced and golgi lipoprotein particles were reduced or absent in all but 1 case. Rough ER was normal. Bile stasis was absent. Some disintegrating liver cells were present in all samples. **Conclusion:** In RS, Grade I neurological status is not synonymous with "mild" disease because: 1) some Grade I children have unexpected severe alteration of liver ultrastructure; and 2) certain children unexpectedly progress to severe encephalopathy. All Grade I cases should be hospitalized for glucose infusion and careful observation.

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**A PROGRESSIVE NEUROLOGIC SYNDROME IN SIX CHILDREN WITH CHRONIC LIVER DISEASE AND ALPHA-TOCOPHEROL (VIT) DEFICIENCY.** Jerry L. Rosenblum, James P. Keating, James S. Nelson and Arthur L. Prenskey, Washington Univ. Sch. Med., Dept. of Ped., St. Louis Children's Hospital, St. Louis.

We have followed six children with prolonged obstructive jaundice of infancy (biliary atresia) who are now 6 yrs to 12 y/o. Two can no longer walk unaided. A striking paresis of vertical gaze and wide-based gait developed in two children. Serial neurological exams revealed marked hyporeflexia (6/6) and decreased proprioception in the four cooperative patients. However, touch (4/6) and pin (5/6) sensation was intact. 2 of 3 cases tested had decreased motor nerve conduction velocities but normal electromyograms. Psychometric testing and school performance indicated a wide range of intellectual function. Serum vitamin E levels were low (range  $< 0.1$ - $0.8$  mg/dl; mean  $0.2$  mg/dl; normal  $0.8$ - $1.2$  mg/dl) in all cases despite long-term oral administration of  $\alpha$ T-polyethylene glycol-1000-succinate. Post-mortem neuropathologic and morphometric findings in one child with biliary atresia whose serum vitamin E level was  $0.1$  mg/dl were similar to those observed by one of us (J.S.N.) in chronic vitamin E deficient monkeys and rats with focal neuronal loss in dorsal root ganglia, degeneration of axons in Clarke's column and in the fasciculus gracilis and cuneatus, and a decrease in large myelinated axons in the sural nerve. Further studies are needed to elucidate what role decreased vitamin E plays in this syndrome.

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**HYPONATREMIA AS A POSSIBLE SIGN OF IMPENDING HYDROCEPHALUS IN INFANTS  $\leq 2000$  GMS. AT BIRTH.** Jeffrey Pomerance, Barry Perlin, Rosa Sanchez, Christina

Ukrainski, Janet Meredith (Spon. by B. M. Kagan). Cedars-Sinai Medical Center, Dept. of Ped. and UCLA Sch. Med., Los Angeles.  
 Early diagnosis and treatment of hydrocephalus are necessary for successful outcome. The advent of computerized axial tomography (CAT) scans has facilitated diagnosis. Unless CAT scan is performed routinely, however, diagnosis at the earliest operable stage may be missed.  
 The charts were reviewed of 35 neonates weighing  $\leq 2000$  gms. at birth who received CAT scan evaluations for detection of hydrocephalus. Serum Na levels were measured as part of their routine care. Infants with meningitis or meningomyelocele were excluded from the review as were infants who died prior to 28 days of age. Nine infants had hydrocephalus. Five of these infants had verified (repeated) serum Na levels of  $\leq 125$  meq/L which occurred 1 to 62 days prior to diagnosis of hydrocephalus. Of the 26 infants whose CAT scan did not reveal hydrocephalus, only 4 had verified hyponatremia. ( $\chi^2=5.6$ ;  $p < 0.05$ ).  
 The etiology of the low serum Na levels in the infants who later developed hydrocephalus is unclear. Low Na intake by itself appears to be an unlikely etiology. Spot urine Na levels, urine and serum osmolalities, and increments in infants' weights were not totally consistent with inappropriate anti-diuretic hormone secretion. Presence of hyponatremia may be a high risk marker which identifies the infant at greater risk of subsequent development of hydrocephalus.

**1151**

**THE EFFECTS OF AN EARLY INTERVENTION PROGRAM ON THE LOW BIRTH WEIGHT INFANTS.** A.G. Rosenfield, B.R. Vohr, R.M. Cowett, E. Denhoff, W. Oh. Brown Univ. Program in Med., Women & Infants Hosp., Dept. of Pediatrics, and the Meeting Street School, Providence, Rhode Island.

This study examined the effects of an early intervention program on 93 infants weighing  $< 1500$  gms. at birth. The study infants (mean birth weight = 1230 gms., mean gestational age = 30 wks.) were randomly assigned to an intervention (I) or non-intervention (NI) group. The I group (n = 49) received 40 min. of proprioceptive stimulation daily beginning at 2 to 3 wks. of age which continued throughout the 2 to 3 months of hospitalization. The gestational age, birth weight, sex distribution, socio-economic status, and maternal educational level were comparable between the two groups. The I infants evidenced a higher state rating and their parents visited more frequently than the NI group ( $p < .01$ ). Both positive observations are favorable factors for maternal-infant bonding. Neurological and Bayley scale assessment at 3, 6, 9, 12, and 18 months showed significant improvement with increasing age ( $p < .01$ ), but with no significant differences between the two groups. The data demonstrate the positive role of an early intervention program for enhancing maternal attachment, but fail to suggest a beneficial effect on the neurological and developmental performance at 18 months of age in the low birth weight infant.