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VIREMIA IN HYPERPYREXIC CHILDREN. Terry Yamauchi, David T. Imagawa, Mia Y. Nishita, James A. Gammon, Edward G. Vollman. Harbor General Hospital, UCLA

School of Medicine and Arkansas Children's Hospital, Department of Pediatrics, Torrance, California and Little Rock, Arkansas.

Sixty-three children between the ages of 4 months and 16 years were studied because of temperatures of 39.5°C (103°F) or greater without an obvious source of infection. No child had received antibiotics for at least two weeks prior to this evaluation. Every child had bacterial cultures of blood, pharynx, cerebrospinal fluid (CSF) and urine. In addition, viral culture of pharynx, stool and buffy coat was obtained. Fifty-nine children had their CSF cultured for viruses.

Viruses were recovered from 25 children (39.5%). Fourteen children had viruses isolated from their pharynx, 10 from feces, 4 from CSF and 10 from the blood. Several children had viruses cultivated from multiple sites. Cytomegalovirus was discovered in the buffy coat culture of a single child. Mumps virus was recovered from the blood and pharynx of 2 siblings. One of these children developed parotitis 5 days after culturing. The only symptom in the other child was the initial febrile episode. Echoviruses were retrieved from cultivation of buffy coats in 7 additional children. One child was carrying echovirus in his pharynx and stool as well as blood.

The recovery of viruses from the blood of hyperpyrexia children is demonstrated. Echoviruses were the most frequently isolated viruses from buffy coat cultivation. Viremia should be considered in the evaluation of high temperatures in children.

METABOLISM

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RADIOENZYMATIC (REA) ASSAY OF CATECHOLAMINES. V. Abbassi, S. Montgomery and P. Jose. Georgetown Univ Med Center, Dept of Pediatrics, Washington, D.C. 20007.

Catecholamines play a prominent role in neural and endocrine integrative mechanisms and exert specific effects on lipid and carbohydrate metabolism. Despite their physiologic significance adequate information regarding plasma concentration (concent) and variation under various physiopathologic conditions is lacking. By means of a REA blood concent of epinephrine (E) and norepinephrine (NE), in the fasting state (F) and in response to insulin induced hypoglycemia were studied. 16 children with endocrine disorders and 8 with no apparent disease (controls = C) were included in the study. Fasting levels of E and NE in C (Mean ± SE) were 104 ± 23 and 206 ± 47 pg/ml. At 60' post insulin, E increased to 1111 ± 381 pg/ml, P < 0.05 and NE to 424 ± 121 pg/ml, P > 0.05. In 3 pts with Ketotic hypoglycemia (KH), E and NE at F and 60' were comparable to C group, 100 ± 17 and 458 ± 18 and 294 ± 18 and 243 ± 34 pg/ml respectively. 2 pts with Reactive hypoglycemia (RH) and 1 with GH deficiency had minimal E responses, 45 to 83, 17 to 32 and 33 to 50 pg/ml. A pt with panhypopit (PH) exhibited the highest levels of E and NE at F, 30 and 60'; 1619, 2498 and 2127 pg/ml for E and 1413, 6385 and 2475 pg/ml for NE respectively. In remaining pts, F and 60', E and NE were normal. Contrary to previous observations, E and NE concent and responses in pts with KH were comparable to C group. Surprisingly, however, low E concent and responses were found in 2 pts with RH, and markedly elevated E and NE levels, in PH pt. These observations imply the significance of the REA of catecholamines and warrant further investigation of their role in these endocrinopathies.

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THE FED HUMAN NEONATE-A SUCKLING KETOSIS? Endla K. Anday, Charles A. Stanley, Lester Baker, Albert I. Winegrad and Maria Delivoria-Papadopoulos. Univ. of Pa. and Children's Hosp., Dept. of Pediatrics, Phila., PA 19104

Rats demonstrate a marked suckling ketosis (total ketones = 2.5 mM). We have found evidence suggesting that the capacity for ketogenesis is deficient in fasted human newborns. Therefore, it was of interest to see whether human infants also show an improvement in ketogenic capacity and become ketotic with feeding. The levels of free fatty acids (FFA), b-OH-butyrate (BOB), and acetoacetate (AcAc) in 24 term AGA newborns fasted for the first 8 hrs after birth were compared with levels seen in 14 infants examined at 18 hr of age, 4 hr after a first feed, and 11 infants studied at 36 hr of age, 4 hr after their 4th-6th feed.

	FFA mM	BOB mM	AcAc mM
8 hr fast	1.40 ± 0.07	0.31 ± 0.05	0.05 ± 0.01
18 hr fed	1.36 ± 0.08	0.46 ± 0.04*	0.18 ± 0.02**
36 hr fed	0.93 ± 0.07**	0.25 ± 0.06	0.16 ± 0.03**

p < 0.05

p < 0.01

The small but significant elevation of ketones in the fed infants suggests an increase in ketogenic capacity. However, the magnitude of the increase in the human newborn is markedly lower than that seen in the suckling rat, and suggests that the rat model may not be applicable to the human.

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THE CHANGING FACE OF INFANTS OF DIABETIC MOTHERS

Billy F. Andrews, Robert Lega

University of Louisville, Louisville General Hospital, Department of Pediatrics, Louisville

Improved neonatal diagnostic methodology and therapy have significantly lowered the mortality of infants of diabetic mothers. The small for date or small for gestational age (SGA) infant was unrecognized or of rare occurrence prior to the 1960's. Comparable improvements in obstetric or perinatal care over the past 10 years has altered Farquhar's classic description of infants of diabetic mothers. Also, Pedersen's observation of normal appearing infants of well controlled diabetics is confirmed by our most recent experience with such infants. Fifteen per-cent were SGA, thirty-nine per-cent were appropriate for gestational age (AGA), and forty-six per-cent were large for gestational age (LGA). Pictures of infants with determination of gestational age and birth weight and maternal classification of disease will be presented. The favorable change in appearance (increased SGA infants) has accompanied a decreasing perinatal mortality. The occurrence of an increased number of SGA infants provides a new focus of attention.

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COMA DUE TO TRANSIENT HYPERAMMONEMIA IN THE PRETERM INFANT. R. A. Ballard, B. Vinocur, J. Reynolds,

W. Kane, W. Nyhan, L. Sweetman. Depts. of Ped., Mt. Zion Hosp. San Francisco, CA, Univ. of Minn., Minneapolis, and Univ. of Calif., San Diego.

We have seen 3 preterm infants with respiratory distress and severe central nervous system depression due to transient hyperammonemia. All 3 were 34-36 wk products of uncomplicated vertex deliveries with Apgar scores ≥ 6 at 1 min and ≥ 8 at 5 min. All developed respiratory distress in the first hour of life and later required mechanical ventilation with inspired oxygen concentrations of > 50%. Lethargy progressing to coma with apnea, loss of response to deep pain, and unreactive pupils developed at 14-40 hrs of age, prior to protein feedings in 2 infants. Two had seizure activity. Ammonia levels of 844 to 5660 μmol/l (nl < 70 μmol/l) were obtained 50 to 84 hrs after birth. Two had return of spontaneous movement and respiration during exchange transfusion. The third infant (ammonia level 5660 μmol/l) also required 36 hrs of peritoneal dialysis before regaining responsiveness. Ammonia levels fell to normal after treatment and remained low from 7 days of age. Electrolytes, BUN, liver enzymes, urine and plasma amino acids, and cultures of CSF, blood and urine were unremarkable. All infants subsequently took protein feedings without a rise in ammonia. Two infants later had liver biopsies with normal levels of urea cycle enzymes. All 3 are apparently normal at 8 to 20 mos of age. Transient hyperammonemia of unknown etiology should be considered and treated vigorously in preterm infants with CNS depression and respiratory distress.

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DIABETES MELLITUS AND BONE MINERAL. Gerald J. Bargman and Richard Mazess (Spon. by Russell Chesney) Univ. of Wisconsin, Depts. of Pediatrics & Radiology, Madison.

Bone mineral content was determined in 38 children with insulin dependant diabetes mellitus of various duration. Direct photon absorptiometric scans were made on the radius, ulna and humerus of these patients, and the results were compared to those obtained on normal children taking into account (by multiple regression analysis) age, sex, height and weight. The children with diabetes mellitus deviated from expected bone values by -14.3% (about 1.5 standard deviations). In contrast there was only a minor deviation (-4.6%) from expected height. The extent of bone mineral deviation was not correlated with height deviation indicating that the bone mineral deficit was not associated with growth retardation. The patients were also divided into 4 subgroups based on degree of disease control. The average deviation of bone mineral content was over -10% in each subgroup. There was no association between disease control and bone mineral deviancy (nor height deviancy). These results appear to confirm findings reported in the literature that bone mineral is reduced in diabetes, but this reduction seems to be related to the underlying disease rather than its treatment or degree of control.