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INCREASING SERUM PARATHORMONE (PTH) AND MIGRATORY SUBCUTANEOUS CALCIFICATION IN THE PROGRESSION OF PSEUDOPSEUDOHYPOPARATHYROIDISM TO PSEUDOHYPOPARATHYROIDISM P.S. Venkataraman, R.C. Tsang, F.R. Greer, J. Whitsett, J.J. Steichen, D. Dodson, U. of Cincinnati

Pseudopseudohypoparathyroidism progressing to pseudohypoparathyroidism has not been previously studied. A male sibling of a child with pseudohypoparathyroidism was studied for 2 yrs. At 1 yr he had generalized subcutaneous calcifications which were "migratory", was round faced, slightly obese, with stubby fingers and dimples on the dorsa of his hands. Serum Ca was 9-9.5 mg/dl for the 1st 18 mos and then fell to 8.9, 8.6, 8.3 mgm/dl over 6 mos. Serum P was 5.7-6.2 mgm/dl for 18 mos and rose to 6.4, 6.8 and 7.6 mgm/dl over 6 mos. Serum PTH rose from 54 μ Eq/ml (N <57) to 89, 117, 312, 735 and 750 μ Eq/ml over 6 mos. 1,25 Dihydroxy Vit. D increased from 40 to 69 pg/ml (N=17-44 pg/ml). At 3 yrs there was no calcemic, phosphaturic or urinary cAMP response to PTH. Serum PTH was suppressed by Ca infusion and doubled with EDTA infusion indicating that the parathyroids were sensitive to Ca changes. On x-ray, enlarged cranium, thickened diploe, broad and short 1st and 4th metacarpals were seen. Auto immune antibodies were absent. Serum T4 was 7.2mg/dl (low N) and TSH was elevated 17.6 μ U/ml. Prolactin and cortisol were N. Thus, increasing PTH levels presage overt pseudohypoparathyroidism and may indicate preclinical disease. We speculate that increasing serum PTH reflects increasing compensatory parathyroid production to overcome the PTH receptor defect and serves in the early stage of the disease to maintain normocalcemia.

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ONTOGENY AND THYROID DEPENDENT MATURATION OF β -ADRENERGIC RECEPTORS AND ADENYLATE CYCLASE IN RAT LUNG. J.A. Whitsett, H. Neederman*, J. Pollinger*, Dept. Pediatrics, U. of Cincinnati, Ohio

Catecholamines mediate surfactant release and smooth muscle tone in the lung by interaction with β -adrenergic receptors (BAR) and adenylate cyclase (AC). This study describes the ontogeny of BAR and AC in rat lung membrane and the role of thyroid hormone in the postnatal increase in BAR. BAR were identified with [3 H] dihydroalprenolol ([3 H]DHA). Bmax increased from 46 $\pm$ 4.9 fmole mg $^{-1}$ protein to 491 $\pm$ 39 m $\pm$ SE from d 18 of gestation to adult levels postnatal d 28. Increases in lung BAR occurred between postnatal d 14 and 28, coincident with the known increase in T3 in the rat; we determined lung BAR in hypothyroidism (Hypo) induced with propylthiouracil prior to and after birth, normal age-matched control pups, and euthyroid pups (Euth) maintained on PTU but treated with T4. Euth pups grew normally and by d 28, lung weight, DNA, protein and BAR were identical to controls. Hypo pups grew normally to d 14 but grew poorly thereafter; by d 28 lung weight, DNA, and protein were decreased in Hypo pups. Lung BAR's were similar to controls up to d 14, but were decreased in Hypo at d 28, 290 $\pm$ 15, n=13 vs. 489 $\pm$ 31 fmole $\cdot$ mg $^{-1}$, n=8 in Euth, m $\pm$ SE, p<0.001. Treatment of Hypo pups with T4 on days 25-27 doubled lung BAR by day 28. Finally, maximal catecholamine sensitive adenylate cyclase activity was also thyroid hormone dependent on d 28. We conclude that thyroid hormone dependent factors are required for the postnatal maturation of the β -adrenergic system in the rat lung.

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EFFECT OF GROWTH HORMONE THERAPY ON SLEEP. R. Hk. Wu, Albert Einstein Coll. of Med., Montefiore Hosp. and Med. Ctr., and Bronx-Lebanon Hosp. Ctr., Dept. of Pediatrics, Bronx, NY (Sponsored by M. Cohen).

Total sleep time, sleep difficulty (time spent awake and moving during sleep), REM latency (time to first REM period), number of REM periods, and time spent in sleep stages, REM, I, II, III and IV, were measured in four patients with growth hormone (GH) deficiency before and after GH therapy. Three patients adapted for 2 or more nights prior to the study and each patient had 2 or more studies. One patient had one study after GH.

The percentage of time spent in stage II sleep increased in three patients while in the fourth patient it did not change. The mean percentages (minutes) of stage II sleep were 40.4% (223 min) and 43.2% (218 min) before and after respectively. The amount of time spent in slow-wave (III/IV) sleep was found to decrease in three patients. The mean percentages were 26% and 24% respectively. None of the above changes were statistically significant when the means were compared. Total sleep time (550 $\pm$ 86 and 518 $\pm$ 66 minutes respectively), sleep difficulty, REM latency periods and the number of REM cycles were not different before and after GH. While these data tend to suggest that there is a shift in the stage of sleep in these patients from slow wave III/IV to stage II sleep, they were not statistically significant. No other parameters are significantly affected. These data do not support the data found in animals who were shown to have increases in slow wave sleep after GH administration.

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BLOOD GLUCOSE IN RELATION TO VARIOUS CLINICAL CONDITIONS, PLASMA INSULIN, GLUCAGON AND GROWTH HORMONE IN PREMATURE INFANTS. J.J. Yoon, A.E. Esquea, R. Hk. Wu, Albert Einstein Coll. of Med., Bronx-Lebanon Hosp. Ctr., Dept. of Ped., Bx., NY (Sponsored by M. Cohen).

Blood glucose and glucose regulating hormones: insulin, glucagon and growth hormone were measured in blood drawn from 31 premature infants weighing 1670 gm or less in the following study period: 0-12, 13-24, 25-48, 49-72, 73-96 hours of age. The levels of blood glucose were analyzed in relation to the levels of glucose regulating hormones, intravenous glucose infusion rates and the presence of various clinical conditions; birth weights 1000 gm or less, gestational age less than 32 weeks, small for gestational age, chronic intrauterine stress, perinatal asphyxia, central nervous system (CNS) hemorrhage and mortality. The mean (range) blood glucose levels during the intervals of collection were 145 (26-700), 157 (31-450), 109 (41-465), 145 (65-1000) and 91 (30-203) mg/dl, respectively. Hypoglycemia with blood glucose levels of 20 mg/dl or less was prevented, but transient neonatal hyperglycemia with blood glucose levels of 125 mg/dl or more occurred frequently (80.6% $\pm$ 25 of 31 infants and 32% $\pm$ 41 of 128 samples). Hyperglycemia occurred more often in infants who had perinatal asphyxia or CNS hemorrhage and who died. Insulin, glucagon, growth hormone and intravenous glucose infusion rates do not seem to be responsible for hyperglycemia. Hyperglycemia during intravenous infusion alone seems not to be due to glucose infusion per se, but to be due to concurrent critical conditions that are often associated with hyperglycemia.

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BLUNTED PANCREATIC POLYPEPTIDE RESPONSES IN CHILDREN WITH OBESITY OF PRADER-WILLI SYNDROME. W.B. Zipf, T.M. O'Dorisio, S. Cataland, and J. Sotos, Departments of Pediatrics, The Ohio State University, Columbus, Ohio.

Pancreatic polypeptide (PP) has been shown to mitigate the hyperphagia of genetically obese mice. These animals have low concentrations of PP. Patients with Prader-Willi Syndrome (P-W,S) are characterized by obesity and hyperphagia. Serum levels of PP, glucose, insulin and gastroinhibitory polypeptide (GIP) were obtained at 15 min. intervals for 180 min. after a 300 calorie meal in 7 pts with P-W,S and were compared to responses obtained in 10 normal and 13 obese controls. P-W,S had a mean (X) age of 12.3 years (y) (range 7-16y) and a X body weight (wt) 230% of ideal. Obese controls had a X age of 30.5y (17-44y) and a X body wt 200% of ideal. Normal controls had a X age of 34.1y (19-64y) and X body wt 106% of ideal. Basal and peak responses of glucose, insulin and GIP for pts with P-W,S were slightly but significantly less when compared to normal or obese controls. No significant differences in basal or peak PP responses were noted between normal and obese controls (basal, 103 $\pm$ 30 vs 111 $\pm$ 19; peak, 561 $\pm$ 140 vs 603 $\pm$ 107 pg/ml, X $\pm$ SE). All 7 pts with P-W,S had abnormal PP responses. Five failed to show significant PP release after the stimulation; one had a peak response to 130 pg/ml while the 7th pt had a supernormal response to > 2000 pg/ml. The 6 pts with low or no response had basal PP values of 62 $\pm$ 12 pg/ml and a X peak response of 78 $\pm$ 15 pg/ml. This observation of blunted PP response in a human model of hyperphagia and obesity suggests that PP may have a significant role in appetite control.

EPIDEMIOLOGY

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INCREASED PLANT INGESTIONS 1963-1977: A PROBLEM OF HOME SAFETY ASSOCIATED WITH THE BACK TO NATURE MOVEMENT. Sudhir K Anand, CR Drew Med Sch and ML King Hosp, Dept of Peds, Los Angeles

This study examines if the "back to nature" movement which started in the 1960s has resulted in increased buying of plants by the American public and if there has been a concomitant increase in plant ingestions. Sales of retail nursery stores for 1963, 67, 72, and 77 for the United States (US) recorded by the US Dept of Commerce census of retail trade are: (Amounts in thousands and corrected for inflation to 1967 constant \$):

	1963	1967	1972	1977
Amount	293,069	399,118	554,456	873,053
% of 1963	100	136	189	298

For the same years the incidence of plant ingestions (except mushrooms) in children < 5 years old recorded by National Clearing House for Poison Control Centers is:

	1963	1967	1972	1977
No. all ingestions	46,954	72,661	105,018	94,949
No. plant ingestions	1,054	2,336	4,759	11,819
% due to plants	2.2	3.2	4.5	12.4

These data indicate that the US public has been buying more plants which has altered the home environment. This fact, very likely has resulted in increased plant ingestions by young children. Improved public education regarding safe and poisonous plants may reduce the incidence of poisonous plant ingestions.