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Gonadotropin response to synthetic LHRH in normal children. Eighty five boys and forty nine girls aged 3 months to 16 years and without endocrine pathology received 25ug/m² LHRH (Hoechst) i.v. Boys and girls were distributed into six groups according to bone age (I: 3-6 mo, II: 6 mo-2 yr, III: 2-9 yr, IV: 9-11 yr) or to stage of puberty (V: P₂, VI: P₃). Serum LH and FSH were measured by radioimmunoassay methods. Basal LH and FSH levels increased gradually from group IV throughout puberty. The lowest LH responses were observed in group II and III and were similar in both sexes. In group I, the maximal LH value seen after LHRH was some 2 times higher in boys than in girls: the response in boys was similar to that found in both sexes of group IV, although basal levels were lower. The LH responses increased further with the stages of puberty; at all stages, the values after LHRH were higher in boys than in girls. In girls, the FSH response was the highest in group II, then it decreased steadily throughout infancy and stages P₂ and P₃. In boys, the FSH response was the lowest in group I and then increased steadily until stage P₃.

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Immediate and late effect of cranial irradiation on pituitary function in children.

The effect of cranial irradiation on pituitary function was studied prospectively. Plasma hGH and LH were determined, by radioimmunoassay, hourly from 8 pm to 7 am during natural sleep, prior to, immediately after cranial irradiation (2500 rads), and 6-24 months later, in 15 leukemic children aged 1.5-13 years. Seven normal children, aged 6-12 years, served as controls. hGH levels prior to irradiation were similar to those in the control group (mean value per hour 3.84±2.5 and 4.09±2.4 ng/ml respectively), a finding indicating normal pituitary function prior to irradiation. hGH levels were lower immediately after irradiation in children below the age of 5 years, and returned to normal 6-24 months later. The effect of irradiation on LH levels was uncertain and differently timed.

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Hypercalciuria of renal origin in diabetic children: incidence and response to indomethacin.

From 47 diabetic children 19 had a urinary calcium excretion 2 S.D. above the mean of 58 healthy children. In 13 of these 19 children the hypercalciuria persisted on follow-up and was independent of the glucose excretion. The hypercalciuric group compared to the normocalciuric diabetic group had a significantly higher pH and calcium/creatinine ratio in fasting urine samples, whereas the blood pH, bicarbonate, glucose and the serum calcium, ionised calcium, phosphate, alkaline phosphatase, iPTH, hCT, hGH levels were not different. In the hypercalciuric group an oral calcium load increased the TmP/GFR but not the urinary calcium/creatinine ratio and decreased the urinary cAMP excretion. Indomethacin lowered the calcium/creatinine ratio in fasting urine samples of the hypercalciuric, but not of the normocalciuric group without changing the sodium excretion.

It is suggested that prostaglandins contribute to a defective renal tubular function, causing hypercalciuria.

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Binding capacity of serum Testosterone estradiol Binding Globulin (TeBG) in obese children

Serum TeBG binding capacity was measured by polyacrylamide gel electrophoresis in 4 groups of obese subjects with weight excess ranging between 30 and 120% (20 prepubertal and 13 pubertal boys, 12 prepubertal and 11 pubertal girls). In prepubertal obese children, values were identical in boys ($2.52 \pm 1.04 \mu\text{g}\%$) and girls ($2.65 \pm 0.74 \mu\text{g}\%$), but significantly lower ($p < 0.01$) than in controls and negatively correlated to the percentage of obesity ($R = 0.51$, $p < 0.01$). At puberty, TeBG values remained unmodified in obese girls ($2.17 \pm 0.72 \mu\text{g}\%$) but decreased significantly ($p < 0.01$) in males ($1.39 \pm 0.49 \mu\text{g}\%$). In obese subjects plasma testosterone and estradiol were normal while plasma DHA was elevated and, in girls, correlated negatively with TeBG ($R = 0.66$, $p < 0.01$). The data suggest that TeBG is decreased in obesity independently of gonadal steroids levels but a relationship may be speculated with an increased production of adrenal androgens.

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Plasma ACTH and compound S response to a single oral dose of metyrapone.

Plasma ACTH (direct radioimmunoassay), cortisol (R.I.A.) and compound S (R.I.A.) were measured during insulin induced hypoglycemia or 3 to 7 h after a single oral dose (1 g/m²) of metyrapone taken at 7 a.m. or at midnight. The mean maximum ACTH concentration after hypoglycemia ($316 \pm 196 \text{ pg/ml}$) was comparable to the mean maximum 7 h ($507 \pm 344 \text{ pg/ml}$) and 3 h ($416 \pm 366 \text{ pg/ml}$) after metyrapone. Compound S rose to $6.2 \pm 3.9 \text{ ng/ml}$, plasma cortisol was suppressed ($6.2 \pm 2.8 \mu\text{g}\%$) 3 h after metyrapone and stimulated ($17.4 \pm 6.6 \mu\text{g}\%$) 7 h after the midnight dose. Within 70 children tested a diminished or absent response was observed in patients under cyproterone acetate after steroid therapy (2), in hypothalamo-pit. insufficiency (4) and anorexia nervosa (1). The single dose metyrapone test is a safe and reliable procedure for the assessment of the hypothalamo-pituitary adrenal system. If the test is based on the compound S and cortisol response alone, it does not require venipuncture.

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ANABOLIC EFFECT AND HORMONAL RESPONSE DUE TO PARENTERAL AMINO ACID NUTRITION OF PREMATURE INFANTS.

Since prognosis of neonatal diseases, especially RDS, in premature infants is impaired by nitrogen imbalances, we compared the effect of 3 amino acid mixtures (AA) on nitrogen balance, blood glucose (BG), plasma insulin (IRI), human growth hormone (hGH), glucagon and cortisol. 3 AA-mixtures were supplied i.v.: A) AA/sorbitol/xylitol each 5g/100ml, B) AA 5g/100ml, C) AA 5g /glucose 10g/100ml. RESULTS: In all 3 groups nitrogen balance immediately became positive. BG showed a mild elevation in group A, no change in B and a sharp rise up to 180mg% after 60 min. in C. IRI corresponded well with BG in A and B. C showed an excessive IRI-rise (104 uU/ml after 60 min.). Glucagon was not affected in A and C. In B there was a rise up to 273pg/ml after 60min. hGH was well stimulated in all groups with an average maximum of 43ng/ml after 60 min. Cortisol showed mean levels of 6ug/100ml without significant change. CONCLUSION: AA-infusions and re-actively high endog. hGH lead to pos. nitrogen balance. AA did not affect IRI, glucagon and cortisol reaction.