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JUVENILE HYPERTHYROIDISM (J H): THERAPEUTIC OPTIONS ACCORDING TO THE PREDICTION OF THE EVOLUTION. Iorcansky, S.; Grunero de Papendieck, L.; Belgorosky, A.; Rivarola, M.A.; Bergada, C. CEDIE-Division de Endocrinología Htal. Niños "Gutierrez", Bs. As., Argentina.

At present no Laboratory test is available to predict the evolution of J H (Graves' disease). A follow up to 2 to 14 y. was carried up in 59 patients aged 2 4/12 to 17 y. old ($\bar{X} \pm SD$: 9.4 \pm 3.9). They all received antithyroid drugs as initial treatment. Thirty six patients followed for 3 to 14 y., could be reevaluated with T₄, T₃ and TSH and/or TRH after treatment in at least 2 occasions: at short-term (ST: 1-2 y. post onset of treatment) and at long-term (LT: more than 3 y.; $\bar{X} \pm SD$: 6.23 \pm 3.3). Twenty three patients (64%) remained hyperthyroid (Hper) between ST and LT and 9(25%) hypo or euthyroid (Hpo/Eu) between ST and LT; only 4 (11%) changed from Hper at ST to Hpo/Eu at LT. Thus, 89% did not modify their thyroid function between ST and LT. The period of the evolution from Hper to Hpo/Eu showed two distinct populations, one with a $\bar{X} \pm SD$ of 17.3 \pm 3.8 months and another with $\bar{X} \pm SD$ of 9.4 \pm 2.5 y. It is concluded that evaluation of thyroid function at ST is useful to predict their status at LT since 90% of patients showed no variations. Since patients who changed thyroid function from Hper to Hpo/Eu at ST did it in \bar{X} 17.3 months it is advisable to wait up to this time to select another therapeutic options. If Hper persists the possibility of I 131 administration should be considered to avoid the excessively long treatment required by the unrelenting course of this disease.

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GROWTH AND GROWTH HORMONE SECRETION IN CHILDREN WITH RENAL TRANSPLANTATION. Domene, H.M.; Jasper, H.; Ferraris, J.R. División de Endocrinología, CEDIE, Htal. de Niños "R.Gutierrez" y Sección Nefrología Pediátrica, Htal. Italiano, Buenos Aires, Argentina.

Impaired growth is a major problem in children with renal transplantation (Tx). Poor allograft function and corticosteroids administration have been mentioned as probable causes of decreased statural growth. The aim of the present study was: 1) to determine the effect on height velocity (HV) of a reduced methylprednisone (MP) dose and 2) to evaluate growth hormone (hGH) dynamics by studying the spontaneous 24 hours secretion. We studied 7 Tx patients (chronological age 8.6 to 15.2 years), with serum creatinine from 0.5 to 0.8mg/dl, receiving MP at a doses of 0.23 \pm 0.02 mg/kg/day ($x \pm sd$) with a previous HV of 2.58 \pm 1.14 cm/yr. After one year with a lower MP dose (0.17 \pm 0.01 mg/kg/day), HV was 3.42 \pm 1.94 cm/yr. (n=7) and 2.39 \pm 1.03 cm/yr. in 4 patients who remained prepubertal during that year. In these 4 patients mean hGH concentration was 2.59 \pm 0.98 ng/ml, no different from a normal control group: 2.78 \pm 0.76 ng/ml (n=3). Not hGH peaks > 5 ng/ml were detected in 2 of these patients, while normal controls showed 1 to 4 peaks. In conclusion a lower MP dose did not improve HV in prepubertal patients with Tx and decreased growth could be related, in some children, to an impaired hGH secretion.

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GROWTH HORMONE DEFICIENCY TREATMENT WITH DNA RECOMBINANT MET-HUMAN GROWTH HORMONE (MET-rhGH). Heinrich, J.J.; Martinez, A.S.; Domene, H.; Jasper, H.; Miras, M.; Agrelo, F.; Bergada, C. CEDIE, Div. de Endocrinología, Hosp. de Niños "R.Gutierrez" y Servicio de Endocrinología, Hosp. de Niños de Córdoba, Bs. As., Argentina.

Eighteen hypopituitary children, 6 girls and 12 boys, between 2 and 15 years of age, were treated for eighteen months with met-rhGH (Somatomorm). Dosage used were 0.5 IU/kg/week by daily subcutaneous (s.c.) or three times a week intramuscular (i.m.) injections. Two of the children were twins with congenital isolated growth hormone deficiency of the type 1 A. One patient entered puberty six months after the treatment was started. These three children were not included in the growth evaluation.

ROUTE	n	AGE years	BONE-AGE-RUS			GROWTH VELOCITY cm/year			
			start	18m	before	0-6m	6-12m	12-18m	
	7	8.8	4.65	7.50	3.33	12.84	9.50	8.48	
s.c.	8		2.39	2.44	2.64	2.14	3.19	1.75	2.99
	8		8.94	6.11	8.84	3.78	10.84	7.33	6.49
i.m.	7		1.99	2.40	2.20	1.38	3.01	1.09	2.79

Antibodies against met-rhGH developed in 6/8 and 3/8 patients injected by s.c. and i.m. route respectively, but titers were low and did not impair growth velocity. Both twins with type 1 A GH developed early very high antibody titers. Growth velocity fell in one but not in the other. Although no significant differences in the growth promotion effect were noted between both treatment groups antibody development was more frequently seen after s.c. met-rhGH administration. All patients increased significantly the growth velocity and final height prognosis, without local or systemic side effects.

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LACK OF CORRELATION OF TOTAL ESTRADIOL (To E2) AND NON-SHBG-BOUND E2 (non-SHBG-b E2) WITH GROWTH VELOCITY DURING PUBERTY IN NORMAL BOYS. Belgorosky, A.; Martinez, A.; Escobar, M.E.; Heinrich, J.J.; Rivarola, M.A. CEDIE. Hospital de Niños, Buenos Aires, Argentina.

Recent evidence suggests that normal growth during male puberty depends on the combined effect of testosterone (T) and growth hormone while the role of estrogens, if any, has not been established. Correlations between growth velocity (GV) and serum total T (To T), non-SHBG-b T, To E2 or non-SHBG-b E2 was made in 16 normal boys (mean \pm SD chronological age, 14.1 \pm 1.4, mean \pm SD bone age 12.3 \pm 1.64 years) in Tanner's stages II-IV of genital development. GV was evaluated in a 6-months-period but growth was followed up for at least 1 year. None of the subjects had overcome their peak height velocity as judged by 3 consecutive measurements. T and E2 non-SHBG-b fractions were calculated by a mathematical model based on the law of mass action. Mean \pm SD To T and non-SHBG-b T were 222 \pm 48 and 75 \pm 22ng/dl respectively while To E2 and non-SHBG-b E2 were 18.1 \pm 4.4 and 9.07 \pm 2.88 pg/ml respectively. To T and non-SHBG-b T showed a highly significant correlation with GV ($p < 0.001$) while no significant correlation was found between To E2 or non-SHBG-b E2 and GV. These studies suggest that serum E2 does not play a role in the pubertal growth spurt of boys.

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MAMMARY GLAND DEVELOPMENT UNDER THE EFFECT OF A PLACENTAL PROTEIN. Calaff, G.; Capurro, M.T.; Beas, F. Universidad Metropolitana de Cs. de la Educación. Centro de Investigaciones Materno Infantil, Hospital Paula Jaraquemada, Facultad Medicina, Universidad de Chile, Santiago, Chile.

A protein from human placenta, the Uterotrophic Placental Protein (UTPH) was isolated in our lab (Beas & Flores, 1969). It evoked rat sac stimulation in pigeons, inhibited mammary growth produced by estradiol (E) and progesterone (P) in ovariectomized rats and increased DNA synthesis in 5 day organ cultures of mouse mammary gland. The objective of this work was to analyze the effect of UTPH on mammary gland development in intact virgin Balb/c mice injected during 8 days. There were four experimental groups: (I) Control: 0.3 ml saline sol., (II) E + P: 0.4 ug/0.3 ml and 0.8 mg/0.3 ml respectively, (III) E + P + UTPH: 200 ug/0.3 ml and (IV) UTPH. Results on wholemount mammary gland indicated that there was: a) not significant difference among groups vs control in the number of mammary gland ducts. b) a significant increase in number of terminal end buds (TEB) in group II vs I ($p < 0.0005$) and III ($p < 0.0005$) or II vs IV ($p < 0.0005$). c) a significant increase in number of alveoli of group II ($p < 0.0005$) and III ($p < 0.0005$) vs control. We can conclude from these studies that UTPH inhibited E + P action on TEB development while had no effect on ducts and alveoli.

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PULMONARY FINDINGS IN POSTMORTEM EXAMINATIONS OF PEDIATRIC ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS). Adams, J.A.; Birriel, J.A.; Saldana, M.J.; Vernon, D. Department of Pediatrics, University of Miami School of Medicine, Miami, Fl., U.S.A.

A retrospective study of autopsies of children with the diagnosis of AIDS was done. From March 1983 to September 1986 a total of 24 autopsies were done in children with AIDS at the University of Miami/Jackson Memorial Hospital. Twenty three cases were reviewed as to the pulmonary pathology. In 21 cases the primary cause of death was pulmonary. Of these, 12 (57%) had a Gram negative (G-) bacterial pathogen alone or in combination with lymphoid interstitial pneumonia (LIP) or Pneumocystis carinii (PCP). Of the 21 cases 8 (38%) had LIP. Four of the 8 also had G- pneumonia (Pseudomonas aeruginosa, E.coli). One case of LIP with Staphylococcus aureus pneumonia, 2 cases of LIP and Cytomegalovirus (CMV), and one case of LIP and PCP. The histological changes of diffuse alveolar damage and barotrauma correlate well clinically with the number of days on ventilatory support, and oxygen concentration utilized. Nine of the patients expired as a consequence of LIP alone. Our data indicates: # 1 Multiple pathogens appear to play an important role in end stage respiratory failure in these children. # 2 LIP alone does not appear to be a cause of end stage respiratory failure, however, LIP alone with either viral or bacterial pathogens account for 33% of the pulmonary pathologic diagnosis. # 3 Bacterial pathogens play an important role in end stage respiratory failure in AIDS. # 4 The use of systemic steroids for the treatment of LIP in end stage, would be contraindicated in light of the mixed pulmonary pathogens.