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HYPOTHALAMIC-PITUITARY FUNCTION IN HYPO-TBG-NEMIA (Ho-TBG). CA. Longui; JR. Coelho-Neto; MSO. Shimizu; TM. Carvalho; BF. Cesar; MAM. Aguiar; CP. Souza; AG. Rodriguez; TSS. Lins; BJ. Schmindt. APAE de Sao Paulo - Sao Paulo - Brasil.

Neonatal T4 detected 128 cases with TBG < 5 ug/ml; 65 were followed-up; mean CA=1.1y; (M=61; F=4). In 43 cases the Z score of stature at the first and last observation were=0.08 (1.3) and 0.74 (0.9), respectively. No signs of hypothyroidism were observed. In 65 cases we found a basal T4=1.8 (0.6); free T4 (DPC)=0.5 (0.3); free T4 (BAXTER)= 1.3 (0.4); TSH= 2.9 (1.7). A TRH test (7 ug/kg) was performed in 12 cases (table 1):

	BASAL	20'	40'	60'	90'
HO-TBG n=4(I)	1.7 (0.5)	13.5(0.7)	12.6(1.3)	10.2(2.1)	8.3(2.2)
HO-TBG n=8(II)	** 6.1(1.3)	* 37.0(4.7)	* 32.7(3.9)	* 27.6(3.9)	* 21.7(3.5)
CONTROL	2.7(1.4)	13.8(4.0)	10.0(4.0)	10.0(5.0)	7.9(3.6)

Ho-TBG patients showed normal growth and no sings of hypothyroidism. Free T4(DPC) suffered interference by HO-TBG state. Some increased basal values and TRH hyperresponsive pattern suggest that pituitary negative feedback could be altered in patients with Ho-TBG.t (student) test: * p<0.0005; ** p<0.005.

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NOCTURNAL TSH SURGE IN CHILDREN WITH HYPOTHALAMIC PITUITARY DISORDERS AND PRIMARY HYPOTHYROIDISM. L. Gruñeiro, A. Chiesa, A. Martinez, J. Heinrich, C. Bergadá. Endocrinologia, Hosp. de Niños "R. Gurierrez", Buenos Aires, Argentina.

The nocturnal TSH surge (△TSHn) was studied in 23 control children (group I), 33 with hypothalamic pituitary disease (group II) and 12 patients with primary hypothyroidism, 10 with mild (group III)and 2 with overt hypothyroidism. TSH was measured by IRMA; TSH was calculated as the % increase in the night TSH over day TSH. The results were (X±SE).

Group	Day TSH(uU/ml)	Night TSH(uU/ml)	TSHn(%)	Peak TSH-TRH
I	1.57 ± 0.2	4.12 ± 0.4	177.48 ± 20.6	14.5 ± 1.1
II	2.11 ± 0.2	3.18 ± 0.3	94.37 ± 16.7	19.2 ± 1.8
III	3.15 ± 0.8	6.06 ± 1.1	178.79 ± 38.7	35.4 ± 2.6

*p > 0.01 vs group I.A△TSHn was found in 16/33 patients of group II (48.5%), in 10 of them the TRH test was normal; in the remaining 17 with △TSHnpresent the TRH test was normal in 9. Group II was divided into patients who were hypothyroid FT4 < 0.7 ng/dl (group III) or euthyroid FT4>0.8 ng/dl (group II2).II1, n=9,△ TSHn:50.3±15.3%, deficient in 7/9 (77%), TRH test was abnormal in 5/9 (56%); II2, n=24, △ TSHn: 110.9 ± 21.5%, deficient in 9/24 (37.5%), TRH test abnormal in 10/24 (62.5%). The TSHn was found deficient in 77% of patients with central hypothyroidism, normal in mild hypothyroidism but absent in overt hypothyroidism. We conclude that△TSHn did not provide a complete discrimination between euthyroidism and central hypothyroidism. The△TSHn plays and important role in the pathophysiology of central hypothyroidism but many others factors may be implicated.

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HYPOTHALAMIC-PITUITARY.THYROID ABNORMALITIES IN RENAL TRANSPLANT CHILDREN BEFORE AND AFTER DEFLAZACORT THERAPY. T. Pasqualini, P. Fainstein-Day, R. Gutman, M. Balzaretti, A. Eymann, J. Ferraris. Departamento de Pediatria and Serv. de Endocrinologia, Hosp. Italiano, Buenos Aires, Argentina.

In chronic renal failure we found central hypothyroidism (J Pediatric 1991; 118: 873), so we attempted to study thyroid hormone leveles, TSH/TRH response and the circadian TSH pattern in 9 children (9-16 years old) after renal transplantation (Tx), before and a year after substitution of methylprednisione (MP) with D. D is an oxazoline compound derived from prednisolone with similar antiinflammatory actions but fewer side effects. Renal fuction remained stable. Mean concentrations of T3 (2.3 ± 0.3 vs 2.3 ± 0.4 nmol/L), total T4 (112 ± 26 vs 110 ± 19 nmol/L) and basal TSH (3.4 ± 0.8 vs 2.8 0.9 mU/L) were normal; mean free T4 (13.4 ± 3.5 vs 14.2 ± 2.4 pmol/L) was low (p<0.01). Five patients on MP and 5 on D had deficient TSH increment to TRH; 3 out of patients on MP and 1 out of 5 on D had a nocturnal TSH surge below the normal range (47% - 300%). The association of low free T4, deficient TSH/TRH response and impaired nocturnal TSH surge, support the hypothesis that some renal transplant patients have central hypothyroidism. The circadian TSH pattern seems to improve on D therapy.

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JUVENILE AUTOIMMUNE THYROID DISEASE: CYTOLOGY AND IMMUNOPHENOTYPE OF THYROID INFILTRATING CELLS AND THEIR SEROLOGIC CORRELATION. (PART II). V. Herzovich, J. Goldberg, J. Rossi y S. Iorcansky. Serv. Endocrinol. Patol. Hosp. Garrahan, Bs.As., Argentina.

The influence of infiltrating lymphocytic immunophenotype (Part I) and cytological findings on samples obtained by fine needle aspiration biopsy (FNABT) were correlated with the presence (+) and titers of circulating thyroid antibodies (Ab) in 43 patients with autoimmune thyroid disease (chronic Lymphocitic Thyroiditis= CLT, n=23 and Graves Disease= GD, n=20). Ab (Antimicrosomal= MiAb and Antithyroglobulin= TgAb)were determined by hemagglutination. CLT:Cytology: 23/23 pts had CLT MiAb+: 17/23 (74%); Titers:Range 1/400 - 1/25.600. TgAb+: 5/23(22%)Titers:range 1/100 -1/600. GD: Cytology: 7/20 pts were CLT, 12/20 were normal and 1/20 adenomatous goiter (AB). Antibodies were determined in 18/20: MiaAb+: 15/18 (83%) Titers: range 1/400 - 1/409.600; TgAb+:10/18 (55%) Titers: range 1/100 to 1/6400. 6/7 pts with CLT cytology had MiAb+ and 4/7 TgAb+. 9/11 pts (10 with normal cytology and 1 A. Goiter) had MiAb ranged 1/400 - 1/102.400 and in 6/11 had TgAb ranged 1/100-1/6400. Less serological expression were seen in CLT (were) the prevalence of intrathyroid T-Cells and other sings of cellular aggression (Part I) would suggest mainly a cellular immune reaction. 2) Higher titers of both Ab were found in GD, Together with the prevalence of B lymphocytes among the infiltrating cells (Part I) would sustain mainly a humoral mechanism of autoimmunity. 3) Normal cytology in GD along with high titers of Ab would suggest antigen presentation in lymphoid organs (ei.nodes) but not within the thyroid itself. Thyroid gland would be a "passive captive" of specific events of the whole immune system, as Volpé has suggested.

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MOLECULAR ANALYSIS OF Y CHROMOSOME IN TESTICULAR DIFFERENTATIATION. S. Copelli, A. Goldberg, A. Billerbeck, D. Damiani, V. Varela, C. Bergadá and H. Targovnik. Centro de Investigaciones Endocinológicas. Hospital de Niños R. Gutierrez. Buenos Aires. Cátedra Genética y Biologia Molecuar, FF y B., Universidad de Buenos Aires. Lab. de Transplantes, FM, Universidad de Sao Paulo.

The aim of our work was to investigate the testicular differentiation mechanism in true hermaphrodites with karyotype XX in order to study the SRY gene and Y heterochromatic region (Yq12-Yqter) by PCR (Polymerase chain Reaction) in these patients and another gonadal patologies. A total of 10 patients were studied: 8 Trues Hermaphrodites (TH): 6 46,XX, 1 45,X/46,XY and 1 45,X/46XY/46,Xinv (Y); a female with Swyer's syndrome 46,XY and 1 Dysgenetic Male pseudohermaphrodite 46,Xr (Y). The SRY gene was present in TH X/XY, X/XY/Xinv (Y) and DMP Xr (Y); it was absent in 6 XXTH and XY female. The Y heterochromatic region was amplified in TH X/XY, X/XY/Xinv (Y) and XY female, and without amplification in THXX and DMP Xr (Y). These results suggest that XXTH could have arisen by an autosomic gene involved in sex reversal or the SRY gene could have been lost in the early stages of embrionary development. The absence of testes and female phenotype in the XY female could be explained by the absence of the SRY gene because of an Y chromosome deletion in the paternal gametogenesis. These data illustaste the usefulness of PCR in the molecular analysis of these patients.

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STEROID BIOSYNTHESIS IN PATIENTS WITH MALE PSUDOHERMAFRODITISM. STUDIES PERFORMED IN LEYDIG CELL MESENCHYMAL PRECURSORS IN CULTURE. E. Pellizzari, S. Ayuso, S. Campo, H. Chemes, E. Boulgourdjian, C. Bergadá, S.Cigorraga. Centro de Investigaciones Endocrinol. Hosp. de Niños. R. Gutierrez. Buenos Aires, Argentina.

Among the cause of male pseudohermafroditism(MPH),disorders of testicular function, which include enzymatic defects in the testosterone (T)biosynthesis, have been demonstrated. In a 8 1/2 years old patient with MPH (MG, 46 XY), with ambiguous genitalia and raised as a female, plasmatic levels of the following steroids were determined: Pregnenolone (P5), Progesterone (P4), Androstenedione (☖ 4), 17∝hydroxyprogesterone (17∝OHP4) and T. Values were found in the normal prepubertal range. Lack of response to acute hCG stimulation for these steroids was observed. After gonadectomy, Leydig cell mesenchymal precursors were isolated as previously described (Biol.Reprod.46:793, 1992).Cells were cultured for 6 days and T,P5,P4 17OHP4, ☖ 4 and dehydroepiandrosterone (DHA) levels were determined by RIA in the culture medium. Results were compared with those found in two other patients (PM, VT) bearing the androgen insensitivity syndrome.Levels of T (pg/h/ugDNA) were as follows, MG:0.35±0.06, PM:101±10 and VT: 141±15. Similar results in FSH or hCG stimulated cultures were obtained. The overall steroid production in the three patients was: MG: 108, PM; 184 and VT: 319 (pg/h/ugDNA). A marked modification of T/☖ 4 relationship was observed in MG (MG: 0.03, PM: 64 and VT: 3.97). These results suggest that MG male pseudohermafroditism is due to 17β hydroxysteroid dehydrogenase deficiency.This deficiency could not be demonstrated at the peripheral level but it was evident in Leydig cell mesenchymal precursors in culture.