3 «C ANDROSTANEDIOL GLUCURONIDE AS A MARKER OF ANDROGENIC ACTIVITY IN GIRLS WITH PREMATURE PUBARCHE. M. Gryngarten, ME. Escobar, S. Campo, S. Ayuso, P. Bedecarrás, C. Bergadá. CEDIE. Div. de Endocri-nol. Hosp. de Niños R. Gutierrez. Bs.As., Argentina. Previous reports have postulated that 3 androstanediol glucuro-nide(30C AG) constitutes a marker of the 5 reductase activity in peripheral tissues. We studied the serum levels of androgens and their metabolites in 20 girls with premature pubarche (PP), EC X: $(\Delta + 2, 3)$ years, and in a control group of 12 girls, 5 of them prepubertal and the other 7 pubertal. 17 hidroxiprogesterone (170HP), Androstenedione ($\Delta + A$), dehidroepiandrosterone sulfate (DS), tes-tosterone (T) and 3 AG were determined by RLA. All patients showed public hair Tanner grade II-TII. Besides public hair, 13 girls had one or more associated androgenic signs like acne; or face, trunk or limbs hair. Serum T and 170HP levels were in prepubertal range in girls with PP except in one that showed basal 170HP of 2 ng/ml and a response to ACTH stimulation of 20, which lead to a diagnosis of non classical 210Hlase deficiency.

		3 oC AG	3	AG/∆4A	
Prepubertal C	n=5	2.08 ± 1.0	1.47	/ <u>+</u> 0.79	
Pubertal C	n=7	2.96 + 1.6	1 p<0.05 1.12	2 ± 0.61	
PP	n=20	6.89 + 4.6	5 2.23	3 ± 1.57	
PP with androgenic	n=13	7.40 ± 4.7	0 2.36	± 1.88	p<0.05

P without androgenic n=7 5.95 ± 4.50 1.90 ± 0.75 The elevated 30CAG levels found in PP agree with the pubertal levels of its precursors $\triangle 4A$ and DS. The high 3 $\propto AG/4A$ ratio found in PP associated with other androgenic signs suggests and enhanced peripheral 5 reductase activity in those patients.

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SERUM LH AND FSH DURING THE FIRST TRIMESTER OF LIFE IN NORMAL (C)

SERUM LH AND FSH DURING THE FIRST TRIMESTER OF LIFE IN NORMAL (C) CHILDREN AND IN AMBIGUOUS GENITALIA. S. Chahin, E. Chaler, M. Maceiras, A. Belgorosky, MA. Rivarola. Servicio de Endocrinologia. Hosp. J.P. Garrahan, Bs.As., Argentina. Little information is available on serum LH and FSH levels during the first 3 months of life measured by a RIA which utilizes a monoclonal antibody specific for & LH subnit. In order to evaluate the gonadotropic profile and the maturation of the hypothalamo-pituitary-gonadal axis, serum LH and FSH were determined by IRMA in 55 control childrens are 590 dayrold the map chronological are 55 control childrens, age 5-90 day-old, the mean chronological age $(X\pm SD)$ was 27.9±15 for males (M) and 26.8±23.2 days for females (ALGO) was 27.5215 for makes (n) and 20.525.2 days for lemates (F). In 5 F with congenital adrenal hyperplasia (CAH), mean age 23.8 \pm 21.4 days and in 7 M babies with 46XY ambiguous genitalia (serum testosterone 4.92 \pm 2.56 nmol/L, good clinical response to androgens), mean age 15.3 \pm 10.5 days. Serum levels of LH in normal androgens), mean age 15.3 \pm 10.5 days. Serum levels of LH in normal females were significantly lower (1.07 \pm 1.80 and 3.85 \pm 3.4 U/L) and those of FSH significantly higher (7.68 \pm 7.12 and 2.82 \pm 2.75 U/L) than in normal M,respectively.No correlation was found between serum LH or FSH with age(5-90 days old) in C F or M. Girls with CAH had serum FSH (0.64 \pm 1.05 U/L) significantly lower than F C (p<0.05)but serum LH(0.20 \pm 0.26 U/L) was not different. In boys with 46,XY ambiguous genitalia, serum LH (2.09 \pm 2.17) and serum FSH (2.28 \pm 2.31) were not significantly different from M C. It is concluded that, 1) serum LH and FSH do not change with age during the first trimester, 2) serum LH is higher in M and serum FSH is higher in F. and 3) inhibitory effect of adrenal androgene is present in (in F, and 3) inhibitory effect of adrenal androgens is present CAH at this age.

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INHIBIN GENERATION IN CRYPTORCHID (CRI) BOYS UNDER 4 YEARS OF LIFE. Unidade de Gonadas en Intersexo - FMUSP - Sao Paulo-Brasil.Fundation de Recherche en Hormologie-Fresnes- France.

tion de Recherche en Hormologie-Fresnes- France. Thirty cryptorchid boys with pre-scrotal testes were divided in two groups:Group 1 (3 bil/12 unil)received hCG (750U/m2/dose)+hHG (100U/m2/dose) 2 times weekly, during 6 weeks; Group II (5 bil/10 unil) received only hCG. Surgical correction was necessary in 28 cases, 22 of wich had a testicular biopsy and subsequent hystomorp-hological analysis. Results were obtained in basal condition and at 3rd. and 6th. weeks of treatment. Peak values are showed:

	LH	FSH	TESTO	INHIBIN	SPERM	At(u2)
UNI	39(10)	7(1.4)	370(206)	450(228)	T=67(44)	T=222(18)
					C=11(10)	C=194(30)
G-1						
BIL	39(16)	6(1.5)	197(122)	310(269)	24(20)	198(20)
UNI	36(14)	4(0.6)	441(290)	257(183)	T=26(18)	T=238(20)
					C=14(9)	C=193(23)

G-11 BIL 44(26) 4(0.5) 515(330) 438(199) 10(7) 207(35) T=topic C=cryptorch. Sperm=spermatogonia/50 tubes At=tubular area.LH(mUI/ml) FSH(mUI/ml) Testosterone (ng/dl) inhibin (UI/L). There was no advantage in hHG association. The hormonal level fell between Srd 6th weeks (down-regulation). There was no correlation between inhibin levels and testosterone, spermatogonia or tubular area. Tubular area and number of spermatogonias were decreased on the affected side in relation to the topic testes and in bilateral

cases in relation to the normal patterns.

IGF-I AND IGF-II IMMUNOREACTIVITY IN CONDITIONED MEDIA FROM PRIMARY CULTURES OF RAT SERTOLI CELLS. H. Jasper, S. Cigorraga, Endocrino-

ygy, "R.Gutierrez" Children's, Hospital, Buenos Aires, Argentina. IFG-I production by rat Sertoli cells has been reported, but evidences of IGF-II production are lacking. Trying to prove it, we measured IGF-II (as well as IGF-I) immunoreactivity in conditioned media from primary cultures of Sertoli cells from 8 day-old rats.After isolation the cells were cultured for 24 hrs in a 1: 1 mixture of mediums F12: DME with 10 ug/ml transferrin, 5 ug/ml insulin, 5 ug/ml vitamin E, 4 ug/ml hidrocortisone, 100 U/ml peni-cillin, 2.5 ug/ml amphotericin B. The cells were then cultured for 48 hs in medium without insulin and immediately afterwards the conditioned media and the cells were collected and analyzed separa-tely. The RIAs employed human recombinant IGF-I and IGF-II for standard and tracer, a polyclonal anti IGF-I antibody and a mono-clonal anti IGF-II antibody. To avoid interference by IGF binding proteins previous to RIAs the conditioned media were extracted with a mixture of formic acid-Tween 20-acetone. With the same purpose 8ng/tube IGF-I was added to the IGF-II RIA buffer. Recovery of added 125I IGF-II after the extraction procedure was 101.2 ± 3.9 % (X SD, n=4). Both IGFs were present in the conditioned media. Serial ilutions of the wxtracted media were parallel to the standard <u>+</u> SD, n=4) dilutions curves. IGF-I ommunoreactivity in the condictioned media was 84 ± 20 pg/ug DNA while IGF-II immunoreactivity was 802 ± 209 pg/ug DNA(X-SEM, n=4). Our results tend to confirme IGF-II production by rat Sertoli cells.

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HORMONAL PROFILE OF CATCH UP GROWTH (CUG) IN SMALL FOR GESTATIONAL AGE (SGA) INFANTS. H. García, C. Henriquez, F. Beas, E. Fernandez,
G. Iñiguez, E. Trabucco, MA. Boric, F. Barrera, R. Rubio and F. Cassorla, IDIMI. U. de Chile, Medical School and Hospital San Borja Arriarán, Santiago, Chile. Our purpose was to compare the endocrine profile of idiopatic SGA

infants who grow rapidly during their first months of life and those who fail to do so,36 SGA infants(17 boys and 19 girls) were followed monthly since birth, measuring the plasma levels of s ral hormones, at 0 and 3 months. The average birth weight Sevewas The infinite set of the set of t month(-1.S.D.). Hormonal levels at three months of age are shown in the table.

INSULIN IGF-T TESTOST. PRI. Τ3 ሞ4 122 UUT/ml ng/ml ng/ml ng/ml ng/dl ug/dl ng/ml 1. 17.8±23 92±48 19±10.0 223±25 11.6±1.5 0.9±0.8 2. 8.3±7.1 95±44 23±15.6 215±49 10.5±2.6 0.7±0.6 3. 12.0±16 57±22 17±10.6 222±30 11.5±1.1 0.2 CONCLUSIONS: We observed CUG in 30% of these infants pg/ml 76.4<u>+</u>37 44.1<u>+</u>30 59.3+40 CONCLUSIONS: We observed CUG in 30% of these infants during the first three months of life. We found significant differences in IGF-I levels between groups 1 and 2 vs groups 3. These results suggest that IGF-I may play a role in CUG.

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JUVENILE AUTOIMMUNE THYROID DISEASE (ATD). FINE NEEDLE ASPIRATION (FNABT) AND IMMUNOAMARCATION (PART I). J. Goldberg, Herzovich, J. Rossi, S. Iorcansky. Serv. Patol. y Endocrinol. Hosp. Garrahan. Bs.As. Argentina.

FNABT was done in patients with ATD (Chronic Lymphocytic Thyroi-ditis= CLT and Graves'Disease= GD), and processed according to the mothod of Hayry at al., applied to follow up transplanted organs. Citospin samples were stained with May Grunewald-Giemsa, and HLA-DR, CD25. CD4, CD8, CD3, CD11 and surface immunoglobulines were DR, CD25, CD4, CD8, CD3, CD11 and surface immunoglobulines were detected by immunofluorescence. In transplanted kidneys, a correcdetected by immunofluorescence. In transplanted kidneys, a corrected increment (inflamatory INDEX) (CI)3,5 indicates immunological activation. We analized thyroid samples, obtaining the CI, and the phenotype of infiltrating cells. 56 FNABT were perfomed on 43 pts. (CLT n=23 and GD n=20),followed up from 1 y. to 4,5y. They were divided in 2 gr., according to their thyroid status.Gr.I:CLT n=29 and Gr.II.GD n=27. Results: ($\frac{1}{2}$ +GD). <u>Gr.I</u>: CI: 6.3 \pm 0.97; B-cells: 33.9 \pm 10.5; T-cells: 61.2 \pm 11.4;CD4:63.1 \pm 6.2;CD 8:34.4 \pm 6.6. CD4/CD8 ratio: 1.9 \pm 0.5 HLA-DR expression was found on 56.6 \pm 18.8% of the follicular cells and CD25 was expressed in 27/29 pts. <u>Gr.II</u>:(CI 4.2 \pm 1.3(Gr.I vs II = p<0.001) B-cells: 58.5 \pm 16.2 (p<0.001) T-cells: 39.6 \pm 17.1 (p<0.02) CD4/CD8 ratio: 1.6 \pm 0.7 (p<0.02) HA-DR was expressed on 2.6.6 \pm 17.3 CD8 ratio: 1.6 ± 0.7 (p<0.02). HLA-DR was expressed on 26.6 ± 17.3 (p<0.001) of follicular cells and CD25 was positive in 7/16 pts.All (Jession) of Hollicular certs and CD25 was positive in 7/18 pDS.AHI CLT showed typical cyclological aspect. In GD 7/20 cytology 12/20 were normal and 1/20 adenomatous goiter (AG).The CI5.3,the expres-sion degree of HLA-DR on follicular cells, as well as the expres-sion of CD 25 on lymphocytes are markers of immune activation.TLC could be considered as a form of rejection, in which cellular ag-gression would play an important role, being in GD mainly humoral.