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HORMONAL RESPONSE OF THE NEONATAL THYROID TO CESAREAN SECTION (CS) DELIVERY. Young M. Kim, Simon Halevy, Mehmet Y. Dincsoy, Mamerto Garcia, Mariano Castro-Magana. (Spon. by Platon J. Collipp). Health Sciences Center, SUNY at Stony Brook, Nassau County Medical Center, Departments of Pediatrics and Anesthesiology, East Meadow, NY.

Perinatal stress may induce a surge of hormonal responses which includes the thyroid hormones. Information is inadequate with regard to the surge of thyroid hormones at the time of delivery as it relates to the type of CS. We studied 33 mother-infant pairs who had elective (E) or primary (P) CS. The infants studied had ($\bar{x}$) gestational age of 38.0 wk. All thyroid hormone measurements made on venous cord blood obtained immediately after delivery on two CS groups follow ($\bar{x} \pm \text{SEM}$):

	n	TSH ($\mu\text{u/ml}$)	n	T4 (μg)	n	T3 (ng%)	n	rT3 (ng%)
CS-E	11	5.2 $\pm$ 1.3 *	23	9.9 $\pm$ 0.3	23	45 $\pm$ 4	22	1305 $\pm$ 66
CS-P	7	15.9 $\pm$ 2.6	10	11.2 $\pm$ 1.0	9	52 $\pm$ 4	8	1375 $\pm$ 102

t test: *p<0.005

The following is comparative serum TSH concentrations ($\bar{x} \pm \text{SEM}$) of subgroups of infants who underwent CS delivery with and without preceding labor:

TSH ($\mu\text{u/ml}$)	(n)	CS Elective	(n)	CS Primary	p
With Labor	7	4.5 $\pm$ 1.59 *	na	10.8 $\pm$ 1.27	<0.02
Without Labor	4	6.5 $\pm$ 2.55	5	17.9 $\pm$ 3.22	<0.05

*p<0.005

The data suggest that labor may suppress TSH and the events surrounding the primary CS stimulates this surge at the time of delivery. The mechanism operative is not clear and needs further study.

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LEUCOCYTE LOCOMOTION IN PATIENTS WITH TURNER'S SYNDROME. Roberto R. Kretschmer, Elisa Vega, Lourdes Nungaray, Martha Lopez-Osuna, and Fabio Salamanca. Unidades de Inmunología y Genética. Unidad de Investigación Biomédica del C.M.N.-I.M.S.S. México, D. F. México.

Leucocyte locomotion abnormalities have been found in chromosomopathies such as Down's, Cri-du-chat and Edwards syndromes, in all of which a variable increase in susceptibility to infections has been described as well. We evaluated the peripheral blood leucocyte (polymorphonuclear (PMN) and monocyte (MP)) locomotion (random mobility, chemokinesis and chemotaxis) in 13 infection-free patients with Turner's syndrome and compared it to that of 10 age-matched healthy girls. Leucocytes were obtained by density gradient centrifugation and the three locomotion varieties were assayed using double-filtered Boyden chambers and zymosan activated serum as attractant. Random mobility, chemokinesis and chemotaxis of MP from patients with Turner's syndrome were comparable to those of controls. So were random mobility and chemokinesis of PMN. Chemotaxis of PMN from patients with Turner's syndrome was significantly ($p < 0.002$) diminished (57 ± 19 PMN/hpf, mean $\pm$ SEM) when compared to controls (93 ± 56 PMN/hpf). These results suggest an intrinsic PMN chemotactic defect in patients with Turner's syndrome that does not translate into an increase in the susceptibility to infections.

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BRAIN STEM AUDITORY EVOKED POTENTIALS (BAEP) IN CONGENITALLY HYPOTHYROID CHILDREN TREATED WITH THYROID HORMONES. E. Laureau(1), R. Hébert (2), M. Vanasse (1), J. Letarte (1), J. Dussault (2), J. Glorieux (1), Université de Montréal, Centre de Recherches Pédiatrique, Hôpital Ste-Justine, Department of Pediatrics, Montréal, Canada(2). Centre Hospitalier de l'Université Laval, Department of Endocrinology-Metabolism, Québec, Canada. We have recently reported that despite early treatment, children with congenital hypothyroidism have lower scores in hearing-speech development, which is evident as soon as 18 months of age. Therefore, we have undertaken to evaluate brain stem auditory evoked potentials (BAEP) in these children. At the present time, we have studied BAEP in 34 hypothyroid children between 5-12 years of age and compared them with those obtained in 24 age and sex matched normal children. BAEP abnormalities were found in 21 children (62%) and consisted of:

- 1° Prolonged peak latencies in 9 children with 2 of them also displaying a prolonged I-V interpeak latency.
- 2° Abnormally short wave V latency or interpeak latencies (I-III, III-V or I-V).
- 3° Three children had both a prolonged wave I latency and a short I-V interpeak latency and were included in each group. Children with prolonged latencies were referred for further audiometric evaluation. From the sparse data available in the literature, we think that there is probably a relationship between these abnormally short BAEP latencies and the elevated levels of plasma thyroxine seen in 18 of these 21 children. More data will be needed to clarify this hypothesis.

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NEWBORN CORD BLOOD CONTAINS FOUR SEPARATE NEUROHYPOPHYSIAL PEPTIDES. Rosemary D. Leake, M. Gore Ervin, Janet A. Amico, Michael G. Ross, Alan G. Robinson, and Delbert A. Fisher, Departments of Pediatrics and Obstetrics, Harbor/UCLA Medical Center, Torrance, CA and Department of Medicine, University of Pittsburgh, Pittsburgh, PA.

Recently we reported the presence of an estrogen stimulated oxytocin vasotocin-like (OT-VT) immunoreactive material in plasma of adult human males and females. Although immunoreactive with an antiserum raised against synthetic arginine vasotocin (AVT), the elution profile of OT-VT by high pressure liquid chromatography (HPLC) indicates that it is distinct from AVT, arginine vasopressin (AVP) or oxytocin (OT). Because fetal life represents an estrogen-primed state, cord blood from 15 vaginally delivered newborns was examined for evidence of OT-VT. HPLC analysis of an extracted pool of cord blood plasma revealed 4 peaks (I-IV) of AVT-like immunoreactivity (irAVT). Peaks II and IV coeluted with synthetic AVP and OT and were attributable to cross-reaction of the AVT antiserum with these peptides. Peak I was identified as AVT on the basis of its coelution with synthetic AVT. Peak III eluted in a manner identical with OT-VT. These results indicate that: 1) in addition to AVP and OT, native AVT is present in the circulation of the human newborn, and 2) human newborn plasma contains an irAVT material that is distinct from AVT, AVP or OT but is identical with a novel OT-VT material observed in plasma of estrogen-primed adults. The physiological significance of AVT and OT-VT in newborn plasma remains to be defined.

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SUPPRESSED URINARY LH LEVELS IN INADEQUATELY TREATED PUBERTAL PATIENTS WITH CONGENITAL ADRENAL HYPERPLASIA. Peter A. Lee, Department of Pediatrics, Univ. Pitt. Sch. of Med., Children's Hospital of Pitt., Pitt., PA.

Radioimmunoassayable urinary gonadotropins and urinary 17 ketosteroid (KS) levels have been determined in 223 24 hour urine samples from 65 patients with 21-hydroxylase deficiency congenital adrenal hyperplasia. Results have been categorized by sex and, based on chronological age, skeletal age and physical findings as prepubertal (Pre), pubertal (Pub) and postpubertal (Post). LH and FSH levels were divided into 2 groups based on KS, elevated ($\uparrow$) or normal (NL) for treatment adequacy.

	LH ($\mu\text{g/ml}$)		FSH ($\mu\text{g/ml}$)		n	
	NL KS	$\uparrow$ KS	NL KS	$\uparrow$ KS	NL KS	$\uparrow$ KS
MALES						
Pre	7.7 $\pm$ 5.4	6.4 $\pm$ 4.4	22.8 $\pm$ 11.7	21.0 $\pm$ 9.4	10	15
Pub	37.3 $\pm$ 27.3 *	18.9 $\pm$ 13.0	61.9 $\pm$ 56.4	62.8 $\pm$ 53.3	24	7
Post	57.4 $\pm$ 34.1	36.3 $\pm$ 25.7	75.6 $\pm$ 50.6	56.2 $\pm$ 27.3	25	14
FEMALES						
Pre	10.1 $\pm$ 11.7	10.3 $\pm$ 8.5	31.7 $\pm$ 20.0	39.9 $\pm$ 32.9	31	9
Pub	53.0 $\pm$ 39.2 *	33.0 $\pm$ 22.1	106.5 $\pm$ 64.4	121.4 $\pm$ 86.4	31	18
Post	56.0 $\pm$ 47.6	47.2 $\pm$ 47.5	106.3 $\pm$ 99.5	115.8 $\pm$ 90.8	34	5

LH levels are significantly lower ($p < 0.03$) when KS levels are inappropriately elevated for age and stage of development in pubertal males and females. This effect is reversible with a significant rise of LH levels (18.1 ± 13.0 to 64.2 ± 34.2 , $p < 0.001$) when KS were suppressed in 10 pubertal subjects.

These data suggest that the elevated adrenal androgens in inadequately treated adrenal hyperplasia suppress developing pubertal hypothalamic-pituitary gonadotropin secretion.

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SOMATOMEDIN-C (SM-C) LEVELS IN THE DIAGNOSIS OF GROWTH HORMONE (GH) DEFICIENCY. Phillip DK Lee, Lois Rountree, Darrell M Wilson, Raymond L Hintz, Ron G Rosenfeld, Department of Pediatrics, Stanford University, Stanford

Radioimmunoassay of SM-C is available through many commercial laboratories. We reviewed our records over the past 2½ years to assess the value of SM-C levels in the diagnosis of GH deficiency (GHD). I-L-DOPA (LD) or clonidine (CL) screening tests for GHD were performed in 84 cases with clinical indications which included height < 3% abnormal growth rate, delayed bone age, and/or pituitary lesion or insult. LD or CL failure (peak GH < 7ng/ml) was followed by an arginine-insulin infusion test (AI) for final diagnosis of GHD. Serum SM-C levels were determined during the screening period and compared to age-related normal ranges (Endocrine Sciences, Tarzana, CA). Complete data were available for 29/34 LD and 41/50 CL cases. SM-C levels were known for 5 of 6 cases diagnosed with GHD (age 3-15 yr). The table presents [cases with low SM-C for age]/[total in each group].

	LD-failed	LD-passed	CL-failed	CL-passed	Total
AI-not done	--	12/18	--	15/29	27/47
AI-passed	3/9	--	2/9	--	5/18
AI-failed (GHD)	2/2	--	3/3	--	5/5

All cases of GHD had SM-C below the normal range for age. Specificity for diagnosis of GHD was 2/11 (18%) for LD, 3/12 (25%) for CL and 5/10 (50%) for LD or CL + low SM-C. Our data show that SM-C is a highly sensitive predictor of GHD and increases the specificity of the LD and CL tests. Our data also suggest that a normal SM-C argues against the diagnosis of GHD and may eliminate the need for further testing.