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Thyroxine of human and bovine milk origin: possible bearing on congenital hypothyroidism.

Thyroxine (T4) concentrations were measured by RIA in 89 milk samples, obtained between 3-165 days postpartum from 40 healthy euthyroid mothers. The mean milk T4 conc. in the first week postpartum was $0.4 \pm 0.1 \mu\text{g}/100 \text{ ml}$ (n=9). The mean T4 concentrations between 8 and 48 days postpartum rose to $2.6 \pm 0.4 \mu\text{g}/100 \text{ ml}$ (n=57), and decreased to $1.28 \pm 0.3 \mu\text{g}/100 \text{ ml}$ (n=23) after 50 days postpartum. T4 concentrations in bovine (n=15) milk samples were less than $0.4 \mu\text{g}/100 \text{ ml}$. The data suggest that milk of humna, but not bovine, origin can provide a significant exogenous source of T4 to the infant. In the hypothyroid infant, T4 in human milk may delay clinical recognition of the disease. Although this exogenous source of T4 may alleviate the disease, it is insufficient to prevent the detrimental effects of hypothyroidism.

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Systemic and local immunity in juvenile autoimmune thyroiditis.

The relative and absolute numbers of circulating and thyroidal T and B lymphocytes were studied in 16 euthyroid patients with juvenile autoimmune thyroiditis (JAIT). The same cell populations were tested for cell-mediated immunity (CMI) to thyroid antigen in the leukocyte migration test (LMT). The diagnostic criteria of JAIT were a firm goitre, circulating thyroid antibodies and lymphocytic infiltration of the thyroid gland. The relative and absolute numbers of circulating T and B cells were normal (73 and 19%, respectively). Of the thyroidal lymphocytes 49% were B and 42% T cells. In LMT, half of the patients were positive when tested with circulating leukocytes, but negative when tested with thyroidal cells. Thus, in JAIT, antigen-sensitive thyroidal T cells are absent or inactive.

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TRH releases FSH in children: an explanation for the elevated FSH levels in juvenile hypothyroidism.

The majority of children with long-standing juvenile hypothyroidism show sexual maturation of characteristic pattern advanced in relation to bone age and mediated by FSH release (N.D. Barnes, A.B. Hayles, R.J. Ryan, Mayo Clin. Proc. 1973, 48,849). The reason for the FSH release was obscure. We have studied the effect of an intravenous bolus of 200 μg TRH in 13 children (4 boys, 9 girls, age range 2.7 - 17.0 years) without thyroid disease. All showed a response in FSH as well as TSH but none a detectable response in LH. Three girls with primary hypothyroidism showed elevated resting levels of FSH and a further rise after TRH (see table).

	Mean Values 0, 20 and 60 min. after TRH			TSH (IU/l)			FSH (IU/l)			LH (IU/l)		
	0	20	60	0	20	60	0	20	60			
Euthyroid (13)	2.1	9.9	6.9	1.1	4.1	3.6	<1	<1	<1			
Hypothyroid (3)	412	993	1010	10.4	18.1	20.6	1.2	1.5	1.6			

Thus TRH releases FSH in both euthyroid and hypothyroid children.

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Plasma levels of adrenaline and alanine in spontaneous hypoglycemia.
24 children with hypoglycemia showed in average a concentration of adrenaline before tolbutamide provocation (30mg/kg) (T) of 66 ± 31 , before insulin load (0.05U/kg) (I) of 46 ± 16 and of 55 ± 17 pg/ml before ketogenic diet (K). In control children ranged the basal adrenaline level between 81 and 98 pg/ml (p 0.15). The adrenaline conc. rose in the controls to average values of 811 (T), 588 (K) pg/ml, and increased 6-fold during I. The children with ketogenic hypoglycemia averaged peak values of adrenaline of 166 (T), 194 (I) and 184 (K) pg/ml. Similar results were obtained in 12 other patients with spontaneous hypoglycemia (type Zetterstroem) (Z). Plasma alanine conc. averaged in controls 332 $\mu\text{mol}/\text{l}$, in children with ketogenic h. 219 $\mu\text{mol}/\text{l}$ (p 0.01). During ketogenic diet provokation the alanine level decreased in controls of 14.5%, in children with Z-hypoglycemia of 24%, in patients with ketogenic h. of 45.8%.

The low alanine concentrations may be caused by a diminished adrenaline liberation after hypoglycemic stimuli, whereas the decreased alanine concentration may be the cause for a decreased substrate availability for gluconeogenesis.

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Twenty-two children with Cushing's disease were submitted to sequential plasma ACTH assays. Most patients were sampled before and after the injection of Lysine-Vasopressin (LVP), 10 IU / 1.73 m². Non treated patients had basal ACTH values within the normal range (<100 pg/ml). After treatment, in spite of the wide dispersion of the natural course of the disease, the rises in ACTH levels were as follow: ACTH values remained moderately elevated (<200 pg/ml and 300 pg/ml respectively before and after LVP) in patients treated with o-p'DDD (2-9 g/day) associated (3 patients) or not (8 patients) with pituitary cobalt therapy. A relapse was observed in 5 patients of the later group. After bilateral adrenalectomy (14 patients) ACTH rose until much higher values. In this last group were observed 7 out of the 8 cases of confirmed (5) or suspected (3) Nelson's syndrome. ACTH values of those patients were as high as 6,000 pg/ml and 25,000 pg/ml respectively before and after LVP. Important rise in ACTH levels at sequential determinations is thus a strong argument for the detection of pituitary tumors and appear to be very useful for management of treatment and follow-up.