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Development of retinopathy in diabetic (D) children and adolescents.

120 D children,adolescents,and young adults (66 ♂and 54 ♀),aged betw.6.5 and 27.5 yrs,90 of which had entered or completed puberty (>T II),were subjected to a thorough ophtalm.examination including fluorescein angiography(FA)and photographic documentation of the right fundus.D duration ranged between 1 and 20 yrs (groups 1:1-4.9 y(n=57);2:5-9.9 (37);3:10-14.9(19); 4:>15(7)).Metabolic control was evaluated using outpatient visit measurements of glucosuria,post-prand. blood glucose,growth and weight increments,and episodes of severe metabolic derangements.
Ophtalmoscopy and fundus photogr.revealed vascular changes in 10%,while FA documented microangiopathy (Stages I:1-5 microaneurysms(MA);II:6-10 MA;III:>11 MA;IV:proliferative retinopathy)in 30% of these pat. Its incidence increased with age (from 0% <10 to 50% >16 yrs),duration of D(4% in group 1,38% in 2,53% in 3,and 86% in 4,3/4 of all changes representing stage I) and deterioration of control(13% foll.longterm"good", 29% foll."fair",and 38%foll."poor"control).Adolesc. females tended to show more severe changes.In 3young women aged 17,20,and 21 yrs,proliferative retinopathy was found after 7,8,and 11 yrs of D.One of these had maintained "good" control during 7 out of 8 yrs of D.

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The influence of human growth hormone (HGH) on the thyroxine (T_4)-triiodothyronine (T_3)-ratio in HGH deficient patients.

To study the HGH-dependency of the T_4-T_3-conversion rate 12 patients with proven HGH-deficiency were examined. 8 patients showed additionally an impared TSH secretion compatible with secondary hypothyreoidism. T_4, T_3, TSH and TBG were measured by specific radioimmunoassay a) under current substitution therapy, b) after its cessation for at least 4 weeks and c) after recommencement in two weeks intervalls.

Results: Under current therapy all values were in the normal range. After cessation of therapy, T_4 decreased in all patients with hypothyreoidism below 5,0 mcg% while T_3 kept the level>80 ng% in 3 cases and was found below the lower limit in the other 5 patients. TSH levels remained unchanged in the normal or low basal range. In the 4 patients with isolated HGH deficiency T_4 and T_3 remained as to be expected in the normal range. 4-6 weeks after recommencement of the prior therapy there was in all cases including those with isolated HGH deficiency a HGH dependent increase of T_4 - T_3 - ratio due to a slight or moderate decrease of T_4 and an increase of T_3.

Conclusion: In HGH deficient patients with or without additonal anterior pituitary insufficiencies HGH substitution cause a significant enhancement in the conversion of T_4 to T_3.

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Serum T4 T3 TBG T3 uptake concentrations in childhood and puberty.

Measurements of serum thyroxine (T4) thyroxine binding globulin (TBG) and triiodothyronine (T3) using RIA as well as T3 uptake % (T3 U) free thyroxine index (FT4I) and free T3 index (FT3I) were conducted in 200 healthy Israeli children. Their age ranged from 1 to 17 years. 140 children from the age of 8 years were divided into five groups according to Tanner's puberty stages. There were no significant age related changes in TBG and T3U. Linear regression of serum conc. of T4 T3 and FT4I showed that each decreased significantly ($P < 0.001$) with age, while FT3I did not change significantly. The correlation with Tanner's staging showed a significant decrease in T4 and TBG conc. after mid-puberty (between stages P2 and P4). T3 however, decreased only toward the last stage of puberty (between P4 to P5). Our present data indicate that the decrease in T4 and T3 conc. are not only due to the decrease in TBG conc. before puberty, however, during puberty TBG might play a more significant role in the decrease of thyroid hormone concentrations.

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Normal values for circulating thyroid hormones (T_4, T_3,reverse T_3), T_3Uptake and Thyrotropin (before and after TRH)of Austrian children.

182 Austrian children (all using Austrian"Vollsalz" with 10mg KJ/kg salt),aged 2 months till 14 years, were investigated in a cross sectional study with the approval of their parents. Measurements of T_4, T_3, rT_3,T_3U and TSH (before and after TRH 5mcg/kg) were done by RIA. Free T_4RIA- and free T_3RIA-Indices and the ratio rT_3/T_3 were calculated. Results:

	T_4 mcg/dl	T_3 ng/dl	rT_3 ng/ml	TSH mcU/ml 0'
X ± SD	8.05 ±2.01	108.8±33.8	0.27±0.09	2.25±1.96
range	6.04-12.07	73.9-174.4	0.18-0.44	0.0-6.16

	TSH mcU/ml 30'	rT_3/T_3	FT_3I	FT_4I
X ± SD	14.33 ± 9.29	0.24±0.02	3.14±0.6	0.22±0.05

Geometric mean serum concentrations of T_4,T_3, rT_3 and TSH(basal)were not age related different; T_4 showed a not significant negative slope with ageing. The geometric mean values of T_3U were different between the age groups. The ratio rT_3/T_3 remaines constant. TRH induced TSH release is unchanged from 2 months till 14 years. Our results differ in part to those of the literature.

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Plasma 17-OH-progesterone at birth and during the early neonatal period in full term and preterm infants.

Assessment of plasma 17α-OH-progesterone (17-OHP) provides a valuable aid in the clinical diagnosis and management of congenital adrenal hyperplasia. However, for this to be useful it is necessary to know the normal values. In contrast to the large number of investigations in full term infants, insufficient data are available concerning 17-OHP levels in preterm infants. In full term and in preterm infants cord and peripheral blood 17-OHP levels were determined, using a commercially available RIA-kit (Sorin). The results (in ng/ml ± S.E.M.) are summarized in the following table.

	Cord blood	Peripheral blood (1st week of life)
Full term	31 ± 1 (n = 45)	2.1 ± 0.2 (n = 51)
Preterm	17 ± 2 (n = 24)	4.1 ± 0.2 (n = 59)

The means found for two groups of infants differ significantly ($p < 0.001$). No differences were found in 17-OHP concentration between male and female infants.

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Increased urinary excretion of 16α-hydroxy-pregnenolone in newborn infants with 21-hydroxylase deficiency.

Urinary excretion of total 16α-hydroxypregnenolone (16α-OH-P'O), pregnanetriol (PT) and 11-oxopregnanetriol (11-O-PT) were determined by capillary gas chromatography in 18 healthy neonates and 3 newborn infants with congenital adrenal hyperplasia (CAH) during the first three weeks after birth. In the 4th week of life all CAH-infants demonstrated salt loosing crisis.
Mean steroid excretion in µg/day (healthy infants vs. CAH (brackets)):

weeks	16α-OH-P'O	PT	11-O-PT
1st	25	12	10
2nd	214 (1317)	49 (93)	141 (142)
3rd	480 (2955)	39 (61)	85 (968)

Conclusion:
The determination of urinary excretion of 16α-OH-P'O is a valuable tool in the reliable detection of 21-hydroxylase deficiency during the first weeks of life when conventional tests may fail.

Supported by DFG SFB 87 Project C-3