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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 opthalm. 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. Ophthalmoscopy 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 3 young 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 (T₄)-triiodothyronine (T₃)-ratio in HGH deficient patients.

To study the HGH-dependency of the T₄-T₃-conversion rate 12 patients with proven HGH-deficiency were examined. 8 patients showed additionally an impaired TSH secretion compatible with secondary hypothyroidism. T₄, T₃, 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 intervals.

Results: Under current therapy all values were in the normal range. After cessation of therapy, T₄ decreased in all patients with hypothyroidism below 5.0 mcg% while T₃ 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 T₄ and T₃ 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 T₄ - T₃-ratio due to a slight or moderate decrease of T₄ and an increase of T₃.

Conclusion: In HGH deficient patients with or without additional anterior pituitary insufficiencies HGH substitution cause a significant enhancement in the conversion of T₄ to T₃.

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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 (F < 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 (T₄, T₃, reverse T₃), T₃ uptake 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 T₄, T₃, rT₃, T₃U and TSH (before and after TRH 5mcg/kg) were done by RIA. Free T₄RIA- and free T₃RIA-Indices and the ratio rT₃/T₃ 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 ₃ I	FT ₄ I
X ± SD	14.33 ± 9.29	0.24 ± 0.02	3.14 ± 0.6	0.22 ± 0.05

Geometric mean serum concentrations of T₄, T₃, rT₃ and TSH (basal) were not age related different; T₄ showed a not significant negative slope with ageing. The geometric mean values of T₃U were different between the age groups. The ratio rT₃/T₃ remains 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'0), 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'0	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'0 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