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Social and vocational adjustment of 38 hGH deficient patients after termination of therapy.

Thirty eight hGH deficient patients aged from 15 to 35 yrs in whom hGH therapy was stopped upon termination of growth, were re-evaluated for their social adjustment by personal interviews. Their characteristics are:

	Number of Patients IGHD	MPHD	Total	Final Height
Male	5	14	19	161.0 ± 6.4
Female	7	12	19	146.8 ± 5.2
Total	12	26	38	

The mean length of treatment period was 6.7 ± 3.2 (1.6-14) yrs, which had begun in 80% of the patients after age 9. It was found that 10 are students (7 in secondary school and 3 at the university); 7 are now serving in the army (another 6 completed army service); 18 have jobs with tenure; 3 are unemployed. Of the entire group, 4 (1 man, 3 women) are married. The successful social and vocational adjustment is attributed to the continuous multidisciplinary team approach these patients had received throughout their follow-up.

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Effects of growth hormone treatment on final adult height in boys with short stature and transitory partial growth hormone deficiency.

Data are conflicting regarding the results of treatment with growth hormone (GH) in patients with short stature and partial or transitory growth hormone deficiency. A retrospective analysis was performed to compare final adult height in GH-treated short boys to an untreated matched control group.

12 boys (group 1) had maximal GH-responses to insulin- and arginine stimulation tests between 4 - 10 ng/ml. At a mean chronological age (CA) of 13.09 ± 1.87 (SD) and a bone age (BA, Greulich-Pyle) of 10.25 ± 1.89 years GH-treatment was started for an average time of 2.44 ± 1.05 years (range 1.17 - 4.08 years). All patients had normal GH-responses following exercise when tested after discontinuation of therapy.

Comparison was made to an untreated control group (group 2) matched for BA (10.04 ± 1.80 years) and height at initial visit (136.00 ± 6.17 cm group 1 vs. 135.75 ± 7.86 cm group 2). Height predictions using the Bayley-Pinneau method were similar in group 1 (168.48 ± 9.14 cm) and group 2 (170.09 ± 6.73 cm). Final adult height was 164.15 ± 5.31 cm in group 1 and 168.87 ± 4.66 cm in group 2. We conclude that GH-treatment of transitory partial GH-deficiency does not increase final adult height.

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Influence of age at irradiation on the occurrence of GH deficiency in children receiving 2400 rads on the brain for acute lymphoblastic leukemia(ALL) or lymphosarcoma(LS)

It has been shown that after cranial irradiation for ALL (2400 rads), the peak plasma GH response after stimulation can be reduced but often without decrease growth velocity. The purpose of this study was to evaluate the frequency of GH deficiency, its relationship to growth and the possible effect of age at irradiation. Children (n=36 : 27 ALL, 9 LS) who had been exposed to a similar cranial radiation (12 x 200 rads over 16 days), were investigated ; chemotherapy had been terminated in 27. Two groups were distinguished according to GH peak values (ng/ml) after AITT: Group I≤8 and Group II>8 ; the others parameters, including chemotherapy and interval time since irradiation (respectively 5 7/12 ± 7/12 and 5 4/12 ± 7/12 yr) are similar in these two groups.

Group	n	Age irradiation (yr)	Decr.growth velocity(n)
I	21	5 2/12 ± 6/12 (m ± SEM)	15
II	15	8 4/12 ± 11/12 **	3

In group I, GH deficiency was complete (13) or partial (8). In this group, children had been irradiated at a significantly younger age ($p < 0.01$).

In conclusion, GH deficiency 1) is a frequent complication ; 2) determines frequently decrease growth velocity ; 3) is more likely to occur in children when irradiation is performed at a younger age.

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Comparison of growth hormone (GH) sleep secretion and responses to pharmacological tests.

GH secretion was studied during sleep in 37 short patients (28 males and 9 females) with height from - 2 to - 6.5 SD. No cause of growth retardation was known in anyone of them. A concommitant complete endocrine evaluation was done everytime with at least two pharmacological tests, using ornithine, arginine and/or insulin. The major GH sleep peak level (SP) was compared to the major GH pharmacological peak level (PP). SP ranged from 2.9 to 46 ng/ml (mean ± SD = 15.8 ± 11 ng/ml) and PP from 4 to 30 ng/ml (mean ± SD = 13.9 ± 8.3 ng/ml). SP and PP were correlated, $r = 0.50$ ($p < 0.01$). SP was consistantly below 10 ng/ml when PP was below 6 ng/ml. However, if individual values of SP and PP were in the same range in 23 patients (12 normal, 5 borderline and 6 blunted), they showed discrepancies in 14 : a lack of SP in spite of a normal or borderline PP in 3 or the contrary in 2, and a borderline SP in spite of a normal PP in 3 or the contrary in 6. This shows that in very short children, a normal or bordeline GH response to pharmacological stimuli does not prelude a lack or an insufficiency of GH sleep secretion. In such cases, measurement of GH sleep secretion may offer a rationale for a trial course of hGH treatment.

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Correction of obstructive sleep apnea and sleep entrained growth hormone release by tracheostomy in achondroplasia.

Obstructive sleep apnea (OSA) may occur in patients with achondroplasia. Since the bulk of growth hormone (GH) is secreted in relation to slow wave sleep (SWS), disordered sleep may hinder GH release and subsequent growth. To examine these relationships, a 9 yr old prepubertal male achondroplastic dwarf with growth failure and OSA was studied with polysomnography and q 20 min sampling for sleep entrained GH before and 4 mos. after therapeutic tracheostomy (T).

	growth velocity (cm/yr)	apneic episodes (per hr)	%slow wave sleep	GH secret. episodes (during sleep)	Total GH secret. (during sleep)
before T	3	105	none	1	24µg
after T	5.3	none	25.6	2	73µg
nl for age	5	none	13-30	2-3	54-122µg

Correction of sleep apnea normalized SWS and led to normalization of sleep entrained GH secretion and distinctly increased growth rate sustained 8 mos. after T. These results suggest that OSA in achondroplasia may further impair growth in these youngsters. Deficient sleep entrained secretion of GH is fully reversible by therapeutic tracheostomy. This is the first documentation of reversible GH deficiency caused by disordered sleep.

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Enhanced hGH pituitary secretion rate associated with somatomedin deficiency as manifested in Laron type dwarfism (LTD)

In 3 patients with LTD and 8 normal subjects the following parameters were determined:

	Age	Sex	MCR (ml/min/m^2)	IC-hGH(ng/ml)	PR(ng/ml/m^2)
LTD	19	F		25	
	19:9	F	73.4	33.9	720
	27	M	75.3	9.3	2480

Integrated 24h hGH concentration (IC-hGH) was elevated in the 2 teenager LTD females and at the upper limit of normal in the young LTD male. The metabolic clearance rate (MCR) was lower in the 2 LTD patients studied than in normal subjects, as in patients with hGH deficiency. The production rate (PR) was elevated in the girl (within the range of acromegaly) whereas in the young man it was only slightly elevated. All 3 LTD patients showed secretory 24h patterns having more frequent and greater fluctuations than in normals. In view of recent findings that the primary defect in LTD is an inability of liver receptors to bind hGH, causing a lack of somatomedin (SM), it is suggested that the enhancement and irregularity of hGH secretion in LTD is due to a dual mechanism, both pathways being related to SM deficiency: 1) lack of negative feedback on pituitary hGH and possibly on GH-RH; 2) lack of stimulating effect on somatostatin secretion.