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ADULT HEIGHT OUTCOME IN BOYS WITH IDIOPATHIC SHORT
STATURE.

Final adult height was recorded in 54 boys over 20 years of age who were investigated for short stature in childhood. All had exhibited normal GH response to provocative stimuli and received no treatment. 29 out of them (group I) reached an adult height above - 1.5 SD of control population, $\bar{x}$ = 171 cm (range 167-180). Their mean pubertal growth spurt was 28.6 cm (range 22-33) and started when chronological age (CA) was 14.85 ± 1 years, height age (HA) 11.75 ± 1 y. and bone age (BA) 12 ± 1 y. 25 subjects (group II) reached adult height below - 1.5 SD, $\bar{x}$ = 160.5 cm (range 150-166). The pubertal growth increase was 23 cm (15-27) and started for CA of 13.95 ± 1 y., HA of 10 ± 1 y. and BA of $12.5 \pm .75$ y. Mid parental height were 163.7 (group I) 160.5 (group II) respectively. Mean GH responses to conventional tests were 26 ng/ml (12-64) in group I, 19 ng/ml (10-36) in group II respectively. When IGF I levels were measured in prepubertal stage, subnormal values were observed in group II. During puberty IGF I levels were low in both groups. By way of comparison, the final height outcome of 7/10 adolescents suffering from transient untreated GH deficiency was identical to that observed in group I (the mid parental height and the clinical pubertal parameters were the same). The differences in prognosis between the two groups of constitutionally short children must be kept in mind when GH treatment is discussed.

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GROWTH OF CHILDREN IN LARGE FAMILIES.

Data were collected retrospectively from 1,811 children, 6-12 years of age, in a rural area with predominantly large families. Data were analyzed as percentiles (%) for the respective ages. The mean heights of boys and of girls in the study cohort were 41.9% and 43.3%, respectively. Height of boys in families of 2-3 children was 47.1% (n=46), compared to 41.8% in families of 4-5 (n=179), 37.6% in families of 6-7 (n=116) and 33.7% in families of 8-14 children (n=151). Girls' mean heights were 48.6%, 42.3%, 41.5% and 42.6% in families of 2-3, 4-5, 6-7 and 8-14 children, respectively. Weight did not differ between children of large and small families. Birth order had no significant effect on height or weight. In boys, a short preceding birth interval of <2 years was associated with smaller mean heights of 37.8%, compared to 41.8% and 43.2% when birth intervals were 2-3 years and >3 years, respectively. Growth of girls and weight gain of boys and girls were not affected by birth intervals. Conclusions: 1) Family structure has a profound effect on growth. 2) Growth correlates with size of family and birth interval, but not with birth order. 3) Boys are affected more than girls. 4) Height is affected more than weight, excluding nutritional factors. 5) This growth deficit is viewed as partial psychosocial dwarfism.

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AMNIOTIC FLUID (AF) ARGININE-VASOPRESSIN (AVP) CONCENTRATIONS IN
NORMAL PREGNANCIES AND INTRAUTERINE GROWTH RETARDATION (IUGR).
AVP is released by the fetus not only in response to osmotic, but
also to nonosmotic stimuli such as hypoxia. Once released, AVP is
excreted via fetal urine into AF where it has a long half life
compared to plasma (ca 8 hours). The hypothesis of the present study therefore
was, that fetal hypoxia might be diagnosed by elevated AF-AVP levels. As a model
we used IUGR fetuses which are known to be hypoxic. 25 infants entered the study
because IUGR was suspected by ultrasound. The normal AF-AVP levels as control
values were determined in 93 uncomplicated pregnancies where amniocentesis was
performed for other reasons (L/S ratio determinations, genetic reasons, rh-
sensitized pregnancies with rh-negative fetuses). HPL values were measured by
RIA as were AF-AVP values.

Results: The mean AF-AVP levels in normal pregnancies from 16 to 41 weeks of
gestation was 2.37 ± 1.16 SD pg/ml. There was no significant difference with
different gestational age. At birth 7 of the 25 infants had normal birth weight
and 18 actually had IUGR. There was a significant correlation between the degree
of IUGR and AF-AVP levels ($r=.59, p<0.05$). The correlation between the HPL values
and the degree of IUGR was the same as for AF-AVP values ($r=.59, p<0.05$).
Discussion: 1) To our knowledge we report AF-AVP values for the greatest group
of normal pregnancies yet published. 2) AF-AVP levels are elevated in IUGR. The
levels dependent on the severity of IUGR. These data support the usefulness of
AF-AVP determinations for the diagnosis of fetal wellbeing as demonstrated in a
previous study in rh-erythroblastosis (1). The elevated AF-AVP levels in IUGR
might explain findings of diminished urine production of these fetuses (2).

(1) Stegner et al. Acta Endocrinol 112,267,1986

(2) Wallenberg et al. J Perinat Med 6,233,1977

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METHIONYL GROWTH HORMONE (met-GH) IN A DOSAGE OF 4 IU/
m² DAY PROMOTES GROWTH IN TURNER SYNDROME.

In a Dutch multi-center study 16 girls with Turner Syndrome were
treated with met-GH in a dosage of 4 IU/m² body surface adminis-
tered s.c. once a day. In girls older than 13 years low-dose ethi-
nyl-estradiol (EE) therapy (0.1 ug/kg body weight o.d.) was added.
Ages ranged from 7.9 to 15.2 years and bone age from 6.0 to 12
"years". Heights, expressed as standard deviation scores, were be-
tween -2.5 and -5.1 (median -3.9). Endogenous GH secretion was
assessed by arginine infusion, exercise test, GRF (1-29)-test and
plasma SM-C/IGF-I. Mean ($\pm$ S.D.) growth velocities were:

Baseline	0 - 3 m	3 - 6 m	6 - 9 m	0 - 9 m
3.4 ($\pm$ 0.8)	8.6 ($\pm$ 2.2)	6.4 ($\pm$ 2.4)	6.8 ($\pm$ 2.2)	7.2 ($\pm$ 1.6)

During the first 6 months of therapy the mean ($\pm$ S.D.) bone age
(Greulich and Pyle) advanced from 9.1 ($\pm$ 1.2) to 9.4 ($\pm$ 1.2) "years"
and the predicted adult height from 148.2 ($\pm$ 4.5) to 151.0 ($\pm$ 4.2)
cm. The growth response (growth velocity during 9 months of thera-
py minus baseline) ranged from 0.8 to 7.0 cm/year and was negati-
vely correlated with chronological age ($r = -0.48, p < 0.05$). In the
4 girls older than 13 years, who also received EE, the growth res-
ponse was 3.3 ± 0.9 , compared to 4.0 ± 1.6 in younger girls.
Growth response was not correlated with maximum plasma GH levels
during provocation tests. Anti-GH antibodies with a binding capa-
city of > 0.2 U/l were found in 5 out of 10 patients. In one of
them a very high value (3.7 U/l) was found.

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SHORT TERM RESPONSE TO SOMATREM IN TURNER SYNDROME
ASSESSED BY KNEMOMETRY AND CONVENTIONAL AUXOLOGY

The knemometer, an instrument designed to measure increments
in lower leg length, has been fully assessed and found to be a
precise means of determining short term growth patterns. The
technique has been incorporated in a trial designed to determine
the proportion of children with Turner syndrome responding to
exogenous Somatrem (Somatonorm), 2u daily by subcutaneous
injection, for a 4 week period within an intensive 13 week
measurement protocol and to assess the predictive value of
knemometry to identify the subsequent change in growth velocity
measured by conventional auxology over 6 months.

Thirteen girls (7 Turner mosaics; 6, 45XO karyotype) aged
5½-19½ years who had received no sex steroid therapy were studied.
Eight showed an acceleration of tibial growth in response to 4
weeks of Somatrem therapy when compared to a pre- and post-
treatment 4 week period. Four children subsequently showed a
significant change in height velocity (defined as an increase of
>2cm/year over the previous year's value). All short term non-
responders also failed to respond in the long term. Only half
those identified as responders in the short term were also long
term responders. In this small series knemometry was able to
predict those children in whom growth hormone therapy was not
beneficial.

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OXANDROLONE TREATMENT INCREASES FINAL ADULT HEIGHT
IN TURNER'S SYNDROME.

Oxandrolone treatment (Ox) induces an acceleration of growth ve-
locity in Turner's syndrome. However, the effect of such therapy
on final adult height remains controversial. 56 pts (age at start
of therapy: 6 - 17 years, karyotype 45 XO: n = 32, mosaicism or
structural abnormalities: n = 24) were treated with 0.1 mg Ox/
kg/day for 2 to 6 years. Growth velocity and final height were
compared with those of an untreated control group (Ranke et al.:
Eur. J. Ped. 141, 81, 1983). Growth velocity was significantly
higher in treated than in untreated patients ($p < 0.01$) and de-
creased with duration of Rx. Bone age did not accelerate during
treatment. After 2-6 years of therapy 27 pts were older than
19 years. Height was significantly higher than in controls (150.8
 ± 5.6 vs. $146 \pm 5.8, p < 0.05$). There was no difference between
pts with karyotype 45 XO and mosaicism or structurally abnormal
X-chromosomes. Pts started with Ox - Rx at age 13 - 15 years
(n = 15) seemed to be taller than pts (n = 10) 16 - 18 years old
at start of Rx (151.6 ± 6.4 vs. 149.8 ± 4.8 cm). However, the dif-
ference was not statistically significant. Our results indicate
that Ox therapy causes a moderate, but significant gain in final
height in Turner's syndrome.