**65**

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Plasma Somatomedin activity: removal of the in vitro and in vivo cortisol-induced inhibitory effect.

The in vitro addition of hydrocortisone at physiological concentrations to the assay, inhibited the plasma somatomedin (SM) action on porcine rib cartilage segments, as estimated by its incorporation of $^{35}$S-sulphate and $^{3}$H-methyl-thymidine.
A method has been devised for removing this inhibitory effect. By incubating plasma in a dextran-coated charcoal (DCC) suspension for 2 hours at 4°C temperature before assaying, SM, to which hydrocortisone had been added at a concentration of 1 µg/ml, returned to its original activity. In plasma from children treated with high doses of glucocorticoids, the low SM activity practically normalized after treating the plasma in DCC suspension.
The data further suggest that the administration of excessive glucocorticoids may cause growth retardation by interfering directly with SM action on cartilage metabolism rather than by decreasing SM generation.

**66**

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Hormonal control of testicular growth in monorchid prepubertal children.

Monochidism provides a model to study the changes of FSH, LH and testosterone (T) secretion in relation to testicular growth before puberty. 7 congenital monorchid subjects (EM) aged 8-12 years and 3 post-surgical monorchid subjects (SM) aged 9 years were compared with a control group of 30 children aged 8-12 years. The response to LHRH (100 µg/m2/iv) and to HCG (1500 iu x 3 on alternate days) was evaluated. Monorchid subjects of both types had an increased mean testicular volume of 4.8 ± 1.8 (controls 1.72 ± 0.65) and comparable responses to LHRH with mean FSH peak values of 3.35 ± 1.99 mU/ml superior to controls (1.90 ± 0.51 mU/ml, $p < 0.001$). However the mean T response to HCG was normal in CM (2.93 ± 0.74 ng/ml) and decreased in SM (1.68 ± 0.35 ng/ml) with control values of 2.75 ± 0.35 ng/ml.
In conclusion, congenital absence of one testis stimulates FSH secretion and testicular growth without change of the response to HCG.

**67**

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Measurement of serum thyroxine (sT4) by homogeneous enzyme immunoassay in normal children and in thyroid disorders.

As radioimmunoassay (RIA), enzyme immunoassay is based on the antigen-antibody reaction, labelled T4 bound to an antibody being displaced by unlabelled T4. T4 is labelled by coupling to malate dehydrogenase, which catalyzes the conversion of malate to oxaloacetate, with reduction of NAD to NADH. The enzyme's activity appears only at formation of the antigen-antibody complex with measurable optic density variations. By this method, sT4 was measured in 150 normal children aged 1day to 16 years. The values ranged from 4 to 11µg/dl, intraassay and interassay variations being 3 and 5%. The limit of detection was <0.5µg/dl. sT4 was also determined in children with thyroid disorders, and the correlation of the values with RIA was highly significant ($r=0.94$, $p<0.001$). When compared with RIA, the advantages of this method are the suppression of any manipulation of radioactivity and the rapidity to obtain the results, in the hour following the start of assay.

**68**

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Somatostatin and l-Dopa loading for study of Growth Hormone reserve.

Author's earlier studies /Péter and Szentistványi, 1977/ were continued in which a moderate rise of GH serum level was detected mainly at 150' and 180' after iv. bolus administration of somatostatin/SRIF/ in children. This rise is regular but the peak mean serum level is very low /8,05±3,02 ng/ml/. An iv. bolus of SRIF and p.o. l-Dopa was administered in a combination for 16 normal children with the parental consent according to the following protocol: at 0 minute 5µg/Kgbw SRIF and 120' later 250-500 mg l-Dopa were given. The increase of GH level was significant at 150' and 180' /the peak mean serum level is 22,5±16,04 ng/ml; extreme values 9,7-57,5 ng/ml. The dose- and time relations are very important because the somatostatin can suppress the rise in serum GH evoked by l-Dopa! By means of SRIF-l-Dopa loading test can help the differential diagnosis of dwarfism in a harmless form too.

**69**

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Evaluation of a Gonadal Stimulation Test in Children with Pseudohermaphroditism

25 children with male and female pseudohermaphroditism(phm, phf) and 1 child with hermaphroditismus verus(hv) were tested. Testing was helpful in the decision of castration and of social sex. While on dexamethasone 5000 IU HCG/d, and after a break of 3 days 300 IU FSH/LH(HMG)/d were administered each on 5 consecutive days. Plasma estradiol(E2) and testosterone(T) were determined by RIA every day. In phm (n=19) E2 did not change, but T increased from a basal value of $\bar{x}$=821(range: 45-11 200)pg/ml to 3 813(200-16 800) pg/ml during HCG-stimulation. There was an insufficient response in 5 and no gonadal stimulation in 4 cases. In phf (n=6) the basal value of E2 was 63(45-130)pg/ml. It increased to 498(70-1 325)pg/ml during HMG-stimulation. The increment of T was not significant. In 3 patients tests suggested ovaries, in 3 others no gonades. But in one of these "agonadal" patients potent ovaries could be demonstrated by histology. The failure of the test may be explained by the fact that HCG preceeded HMG-stimulation. In prepuberal ovaries FSH-sensitive follicles may be lutenized by HCG, which makes them unsensitive to the following-stimulus. Thus we give HMG before applying HCG.

**70**

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Conversion of 25-(OH)D3 to 24,25-(OH)$_2$D3 and nuclear binding of 24,25-(OH)$_2$D3 in chondrocytes.

Conversion of 25-(OH)D3 to 24,25-(OH)$_2$D3 and stimulation of proteoglycans synthesis in cultured chondrocytes has been previously demonstrated (Corvol et al., 1978). To investigate subcellular localization, labelled vitamin D metabolites, 26,27-$^{3}$H, were incubated with cultured chondrocytes from prepubertal rabbits for various time periods. Medium and cells were extracted separately with methanol-chloroform (v : v) and analyzed by high pressure liquid chromatography. When labelled 25-(OH)D3 was incubated, 8 x $10^{-9}$M, most radioactivity was recovered as 24,25-(OH)$_2$ D3 in the mitochondrial fraction and reached a plateau after 60 min. Same labelled 24,25-(OH)$_2$D3 was also found in the nuclei. When labelled 24,25-(OH)$_2$D3 was incubated, the radioactivity was mainly retained in the nuclei. Nuclear uptake of this label was inhibited by preincubating the cells with excess non labelled 24,25-(OH)$_2$D3. There was no specific nuclear binding of 25-(OH)D3. In conclusion, in chondrocytes the 24-hydroxylation of vitamin D is within mitochondria as already shown for the kidney. Specific nuclear binding of 24,25-(OH)$_2$D3 suggests that this is the active vitamin D metabolite in chondrocytes.