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- 2 Secretin release in Children with Coeliac Disease. D.I. JOHNSTON*, H. PATEL*, S.R. BLOOM* (Intr. by D.B. GRANT). Dept. Child Health, University Nottingham and Hammersmith Hospital, London.

Immunofluorescent studies have provided indirect evidence of disordered secretin release from the jejunal mucosa S-cells in coeliac disease. We measured the plasma secretin response to duodenal acidification in 15 normal children, 6 with active coeliac disease, and 6 with treated coeliac disease. Citric acid (0.5M, 5ml/Kg) was infused into the duodenum prior to jejunal biopsy. Plasma secretin was measured by a specific and sensitive radioimmuno assay. In the normal group secretin from from 1.3 ± 0.7 pmol/l to 24.7 ± 3.5 pmol/l at 10 minutes, in the active coeliacs from 1.7 ± 0.9 pmol/l to 6.3 ± 1.2 pmol/l and in treated coeliacs from 0.9 ± 0.6 pmol/l to 33.8 ± 6.9 pmol/l. The secretin response in active coeliac disease was significantly reduced ($p < 0.001$) and may contribute to impaired pancreatic exocrine function.

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Intraamniotic thyroxine (T4) effect in high risk pregnancies.

We reported previously the beneficial influence of intraamniotic T4 injection in accelerating human fetal lung maturity. 48 hr. after T4 (250 µg) intraamniotic injection mean cord serum T4 conc. in 15 premature newborns was 17.6 ± 1.9 µg/100 ml, while the T4 conc. in a matched group was 11.6 ± 0.9 µg/100 ml, and rose 12 hr. postpartum to 23.7 ± 2.4 µg/100 ml. No change in maternal T4 conc. was observed. Mean cord serum T4 conc. in 17 premature newborns delivered more than 3 days following T4 intraamniotic injection, however, were not significantly different than controls. These data suggest that a) T4 (250 µg) injected intraamniotically is absorbed by the human fetus, b) the hyperthyroxinemia is only transient, and c) does not block the postnatal T4 surge.

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Substitutive therapy in idiopathic hypogonadotropic hypogonadism (I.H.H.): resistance to HCG therapy possibly induced by long term testosterone (T) administration.

Seven patients with male IHH were treated with HCG for 12 to 40 months (m): 2000 IU twice a week (w) for 3m, then 5000 IU twice a w from the 4th m and 3 times a w after the first year in case of insufficient results. Five patients had previously received long term T therapy. Response to HCG was appreciated on morphological and clinical changes and on plasma T levels (ng/dl) determined 24 h after HCG injections. Rapid good responses (plasma T > 400) were seen within 3 and 4 m in 2 cases, 16 and 17 years old, who did not receive prior T. Good responses occurred only after 8 and 12 m of HCG in 2 cases, 17 and 29 years old, previously treated with T for 5 and 6 m. Responses remained weak after 18 to 26 m of HCG (plasma T: 55 - 250) in 3 cases, 18, 19 and 24 years old, previously treated with T for 10, 14 and 18 m. The quality of the responses to HCG seemed to be negatively related to the duration of the previous long term T therapy. From these data we suggest that serious considerations should be given to the fact that long term T therapy may induce later resistance to HCG in patients with IHH.

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Intelligence quotient (IQ) in patients with congenital adrenal hyperplasia (CAH), their parents and healthy siblings.

The IQ (Hamburg-Wechsler) was determined in 33 patients with CAH (15 m, 18 f) aged 6 to 36 yrs (21-hydroxylase deficiency, 16 with, 17 without salt-wasting), in 29 healthy siblings (8 m, 21 f) and in 48 parents (21 m, 27 f). The mean IQ in the parents was 104 ± 9 (SD), in the CAH-patients 111 ± 14 and in the siblings 114 ± 11 . The values in patients and siblings were higher ($P < 0.001$) than in the parents, but the difference between patients and siblings was not significant. These findings do not support an influence of androgens on the IQ nor the hypothesis of coupled genes for CAH and intelligence. The Hamburg-Wechsler normal values were established 21 years ago. The changed environmental factors probably account for the generation difference. It is concluded that the IQ in CAH-patients is not different from that in normal children.

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Sex differences in estrogen excretion in early infancy.

Urinary estrone (E1) and estradiol (E2) glucuronide and sulfate were determined separately by RIA in 44 newborn and young infants (26 boys, 18 girls). In 7 cases the estrogen excretion was followed up over one month. The conjugates were hydrolyzed enzymatically; the estrogens were purified and separated by Sephadex LH 20 chromatography. During the first month, children excreted more sulphated than glucuronized estrogens with a ratio of 8:2, but within the first year, this ratio was reversed to 3:7. In both sexes E1 + E2 excretion was 10 times higher during the first 2 months than at the end of the first year. Boys showed a sharp decline during the third month of life; girls, however, did not reach low values until the sixth month. While the E2/E1 ratio in boys was one, girls showed predominance of E2-excretion with a mean ratio of 2.5:1 during the first 6 months of life.