159 PLACENTAL TRANSFER OF PARATHYROID HORMONE

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We have investigated the placental transfer of three parathyroid hormone (PTH) fragments: 35-84, 44-68 and 65-84. The transfered fragments were identified by different methods. A cotyledone from human placenta approximately 3x4 cm was prepared for in-vitro perfusion. PTH extract was infused into the fetal artery (two perfusions with each fragment) for 20 min. Perfusate samples from maternal side (MS) and fetal vein (FV) were taken at intervals of 5 min over 40 min following start of perfusion. The identification of the PTH fragments was performed by: high-pressure-liquid chromatography (HPLC); preparative Flat-Bed electrofocusing (PFBE) and by gel filtration (GF). PTH transfer was detected 3 min after the start of perfusion.

Fragments	GF min		PFBE pH		HPLC min	
	FV	MS	FV	MS	FV	MS
35-84	110	110	8.0	8.0	39	39
44-68	146	146	7.4	7.4	13	13
65-84	152	152	6.4	6.4	18	18

Our results showed that the perfused and transfered fragments were identical.

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160 DEGRADATION OF CALCITONIN BY HUMAN PLACENTA

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We have investigated the degradation in vitro of calcitonin (CT) in human placental homogenates.
Mitochondrial fraction:placental tissue (0.5g) was homogenized and centrifuged for 1 hr at 13000 g.
Microsomal fraction: 5 g placental tissue was homogenized and centrifuged for 1 hr at 100000 g.
Human synthetic CT was incubated in 2 ml supernatant from each subcellular fraction at 4°, 20° and 37°C. Half-milliliter aliquotes were removed at intervals and immediately placed at -20°C. The CT values were measured by RIA.

Results: Percentages of degradation during incubation with placental mitochondrial fraction

°C	n	5 min	10 min	15 min	20 min
20°C	30	12.9%*	22.8%*	26.5%*	28.5%*
37°C	37	18.7%*	25.5%*	31.3%*	35.6%*

*$p < 0.001$

After incubation with the microsomal fraction a degradation of only up to 0.9% was measured at both temperatures.
Conclusion: The capacity of the placenta to degrade calcitonin suggests its physiological importance for the calcium homeostasis during pregnancy.

161 GONADAL FUNCTION IN PATIENTS WITH DOWN SYNDROME

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Patients with Down syndrome are reported to have delayed puberty and abnormal sexual development. However, the role of institutionalized life has not always been considered. For this reason, we studied a group of patients who were raised in their family environment (age range: 1 month to 24 years). Two of the male subjects had abnormal sexual differentiation, one with hypospadias, and one with bilateral cryptorchidism. None had significantly delayed puberty.

Serum gonadotropin levels were determined in the sexually mature male subjects (> 16 years). Serum levels of FSH in all subjects (mean 15.6, range 12.3-22.3 mIU/ml) were above the range for normal adult men (0.6-11.2 mIU/ml), but serum levels of LH (mean 14.3, range 9.1-21.6 mIU/ml) were above normal (3.0-12.0 mIU/ml) only in about one half of the subjects.

In male infants with Down syndrome ($\leq$ 6 months), serum FSH levels (mean 4.2, range 0.6-8.8 mIU/ml) and serum LH levels (mean 7.6, range 5.8-9.1 mIU/ml) were all in the normal range for this age group. However, plasma testosterone levels (mean 22, range 0-44 ng/dl) were all inappropriately low despite normal LH values.

In summary, our data suggest that 1) the underlying defect in abnormal sexual development in patients with Down syndrome is primary gonadal failure, and 2) this defect is already present in the first year of life.

162 EFFECT OF INTRANASAL Gn-RH IN PREPUBERTAL CRYPTORCHIDISM.

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To evaluate clinical and hormonal response to intranasal Gn-RH, 12 cryptorchidism boys, 10 unilateral (UC) and 2 bilateral (BC) were studied. All of them were treated with 200 μg of Gn-RH sprayed 6 times daily during 4 weeks. Serum samples were collected during intranasal Gn-RH stimulation test. The tests were performed after the first inhalation on the first and last day of treatment. Levels of LH, FSH, PRL and testosterone were measured by RIA and LH biological activity by bioassay (bio LH). In 4 patients (Group I) 3 with UC and 1 with BC, complete descent of the testes occurred. Other 4 patients (Group II) 3 with UC and 1 with BC needed an additional 4 weeks of treatment to complete descent. In the remaining 4 (UC) patients (Group III) testicular descent occurred outside the inquinal canal. Basal LH and FSH levels (RIA) were similar at the beginning and at the end of treatment and the initial response did not differ from group to group. FSH response showed a significant decrease at the end of treatment. Testicular descent occurred in 50% of patients being a good correlation between LH biological activity and response to treatment. This would suggest that a major degree of hypophyseal maturity would be necessary to release bio LH improving therapeutical results.

163 ABNORMAL Y CHROMOSOME IN 45,X/46,XY MOSAICISM.

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The mosaicism 45,X/46,XY is one of the sex chromosome abnormalities with a wide phenotypic expression. In 23 cases of XO/XY mosaicism diagnosticated by us, the phenotypic manifestations vary from phenotypic females with clinical Turner's syndrome and bilateral streaks (2 cases) to phenotypic male with infertility disorder (1 case), being the asymmetrical gonadal differentiation with ambiguous external genitalia (15 cases) and males with hypospadias and bilateral normal or dysgenetic testes (5 cases) the most frequent expressions.

The use of chromosome banding techniques, mainly Q and C bands, allowed to find an unexpectedly high proportion of abnormal Y chromosome in XO/XY mosaicism, suggestiong that such altered Y chromosome may predispose to anaphase lag or to mitotic non-disjunction leading to XO/XY or XO/XY/XYY mosaicism, respectively.

From own 10 cases re-studied with chromosome bands techniques, five of them (50 %) presented an abnormal Y chromosome: 3-Yqh- and 2-idic (Yq). The three patients with the Y chromosome without the heterocromatic region, studied with high resolution chromosome methodology, showed to be an isodicentric for the short arm of Y chromosome: idic (Yp). We suggest that the origin of the mosaicism XO/XY could be due to an abnormal and unstable Y chromosome such as an isodic Yp.

164 THE SIGNIFICANCE OF POSTNATAL GONADOTROPHIN SURGE FOR TESTICULAR DEVELOPMENT IN NORMAL AND CRYPTORCHID TESTIS.

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The hormonal and histological findings in cryptorchidism led us to investigate the following theory: The postnatal FSH and LH increase is not only responsible for setting of the gonadostat but a major pre-requisit for an adequate testicular development for later fertility. Testicular biopsies (36th week of pregnancy to 9 months postnatally) have been investigated from 29 cryptorchid infants operated without prior hormone treatment and compared to 34 biopsies of normal testes (9 post mortem, 25 biopsies because of suspected testicular damage during surgery for hydrocele or inguinal hernia). The absolute and relative number of spermatogonia increased from 50 to 100 per 50 tubuli in normally descended testes. In cryptorchid testes, however, the number of spermatogonia remained low and did not increase with age. In contrast, the total number of germ cells was always comparable in both groups. This gives evidence for a lack of transformation of gonocytes into spermatogonia in cryptorchid testes, which furthermore showed in contrast to normal testes no increase in the number of Leydig cells. The postnatal development of Leydig cells and the transformation from gonocytes into spermatogonia parallels the LH and FSH increase in the first few months of life. The activation of the gonadotrophin-gonadal axis could thus not only be responsible for the "setting" of the gonadostat but a pre-requisit for an adequate testicular development.