Decrease in luteinizing hormone (LH) by L-dopa in isosexual precocious puberty (IPP); possible relation of LH to behavior. Chandra M. Tiwary. (Intr. G. Van Leeuwen) Univ. NE Coll. Med. Omaha, NE.

An 18-months-old boy was evaluated for a 6 months history of increased stature, enlarged penis, and pubic hair. He was aggressive, unfriendly, disobedient and exhibited a short attention span and unpredictable behavior. Examination: muscular boy, HT 93 cms. (> 97%) WT 17.9 kg. (> 97%) Head circumference 59 cm. (> 50%) BP 100/60 mm., pubic hair stage II (Tanner), phallus 7 cm. long, testes symmetrical, of normal consistency, 7.5 ml. vol. each. Prostrate palpable rectally. Bone age 4 years. Normal studies included: brain scan; pneumoencephalogram; intravenous urogram; serum thyroxin; growth hormone and cortisol response to insulin; dexamethasone suppression test; cortisol response to vasopressin and corticotrophin. Serum testosterone (T) 583 ng/%, serum FSH 54.9 ug/%, Serum LH 16 ug/% (8 a.m.) and 8.3 ug (4:30 p.m.) Growth Hormone response was normal after 250 mg. of oral L-dopa, but the LH decreased by 46.4%. T propionate 250 mg. IM reduced LH by 62.5% on both days (3 and 6). The mother spontaneously reported a striking improvement in his sociability and attention span from 3 days through 19 following T. The implication of these findings will be discussed in the light of further observations.

FUNCTIONAL HYPOPARATHYROIDISM IN NEONATAL HYPOCALCEMIA OF INFANT OF DIABETIC MOTHER: Reginald C. Tsang, I-Wen Chen, Mary Ann Friedman (Introduced by Leonard Kleinman), Univ. of Cinti. Col. of Med., Dept. of Ped., and Radioisotope Lab., Cinti.

In neonatal hypocalcemia of infants of diabetic mothers (IDM), there is associated hyperphosphatemia and calcemic responsiveness to parathyroid extract, consistent with possible hypoparathyroidism. The serum immuno-reactive parathyroid hormone (PTH) levels of 28 IDM (15 class A; 13 B,C,D) were studied serially in the first 3 days of life to determine the PTH responses to changes in serum Ca. In class A IDM serum Ca fell from 10.35 mg% at birth to 7.84 at 24-48 hours and rose to 8.86 at 72 hours. In class B,C,D IDM the corresponding values were 9.6, 6.5 and 7.1. Serum ionized Ca fell in all IDM (4.2 to 2.5 to 3.2 mg%), serum Mg was unchanged, and serum P rose (6.1 to 7.0 to 8.3 mg%). Changes in serum PTH were not correlated with changes in serum Ca from birth to 24-48 hours(r=0.09). In class A IDM at 24-48 hours serum PTH was 134±14% of cord PTH values (paired t,p=0.06) and at 72 hours was 124±13%. In class B,C,D IDM, in spite of significant falls in serum Ca, PTH was correspondingly 118±16% (not significant)and 105±12%. Serum Ca at 72 hours was positively correlated(r=0.67,p=0.05) and serum P negatively correlated (r=-0.75,p<0.05) with PTH responsiveness at 24-48 hours. The present data are consistent with the thesis that functional hypoparathyroidism is a pathogenetic mechanism for neonatal hypocalcemia in infants of diabetic mothers.

Mechanism of growth hormone (GH) release following exercise(E). Chandra M. Tiwary. (Intr. G. Van Leeuwen) Univ. NE Coll. Med. Omaha.

Exercise causes release of GH and has been recommended as a screening procedure for ruling out GH deficiency. We reasoned that the post-exercise injection of glucagon (G), which also stimulates the release of GH, would enhance the sensitivity of the test. We had previously demonstrated that the changes in blood glucose or increase in free fatty acid do not abolish GH response to G. Five children with short stature, ages 5 through 15 years, were exercised for 20 min. (E) consisted of going up and down stairs for 15 min. and running on a level surface for 5 min. G (1mg.) was given IV. Blood for glucose insulin and GH were measured at the pre and post E and 10 and 15 min. post G. GH value is in ng/ml. Only peak GH value is given for post G and glucose values in mg.% are in ( )

Time	Case: 1	2	3	4	5
Pre E	7.8(72)	.9(81)	1.7(80)	4.6(76)	14.4(89)
Post E	16.8(78)	16.3(79)	17.1(59)	20(76)	36.2(97)
Post G	12.2(103)	9.4(107)	5.2(117)	20(126)	27.0(137)

Failure of G to increase GH post exercise despite the hyperglycemic effects of G suggests that the receptor sites may already be occupied by endogenous G. Felig, et.al. (NEJM 1972:287:184) have shown that G rises during E. The possibility that E causes release of other substances which either antagonize the action of G peripherally or competes for the same receptor site as G to cause the release of GH cannot, however, be ruled out.

UTILIZATION OF RADIORECEPTOR ASSAYS FOR QUANTITATION OF SOMATOMEDIN AND INSULIN-LIKE PEPTIDES IN PLASMA AND PLASMA EXTRACTS. Louis E. Underwood, Robert N. Marshall, Sandra J. Voina, and Judson J. Van Wyk, Univ. of North Carolina Sch. of Med., Dept. of Ped., Chapel Hill.

Somatomedin-C, isolated in our laboratory, has been radioiodinated and studied for its binding to human placental plasma membranes. Under optimal conditions, up to 20% of the labeled hormone is specifically bound to the receptor. The curves of competition for the somatomedin (SM) receptor produced by unextracted reference plasma paralleled those produced by a SM standard. Differences in the SM activity between hypopituitary dwarfs and normal children were readily apparent when 2-10 µl of plasma were compared to the reference plasma. The mean relative SM concentration for the 21 dwarfs was 0.35 ± 0.33 (1 S.D.), whereas the concentration for 19 normals was 1.21 ± 0.62. Administration of hGH to a hypopituitary dwarf induced a 3-fold increase in activity.

The SM receptor assay, when used in conjunction with a placental receptor assay using radioiodinated insulin, provides a rapid method for monitoring purification of SM peptides. The SM receptor is more sensitive to SM, but the insulin receptor recognizes a greater variety of insulin-like peptides. The placental insulin receptor cannot detect differences between hypopituitary and normal serum.

These results suggest that under physiologic conditions, somatomedin is preferentially bound to its own receptor rather than to the receptor shared with insulin.

AMINO ACID LEVELS IN FED AND FASTED JUVENILE DIABETICS. Paul M. Tocci and Andrew L. Taylor. (Intr. by William W. Cleveland) Univ. of Miami Sch. of Med., Jackson Memorial Hosp. Miami,Fla.

Circulating amino acid levels were measured in 5 juvenile diabetics and 4 controls one hour before and one hour after breakfast and lunch. All subjects were maintained on identical diets in a clinical research center prior to and during the testing. All diabetic subjects were treated with NPH insulin on one day and regular insulin on another. Taken as a group, the diabetics were not different from the controls except for a lower alanine content in the blood. This was true whether they were taking NPH or regular insulin treatment and regardless of the fed or fasted state. However, striking differences were noted within the diabetic group. Two of the 5 diabetic subjects exhibited very high glycine levels, high serine levels and low levels of alanine, valine, leucine and isoleucine. The other three exhibited low threonine, alanine and methionine levels. In all cases the differences were significant at the 0.01 level. There were no significant differences in the glucose levels of the two groups. The amino acid differences persisted even on the days when normal pre and post prandial blood glucoses were achieved with regular insulin treatment. The data indicate that differences in the circulating amino acids of juvenile diabetics do exist. Whether these differences are due to homeostatic control of the disease or whether they are due to fundamental sub-group differences in substrate utilization or absorption remains to be proven.

THE ROLE OF ADRENOCORTICOTROPHIC AND GROWTH HORMONE ON GLUCOCORTICOID SECRETION IN HYPOPITUITARY (HP) PATIENTS. Vanitha Vaidya, John S.Parks, Rebecca T. Kirkland, Alfred Tenore, Alfred M.Bongiovanni. Univ. of Pa. Dept.of Pediat. Children's Hosp. of Phila.

This study in HP was aimed at evaluating the role of Human Growth Hormone (HGH) on effect of Adrenocorticotrophic Hormone (ACTH) on glucocorticoid secretion. The 5 studied included 3 HP (with partial GH, Isolated GH, and GH and TSH deficiency respectively), and 2 normal controls. Urine collections for basal 17-hydroxysteroid (17H) were followed by collections with ACTH and GH alone, and in combination, for 3 consecutive days. The dosage, HGH 2 IU/day, ACTHAR GEL 25 U/M^2 q.12 hr. Mean basal 24-hr 17H excretion in the 3 HP was 0.38 mgm. There was a 15-fold rise to ACTH alone, a 37-fold rise with ACTH given a second time, whether given alone or in combination with HGH. The 17H excretion on HGH alone was not different from basal. The controls showed a 4-fold rise to the first stimulus with ACTH, and a 9-fold rise to ACTH given the 2nd time. There was no change with HGH administration. Results indicate no potentiation of HGH on the ACTH and hypopits are relatively deficient in 11-oxidoreductase. Tetrahydro F was a prominent component after ACTH in hypopits whereas Tetrahydro E was in the normal.