65 LH AND FSH RESPONSE TO LH-RH IN 27 GIRLS WITH PRECOCIOUS PUBERTY. R. Kauli, Z.Dickerman, R.Prager-Lewin and Z. Laron. Institute of Paediatric and Adolescent Endocrinology, Beilinson Medical Center, Israel.

An LH-RH test was done in 27 girls with precocious puberty (PP), 26 idiopathic and one with a brain tumor, and in 47 girls with normal puberty (pubertal staging according to Tanner). In 1o girls with PP tested before the age of 8 at an early pubertal stage (P2) basal LH (o.58±o.22 mIU/ml) and peak LH (2.1o±1.o5 mIU/ml) were within the normal prepubertal range (basal LH o.43±o.12, peak 3.o3±o.3). However, basal FSH (1.71±o.79) and peak releasable FSH (9.26±5.98) were significantly higher than in pre-pubertal controls (basal FSH o.6±o.2, peak 2.o±o.88). Peak FSH was also higher than peak values obtained from controls of the same pubertal stage (basal FSH 2.2±o.8, peak FSH 6.2±2.3).

17 girls were tested after age 8(8-11) in a later pubertal stage (P3). Basal LH (1.23±o.66) and peak releasable LH (15.o5±6.2) were not only higher for chronological age but peak LH was higher than the values observed in normal girls with a later pubertal stage (P4 - basal LH 1.6±o.2, peak 13.5±1.97). Similar results were observed with plasma FSH in more advanced PP; basal FSH was 1.84±o.8 and the peak was 5.4±2.7, corresponding with the values of more advanced pubertal stages in normal girls (P4 - basal FSH 2.o±o.7, peak FSH 4.5±1.6). It is concluded that in central PP gonadotrophins are secreted by a pattern similar to that in normal puberty at an earlier age, but that the intensity of individual secretion is greater than in controls both for age and stage.

66 EFFECT OF OP'DDD ON SERUM ANDROGENS IN A GIRL WITH METASTATIC VIRILIZING ADRENAL CARCINOMA. S.Korth-Schütz, P. Saenger, L.S.Levine, M.I.New, Dept. of Ped., Cornell Univ. Med. Col., New York, NY.

In metastatic adrenal carcinoma, op'DDD has been reported to decrease urinary 17-KS without causing regression of the tumor. This dissociation between the oncolytic and hormonal effects may be explained by our steroidal data. In a 5 y/o girl with metastatic adrenal carcinoma, serum T, Δ 4, DHEA, DS and urinary 17-KS were extremely elevated. With op'DDD treatment, the DS and 17-KS fell rapidly while there was a marked delay in the fall of free androgens. The persistence of free steroid secretion with decreased conjugate formation suggests that op'DDD specifically altered sulfatase activity before causing tumor necrosis and total decrease in steroidogenesis.

days ot op'DDD (total g)	Serum (ng%) T	DHT	Δ 4	E_1	E_2	D	(µg%) DS	Urine (mg/d) 17-KS
none	165	77	165o	6	2	1o3o	1477	59
12d (36)	144	1oo	1636	4.3	1.3	5o2	3o6	22.4
33d (72)	167	1oo	2148	4.2	1.3	2o4	75	9
55d (1o9)	<2	-	<2	-	-	<4	<1	1.5
normal	3-9	<4	2-35	<o.5	<o.5	2-115	2-27	< 2

A fall in urinary 17-KS may not indicate tumor regression and serum androgens seem to be a better indicator for the steroidogenic activity of a virilizing adrenal tumor. These data provide some new insight into a drug-steroid interaction.

67 HYPERALDOSTERONISM DUE TO BILATERAL ADRENAL HYPERPLASIA INDEPENDENT OF RENIN OR ACTH REGULATION. L.S.Levine, P.Landau, M.Hol brook, N.I.New, Dept.of Ped., Cornell Univ.Med. College, New York, N.Y.

A 11 y/o black boy with significant hypertension (16o/1oo) demonstrated hyperaldosteronism and low renins unaffected by ACTH, dexamethasone or alterations in dietary sodium. With sodium restriction, dexamethasone and Aldactone administration, blood pressure decreased but ACTH did not increase blood pressure. The plasma volume was increased in baseline state. Plasma aldo was very elevated and unchanged throughout while plasma DOC which was normal, increased with ACTH and decreased with dexamethasone. There was no change in DOC with changes in dietary sodium. The high plasma aldo and the normal DOC concentrations demonstrate that abnormal steroidogenesis in this child is restricted to the usually renin-controlled adrenal glomerulosa while the ACTH regulated fasciculata is normal. Arteriography suggested bilateral nodular adrenal hyperplasia. Bilateral renal vein renins were very low. Bilateral adrenal vein aldosterone concentrations were very high. The hypertension appears to be dependent on vascular volume and responds to measures which cause natriuresis. This is a well-documented case of primary hyperaldosteronism due to bilateral adrenal hyperplasia in which the excessive aldosterone secretion appears to be independent of regulation by renin or ACTH. Further, the continued elevation of blood pressure despite decreased aldosterone with metyrapone administration suggests a hormone other than aldosterone is causing hypertension.

68 Psychosocial dwarfism: Normal Somatomedin (SM) P. Saenger, L. Levine, E. Wiedemann, E. Schwartz, S. Korth-Schütz, M. New, Depts.Ped.& Med., Cornell Univ. Med.Col, New York, NY & VA Hospital, Bronx, NY.

Psychosocial Dwarfism (PSD) is a syndrome of severe growth failure in an abnormal home environment with a hostile or a depriving parent. We studied an 8 yr. old boy with PSD. While in the hospital after an initial 3 week period of poor growth he demonstrated rapid catch-up growth (growth velocity 3 cm/month). During the period of poor growth the mean caloric intake was 1663 cal/day, in the period of catch-up growth it was 1515 cal/day. The maximum stimulable growth hormone (GH) was 5.9 ng/ml initially and 13.6 ng/ml during rapid growth. SM was normal (o.79 and o.84 U/ml) in both periods. While under continued observation, with separation from his favorite nurse, his growth velocity dropped to o.5 cm/month, but there was no change in his stimulable GH or SM. The caloric intake remained unchanged at 15o4 cal/day. With the return of his favorite nurse he resumed his previous growth velocity of 3 cm/month and caloric intake during this period was 1521 cal/day. Again GH and SM were normal. He returned to his depriving home and growth velocity decelerated again to o.4 cm/month, SM acitvity remained normal at o.91 U/ml. We conclude from these data that growth failure in this patient with PSD is not due to caloric undernutrition nor to defective GH release or SM generation, but to an as yet unidentified factor inhibiting growth during emotional stress.