SUBACUTE THYROIDITIS IN AN ECTOPIC THYROID GLAND, EVALUATION OF THYROID-PITUITARY RESPONSE DURING HYPERTHYROID, HYPOTHYROID AND RECOVERY PHASE. Nezam Radfar, Frederic M. Kenny, P. Reed Larsen, Univ. of Pgh. Sch. of Med., Children's Hosp. of Pgh., Depts. of Ped. and Med., Pgh., Pa.

Subacute thyroiditis in an ectopic thyroid gland has not been reported. Our patient presented at age 4 10/12 with a mass in the left upper anterior neck. Scan 2 years previously when euthyroid revealed uptake only in the mass. On our evaluation she was euthyroid with enlargement of the mass, measuring 3.5 x 2 cm. RIA T4 and T3 were 8.1 ng (NL 5.1 - 11.5) and 191 ng/100 ml (NL adult 60 - 160)respectively. TSH was < 2.5 μU/ml with no increase following TRH. 2 wks later 24 hr RAI was 1.2%. TBG was 20 μg T4/100 ml (NL 16 - 24). The data were compatible with subacute thyroiditis so no therapy was given. Over the next 4 mos RIA T4 and T3 decreased to 0.8 μg/100 ml and 85 ng/100 ml respectively and TSH rose to > 60 μU/ml. During subsequent 4 mos of replacement therapy, the mass was not palpable. During 3 mos following stopping replacement therapy, sequential T3 72, 97, 115; and T4 1.8, 4.2, 6.8 increased. TSH decreased 120, 56, 42. 24 RAI uptake was 28% (slightly elevated). We conclude that pathological release of thyroid hormones occurring in subacute thyroiditis suppresses TRH-induced TSH release during initial phase of SAT. During recovery, the elevated RAI uptake is accompanied by high TSH levels which decrease as T3 and T4 return to normal.

PUBERTAL RICKETS WITH FAILURE OF RENAL PHOSPHATE AND CYCLIC AMP RESPONSE TO PARATHORMONE; AN EXPLANATION FOR PSEUDO HYPO-HYPER-PARATHYROIDISM. Nezam Radfar, Louie Linarelli, and Frederic M. Kenny, Univ. of Pgh. Sch. of Med., Children's Hosp. of Pgh., Dept. of Ped., Pittsburgh, Pa.

An 11 y o girl had unilateral ankle bowing during the premenarchial growth spurt while on normal dietary vitamin D. Generalized rickets on x-ray, slight decrease in serum Ca (8.8 - 9.7) and increase in P (5.4 - 6.3 mg%) and alk.phos. (8.4 BLU) were associated with failure to increase phosphate clearance with parathormone (pth). Malabsorption was excluded by appropriate studies. Rickets healed completely and ^{47}calcium absorption was normal during 2 years on vit D_2 50,000 IU/day, yet laminas dura were absent and remained so after adult height was reached. Restudy at age 15 years showed failure of increase in phosphate clearance, and CAMP (Δ 2.2 nanomoles/mg creat; vs 36 ± 0.7 SEM controls) with I.V. pth. Basal pth 640 pg/ml (normal 135 - 350) was elevated and decreased to normal 211 following Ca infusion, ruling out primary hyperparathyroidism.

We suggest that renal unresponsiveness to pth and failure to lower renal tubular cell P interfered with conversion of 25-OH-D2 → 1,25-OH-D2 with resulting hypocalcemia, rickets, and hyperparathyroidism during the stress of pubertal growth. This represents a novel explanation of pubertal onset rickets with hyperparathyroidism in absence of renal disease and is probably the basis of so called pseudo hypo-hyperparathyroidism.

PERCENTAGE OF DIALYZABLE ESTRADIOL-17β (DE_2) FROM BIRTH TO ADULTHOOD AND IN SEXUAL PRECOCITY AND PREMATURE THELARCHE. N. Radfar, C. Richards, M. Brych and F. M. Kenny, U. of Pgh. Sch. of Med.

We used equilibrium dialysis to determine % of radiolabeled E_2 unbound to sex hormone binding globulin (SHBG). See Table. DE_2 in cord blood was 6 x that of mothers. Therefore (unlike the mother), SHBG (or other circulating binding protein) affords the fetus little protection against high E_2 levels. A tissue E_2 binding protein could be protective. The lowest DE_2 was in prepubertal children protecting them from secondary effects of circulating E_2 (< 10 pg/ml). In sexual precocity and premature thelarche, DE_2 was similar to adult females; since some cases of thelarche do not have elevated serum E_2, the breast development could be 2° to increased DE_2. Estrogen induced increases in SHBG from adult males → females → pregnant females are responsible for the decrease in % DE_2 between those groups.

Condition	No. of cases	$\bar{X}$ % DE_2	± SEM	
Normal adult M	9	1.79	.07	*p< .01
Normal adult F	8	1.26	.06	*
Mothers at term	7	0.56	.03	*
Umbilical artery+vein	17	3.00	.18	*
M and F < 8 yrs old	7	0.73	.06	*
Sex. Proc. + Thel.	6	1.55	.20	

THE RESPONSE TO LRF IN IDIOPATHIC PRECOCIOUS PUBERTY (IPP) AND CONGENITAL ADRENAL HYPERPLASIA (CAH). E.O. Reiter, D.C.L. Savage, F.A. Conte, M.I. de Groppa, S.L. Kaplan, and M.M. Grumbach, Dept. Ped., Univ. Calif. San Francisco, S.F.

We have previously shown that luteinizing hormone releasing factor (LRF) elicits a sharp increase in releasable LH at puberty. To evaluate premature neural activation of the hypothalamic gonadostat, 100 μg of synthetic LRF was administered to 15 children with IPP (age 1-1/2 - 7-10/12 yr). The peak LH response (8.4±1.8 (SE) ng/ml LER 960) was significantly greater in IPP than in normal pubertal (Stage P2-4) children (4.9±0.3) as well as normal prepubertal (P1) children (1.8±.14). The peak FSH response (8.4±1.4 LER 869) in IPP was similar to P2-4 (6.0±1.2) or P1 (5.3±1.9) girls. In 4 children with precocious thelarche or adrenarche, the LH response did not differ from P1. 100 μg of LRF was administered to 7 prepubertal, glucocorticoid-treated girls with CAH (age 3-6/12 - 9-8/12). The LH peak (2.0±0.4) did not differ from P1. The peak plasma FSH (8.1±1.6) also did not differ from P1 or P2-4 girls, but was significantly greater than in P1 and P2-4 boys. These results suggest that: (1) a pubertal pattern of LH release occurs in children with IPP, presumably secondary to a premature increase in endogenous secretion of LRF; (2) exposure to high levels of sex steroids in CAH during fetal life and infancy did not seem to cause an early maturation of the pituitary response to LRF; (3) the normal female pattern of LRF-induced FSH release (which is significantly greater than in males) was not altered by the prenatal androgen exposure.

SINGLE DAILY DOSE OF ANTITHYROID MEDICATION IN ADOLESCENTS WITH HYPERTHYROIDISM. Joseph P. Repice, Thomas Aceto, Jr., Kathleen Murray, Pediatrics. Sch. of Med., SUNY,Buffalo, N.Y.

Many adolescent patients with hyperthyroidism fail to take antithyroid medication several times daily and, consequently, remain hyperthyroid. Greer,(NEJM,272:888,65) and Barnes, (JCEM,35:250,72) have reported successful therapy of hyperthyroid adults using a large single daily dose (SDD) of propylthiouracil(PTU)or methimazole(M). We have studied 9 adolescents with hyperthyroidism,7 initially treated with a SDD of PTU(300mg/m^2/24hr)and 2 with a SDD of M(30mg/m^2/24hr). Over a 3 month follow-up period, we have monitored several parameters: symptoms of patient;opinion of physician re: thyroid status;weight change;goiter size; WBC, and serum thyroxine(CPB). After 3 months,5 patients were asymptomatic and 4 patients experienced minimal symptoms. The physician considered 2 patients euthyroid and 7 definitely improved but slightly hyperthyroid.Gain in weight, expressed as mean ±S.E. was 3.0 ± 1.3kg(range:-2.5 to 10.9); P<0.035. Goiter size decreased in 7,remained the same in 1, and increased in 1 patient. At no time did the WBC fall below 4,000/cc in any of the 9 patients. Initial thyroxine level was 21.6 ± 1.7ug%, (range 18.1 to 31.0). Decrease in thyroxine was 10.0 ± 2.2 (range 2.0 to 24.8). Although follow-up has been relatively short, these results suggest that a SDD of antithyroid medication is successful and safe in inducing a remission in adolescents with hyperthyroidism and would enhance compliance.

EPISODIC GROWTH OF CHILDREN WITH HYPOPITUITARISM RECEIVING CONTINUOUS HUMAN GROWTH HORMONE (HGH) THERAPY. Iraj Rezvani and Angelo M. DiGeorge. St.Christopher's Hospital for Children Dept. Ped.,Temple Univ. Sch. Med., Philadelphia, Pennsylvania.

It is generally recognized that over short periods of time normal growth is not stable but rather episodic. In addition to illness and nutrition, there are seasonal influences and other unknown factors which alter growth velocity of normal children. The role of growth hormone in these changes is a moot question. We examined the growth rates of hypopituitary children while on constant therapy with HGH. Thirteen patients with idiopathic HGH deficiency (5-18 years old) were treated for 9 to 36 months with HGH (2 U three times weekly). The growth rate for each patient was carefully measured every 26 to 80 days under controlled conditions and was projected as growth velocity per 365 days (examples given in table). Marked variations of growth rates were noted in each patient which were unrelated to season, intercurrent infection or activity. These data indicate that the normal episodic changes in growth rate are not obliterated by treatment with HGH and probably are not related to variations in growth hormone levels.

Projected Growth Velocity (cm Per year)/ Days of HGH Therapy

E.P.	L.N.	E.P.	J.M.	W.B.	M.S.	A.S.
12.5/37	8.0/43	8.2/56	15.5/74	9.5/73	3.8/60	9.2/63
4.4/26	11.5/40	8.2/28	12.2/37	6.8/51	8.7/53	1.6/70
14.2/26	2.7/43	1.0/42	7.6/61	13.3/26	7.3/63	1.9/59
8.9/39	19.7/47	1.0/42	13.3/26	17.2/40	16.5/56	6.5/53
14.1/49	5.2/44	3.9/35	8.7/53	4.6/50	7.3/63	2.1/55