7 DETERMINANTS OF SERUM URATE IN ADOLESCENCE
William R. Harlan, Joan Cornoni-Huntley, Paul E. Leaverton (sponsored by William J. Oliver) National Center for Health Statistics, Hyattsville, Maryland

During adolescence, serum uric acid concentration increases and adult levels are attained. Physiologic factors related to adolescent serum urate levels were investigated using data from the U.S. Health Examination Survey. The survey population comprised 6768 youths, aged 12 to 17 years, selected to represent the U.S. population with respect to age, sex, race, and demographic status. Serum urate increases markedly during pubescence in males; in females the increase is modest. In males, the increase is strongly associated with sexual maturation (Tanner staging) and with skeletal age (radiographic assessment). The relationships among skeletal age, serum urate, and Tanner stage in males are strong enough to permit use of urate concentration in assessing sexual and physiological maturation. In later adolescence, body composition (body mass index and skinfold thickness), and hematocrit are more closely related to serum urate than is sexual maturation. Blood pressure and serum cholesterol have significant but weaker relationships. These relationships are similar to those in adults. Serum urate concentrations are 0.5 mg/dl lower in black male and female youths of all ages, and this consistent difference is unrelated to other factors. During adolescence, the early rise in serum urate concentration is related to physiological maturation, while the levels in late adolescence are similar to those in adults.

8 ADOLESCENT SECONDARY AMENORRHEA: ASSOCIATION WITH HYPOTHALAMIC HYPOTHYROIDISM.
Michael S. Kramer, Arieh Kauschansky, and Myron Genel (Spon. by Howard A. Pearson), Yale University School of Medicine, Yale-New Haven Hospital, Department of Pediatrics, New Haven.

In adolescent girls, secondary amenorrhea can result from a variety of physiologic and psychologic disturbances. Previous reports associating amenorrhea and primary hypothyroidism have not distinguished between the alternative etiologic roles of thyroxine deficiency and hyperprolactinemia.

We have evaluated two girls with secondary amenorrhea who had clinical and chemical evidence of hypothyroidism. Both had low basal T_4's (0.8 and 3.8 µg%), calculated free T_4's (0.1 and 0.7 µg%), and T_3's (51 and 81 ng%). Both had undetectable basal TSH with normal TSH response to TRH. Basal FSH and LH were normal, as was the response to LHRH. Basal prolactins were 6 and 14 ng/ml, and both girls had growth hormone responses of $\geq$15 ng/ml in response to insulin-induced hypoglycemia. Pituitary-adrenal function and reserve were also normal. In neither case were there any historical, physical, or laboratory features compatible with anorexia nervosa. After treatment with l-thyroxine, both girls had a resumption in menses.

These two adolescent girls thus appear to have isolated hypothalamic hypothyroidism. The associated secondary amenorrhea demonstrates that thyroid deficiency alone, without hyperprolactinemia, can interfere with normal hypothalamic-pituitary-ovarian function.

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9 ASSESSMENT OF LEFT VENTRICULAR HYPERTROPHY IN HYPERTENSIVE ADOLESCENTS USING ECHO, ECG, AND X-RAY
W. Pennock Laird, David E. Fixler, F. Doug Huffines. University of Texas Health Science Center, Department of Pediatrics, Dallas, Texas.

We performed chest x-rays, electrocardiograms (ECGs), and echocardiograms (ECHOs) on 35 hypertensive and 35 normotensive subjects 14-16 years of age who were matched for age, race, sex, and body size (ponderal index). All hypertensive patients had elevation of systolic and/or diastolic blood pressure at or above the 95th percentile for age on 3 consecutive examinations. No patient in either group showed cardiomegaly on chest x-ray. Five hypertensive patients (14%) and 7 control subjects (20%) had ECG criteria for LVH, indicating no significant difference between the groups. Analysis of ECHOs showed the mean left ventricular wall thickness (LVT) in hypertensives was 7.8mm±0.87 (SD), compared with 6.5mm±0.81 in the controls. Left ventricular mass indexed to body surface area (LVM/BSA) averaged 82±15 and 70±15 respectively. LVT was then also indexed to BSA and using our control data we defined the upper limits of normal in terms of the 90th percentile values: for LVT/BSA this was 0.470, and for LVM/BSA this was 100. Of hypertensive patients, 23% had LVT/BSA above the 90th percentile and 17% had LVM/BSA, above this level. For controls, only 3% had elevated LVT/BSA, and 6% had elevated LVM/BSA. These findings suggest that ECHO may be a more useful method than either chest x-ray or ECG to detect LVH in hypertensive adolescents.

10 CYTOMEGALOVIRUS (CMV) IN PREGNANT ADOLESCENTS AND THEIR OFFSPRING
David J. Lang, Selman I. Welt, John F. Kummer, Aglaia N. O'Quinn, Robert J. Thompson, Jr., Duke Univ. Med. Ctr., Depts. of Peds. and Ob-Gyn, Durham, N.C.

A prospective study has been established of CMV among pregnant adolescents and their offspring. Results derived from studies of 53 mothers (mean age 15.6 yrs.) and 54 infants comprise this report. ¶ Thirty-seven of the 53 (81%) were CMV(CF) seropositive when first tested (mean estimated gestational age 23 wks). Twenty-three mothers (43% of total and 62% of seropositives) had one or more positive cultures for CMV and three of their infants (5.5% of total) were congenitally infected with CMV. ¶ The geometric mean (GMT) of maximum CMV titers among virus-positive mothers was 48.5 in contrast to 3.7 among those who were virus-negative ($p<0.01$). Twelve of 13 mothers with anti CMV titers of 64 or higher were virus-positive and three gave birth to congenitally infected infants. The CMV GMT was 39 in infants born to virus positive mothers compared to a CMV GMT of 4 in babies of virus-negative mothers ($p<0.01$). All (11) cord blood samples with a titer $\geq$64 were obtained from infants born to virus-positive mothers and included those congenitally infected. The determination of maternal and neonatal CMV antibody thus identified a particularly high risk subgroup. ¶ Pregnant adolescents and their offspring may experience frequent CMV infections (exogenous or endogenous in origin). The long term impact of these infections may be a factor in the poor outcome of some teenage pregnancies.

11 DISPARITY BETWEEN BIOACTIVE AND IMMUNOACTIVE LUTEINIZING HORMONE IN GIRLS WITH GONADAL DYSGENESIS: EFFECT OF ESTROGEN TREATMENT.
Anne W. Lucky, Robert L. Rosenfield and Robert W. Rebar, Univ. of Chicago Pritzker Sch. of Med., Wyler Children's Hospital, Dept. of Ped., Chicago, and Reproduction Research Branch, N.I.C.H.D., N.I.H., Bethesda, Md.

We have studied the ratio of bioactive to immunoactive (B/I) luteinizing hormone (LH) in eight girls with gonadal dysgenesis before and after estrogen replacement therapy with low doses of oral ethinyl estradiol. The estrogen suppression of bioactive LH, as measured by a rat interstitial cell testosterone assay (RICT), was more profound than the suppression of radioimmunoassayable (RIA) LH in five girls who had elevated basal serum LH levels: The B/I before therapy was significantly higher ($p<0.005$) at 2.78±0.12 (n=6) than after three months of 10 µg of ethinyl estradiol (1.44±0.11, n=5), and after three subsequent months of 20 µg of ethinyl estradiol (0.60±0.08, n=3). In two younger patients without elevated basal RIA LH, however, the B/I was low. Estradiol 17-β in concentrations of 0.1, 5 and 10 ng/ml had no effect on testosterone production in the RICT assay.

These data suggest that estrogen affects the qualitative form as well as the quantitative amount of LH secreted, possibly via a structural change in the LH molecule, or by inhibition of another substance with LH-like bioactivity, or by other mechanisms as yet undefined.

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Abstract withdrawn