367 EFFECTS OF INDOMETHACIN ON FLUID INTAKE, URINE VOLUME, URINARY PROSTAGLANDIN E-LIKE MATERIAL AND KALLIKREIN IN VASOPRESSIN RESISTANT DIABETES INSIPIDUS. Hulda J. Wohltmann, Perry V. Halushka, Philip J. Privitera, Harry S. Margolius, and Richard G. Wagner. (Spon. by Jean H. Thurston). Med. U. of S. C., Depts. of Ped., Med., and Pharm., Charleston.

Because indomethacin (I) potentiates the effects of vasopressin (V) in experimental animals, and because of our previous observations that I decreased urine volume (UV), iPGE and kallikrein (UK) in Bartter's syndrome, we studied the effects of inhibition of PGE synthesis with I in a child with V resistant DI. A male (6 yrs.), first presented at 3 mos. with normokalemia, hypernatremia, hyperchloridemia, hyperreninemia, hyperaldosteronism, and J-G cell hyperplasia. Electrolyte and water balance were monitored daily before (4 days), during (5 days), and after (4 days) treatment with I (1-2 mg/kg/day). Placebo was given before and after I. I significantly decreased intake from 8250±288 to 5507±493 cc/24 hr., (P<.02); UV from 6366±717 to 4519±225 cc/24 hr., (P<.05); U iPGE from 808±115 to 421±82 ng/gm creat., (P<.02); and UK from 4.1±0.5 to 3.2±0.1 E.U. (P<.05). After stopping I, intake, UV, U iPGE, and UK rose by the fourth day to pre I levels. Uosm. (187±3), serum osm (285±1), urine cAMP (3.14 uMoles/M^2/24 hrs.), and standing plasma renin activity (9.2 ng/ml/hr.) were not significantly changed by I. Conclusion: The results are consistent with inhibition of endogenous PGE synthesis mediating the effects of I and again demonstrates a relation between renal PGE and UK. The failure to show a significant increase in Uosm. raises the possibility of a non-renal site of action for I. (GM 20387 and HL 17705).

368 LEYDIG-CELL FAILURE AND HYPOGONADISM IN A 21 YEAR OLD MALE. Richard Hk. Wu, (Spon. by Murray Davidson). Albert Einstein Coll. of Med., Bronx-Lebanon Hosp. Ctr. and Montefiore Hosp. and Med. Ctr., Depts. of Pediatrics, Bronx, New York.

A 21 year old male, with normal stature and an XY karyotype, presented with sparse pubic hair, no axillary hair, microphallus and otherwise normal external genitalia. Leydig-cell dysplasia and tubular degeneration were found on testicular biopsy. The mean 24 hr. plasma luteinizing hormone (LH) and follicle stimulating hormone (FSH) concentrations were 50 and 8 mIU/ml respectively. LH levels were higher during sleep than during waking hours. There were no sleep-wake differences in FSH levels. Plasma LH rose to 115 mIU/ml in response to 25 μg of LH releasing factor (LH-RF). Plasma FSH responded poorly to LH-RF. The mean 24 hr. plasma concentrations of testosterone (T) and dihydrotestosterone (DHT) were 95 and 84 ng/dl respectively. Plasma T rose from 120 to 160 ng/dl in response to 20,000 units of human chorionic gonadotropin (HCG), given over a 4 day period. Following an additional 20,000 units of HCG, given in 4 equal weekly doses, the plasma T concentration was 120 ng/dl. These data show that this patient has an unusual form of testicular degeneration characterized by diminished T production and poor T response to HCG. Normal concentrations of FSH suggest sufficient inhibin production. The concentrations of T and DHT in utero, must have been adequate to enable normal differentiation of the external genitalia. This patient's abnormality fits into a spectrum of anomalies with Leydig-cell agenesis at one end and premature Leydig-cell failure (early climacteric) at the other.

369 COMPARATIVE USE OF SERUM 17-HYDROXYPROGESTERONE (17-OHP) AND DEHYDROEPIANDROSTERONE SULFATE (DS) IN CONGENITAL ADRENAL HYPERPLASIA (CAH). William B. Zipf, George E. Bacon, Robert P. Kelch, and Martha L. Spencer, Dept. of Ped., Univ. of Mich., Ann Arbor.

Previous studies have demonstrated the usefulness of serum 17-OHP determinations in children with CAH. Recent reports have suggested that serum levels of DS, an adrenal androgen, might better reflect degree of control and be simpler to measure. Hourly serum samples x 24 hr were assayed for 17-OHP and DS in 6 patients aged 10-19 yrs with 21-hydroxylase deficiency (salt losing, N=4; non-salt losing, N=2) during therapy with Prednisone (P) and after glucocorticoid treatment had been withdrawn for 3 days. After withdrawal of P the 24 hr mean serum 17-OHP levels increased in all patients (64% to 900%) and ranged from 3 mcg/dl to 28 mcg/dl. Urinary 17-ketosteroids increased in 5 of 6 patients. There was no consistent change in mean DS levels which ranged from 1 mcg/dl to 114 mcg/dl on Rx and 6 mcg/dl to 82 mcg/dl off Rx. DS levels increased in 4 patients and decreased in 2 while off Rx. All DS levels on and off Rx were in the normal range for age. Patients considered poorly controlled by urinary 17-ketosteroids and serum 17-OH-P did not have elevated DS. No diurnal changes in DS levels were evident. Serum DS levels from 101 patients aged 2-19 yrs without adrenal pathology were consistent with other reports and showed an increase with age of patient: ($\bar{x}$ ± S.D.: 2-8 yrs = 29 ± 32; 9-12 yrs = 57 ± 49; 13-19 yrs = 131 ± 78). Our data indicate that DS concentrations do not become abnormally high after acute withdrawal of glucocorticoid and would not be a reliable indicator of control in children with CAH.

370 EFFECT OF SYNTHETIC ANDROGENS ON THE HYPOTHALAMIC-PITUITARY-GONADAL AXIS (HPGA) IN BOYS WITH CONSTITUTIONALLY DELAYED GROWTH (CD) AND ISOLATED GROWTH HORMONE (GH) DEFICIENCY. William B. Zipf, Nancy J. Hopwood, and Robert P. Kelch. Dept. of Ped., Univ. of Mich., Ann Arbor.

Fourteen boys with CD were tested with gonadotropin-releasing hormone (GnRH) before and at the end of 6 mo Rx with Fluoxymesterone (F), 10 mg/d (Group I), or Oxandrolone (Ox), 0.15-0.2 mg/kg/d (Group II), to assess the effects of these androgens on the HPGA. At initiation of Rx, 7 boys in Group I, 15.1-16.7 yrs, had bone ages (BA) 12-14 yrs and were in stage 2 puberty while 7 boys in Group II, 10.2-15.9 yrs had BA 7-13 and most were prepubertal. PreRx Δmax LH rise to GnRH was normal in each boy: Grp I, $\bar{x}$ 31.5 mIU/ml (range 16-55); Grp II, $\bar{x}$ 18.2 mIU/ml (range 7-32). Δ Max FSH responses were variable. At the end of 6 mo Rx, all but one boy in Grp I and 4/7 boys in Grp II had significantly lower Δmax LH responses (34-89% reduction) without consistent change in Δmax FSH. Plasma testosterone (T) was reduced by 57-96% when preRx level was >1 ng/ml. Eight boys were restudied 6 mo after completion of Rx: ΔMax LH and T were = preRx levels indicating recovery of the HPGA. All had acceleration of linear growth (3.8-8.9 cm/6 mo) and progressive testicular enlargement without excessive BA advancement. Seven prepubertal boys treated with HGH 2 IU 3x/wk for GH deficiency were studied before and after 6 mo Ox Rx. Both $\bar{x}$ Δmax LH and FSH and basal LH were significantly lower during Rx. Non-aromatizable anabolic steroids reversibly suppress GnRH responsiveness in both prepubertal and pubertal boys and basal LH in prepubertal boys. Short term androgen Rx facilitates physical and psychological maturation in selected boys with CD.

EPIDEMIOLOGY

371 EFFECT OF ANEMIA ON BLOOD AND TISSUE LEAD OF RATS. Carol R. Angle and Matilda S. McIntire, Univ. of Neb. Coll. of Med., Dept. of Ped., Omaha.

Despite the epidemiologic coexistence of anemia and plumbism, the contribution of preexisting anemia to the risk for lead poisoning is incompletely defined. Rats were given oral lead, 54 mg/kg/day x 7. One group of 16 was made anemic (A) by bleeding off 25% of the blood volume on days 1, 3, and 5; sham phlebotomy was done on the same days in 12 non-anemic (NA) animals. Blood lead (Pb-B) and red cell lead (Pb-Rbc) were comparable until after day 5 when the hematocrit (Hct) of A dropped below 30%. At sacrifice on day 7 the comparable values, mean ± SE, were significantly different (*, p < .01) in the anemic (A) rats.

	Hct	Pb-B μg/dl	Pb-Rbc μg/dl
A	25.9 ± 0.6*	77.7 ± 10.6*	211.1 ± 38.3*
NA	41.5 ± 0.9	30.8 ± 2.9	59.7 ± 5.5

as were the tissue leads, μg/gm:

	Kidney	Liver	Brain
A	12.1 ± 1.3*	2.8 ± 0.3*	0.58 ± .09*
NA	7.3 ± 1.2	1.6 ± 0.3	0.37 ± .04

These generalized increases support an increased absorption of lead in anemia consistent with the clinical predilection to lead toxicity of children and adults with low levels of hemoglobin.

372 Experiences With A Multiple Resistant Klebsiella pneumoniae In An Infant Intensive Care Unit. Allan M. Arbeter, Carol Aff, Pamela Dill, Helen Widzer and Stanley A. Plotkin, University of Pennsylvania School of Medicine, Department of Pediatrics, Children's Hospital of Philadelphia.

The emergence of multiple resistant bacteria introduces a difficult era of intensive care medicine. Rectal colonization of neonates in an infant intensive care unit (IICU) with a multiple resistant Klebsiella pneumoniae (MRKp.), resulted in severe disease, changes in routine antibiotic usage to more toxic agents, and imposed severe limitations of a vital community service unit. Colonization with MRKp. (resistant to ampicillin, carbenicillin, cephalosporins, kanamycin, gentamicin) occurred from 6/75 until 7/76. Two epidemic periods of colonization were noted with up to 16 neonates colonized and the occurrence of clinical disease, including septicemia, peritonitis and necrotizing enterocolitis. Associated with increased MRKp. isolation was the emergence of other organisms with identical multiple resistances.

Risk factors for colonization included 1) prior treatment with an aminoglycoside 2) the prolonged presence in the IICU of a colonized infant 3) colonization of faucets and drains.

Control measures included 1) regularly scheduled rectal swab cultures on a gentamicin containing McConkey agar 2) temporary cessation of aminoglycoside use, substituting chloramphenicol or polymyxin B 3) cohorting colonized neonates into an isolation unit 4) elimination of the organism from environmental sources and 5) continued monitoring. These measures were successful but costly.