- 80 *Placental Passage and Fetal Secretion of Aldosterone.* FRANCIS J. BAYARD, ISADORE G. ANCES, ALAN J. TAPPER, VIRGINIA V. WELDON, AVINOAM KOWARSKI and CLAUDE J. MIGEON, Dept. of Ped., The Johns Hopkins Univ. and the Dept. of Obstet. and Gynecol., Univ. of Maryland, Baltimore, Md.

The transplacental passage and production of aldosterone by the mother and fetus were studied near term during a constant infusion of 1,2-³H-aldosterone to 9 mothers at the time of elective cesarean section.

This study showed that there was a transplacental passage of aldosterone. The fetal plasma levels of 1,2-³H-aldosterone, expressed as percentage of the maternal values, ranged from 40.0 to 79.2%. The levels of non-labeled hormone, determined by a double isotope dilution technique, were consistently higher in the fetus than in the mother (mean $\pm$ SD = 17.2 ± 9.5 μ g/100 ml and 3.7 ± 1.4 μ g/100 ml, respectively). Since the specific activities of aldosterone were lower in the fetus than in the mother (6.5 to 23.4%), and if one assumes that equilibrium between maternal and fetal compartments was reached after more than 2 h of constant infusion, it can be concluded that the fetus secretes aldosterone.

The metabolic clearance rate of aldosterone was not modified by pregnancy.

The steroid blood production of the mothers was about twice the non-pregnant values. The relatively small increments might be related to the fact that our subjects were supine for at least 10 h at the time of the study.

- 81 *Effects of Castration and 5-methoxy-N-acetyltryptamine (Melatonin) Upon Luteinizing (LH) and Follicle Stimulating (FSH) Hormones in 21-day Male Rats.* ALLEN W. ROOT and R. DAVID RUSS, Temple Univ. Sch. of Med., and Albert Einstein Med. Center, Philadelphia, Pa.

Male rats, maintained under controlled environmental conditions with 14 h of light daily, were orchietomized or sham-operated on the 21st day and sacrificed on the 42nd day of life. Melatonin (200 μ g) or diluent (2% ethanol) was administered i.p. daily from the 21st through the 41st days of life to groups of intact and castrated animals. Pituitary and serum LH and FSH were measured by double antibody radioimmunoassays. In diluent treated-orchietomized rats the pituitary weight, pituitary LH content and concentration and serum LH and FSH levels were significantly ($p < 0.01$) higher and the pituitary FSH concentration was significantly (< 0.01) lower than in diluent treated intact animals. The administration of melatonin to intact animals was associated with significantly ($p < 0.05$) decreased pituitary LH content and testicular weight, but was without effect upon pituitary FSH content or upon serum LH and FSH concentrations. The administration of melatonin to orchietomized animals was without apparent effect. It is concluded that under the conditions of the present experiment; (1) there is increased secretion of LH and FSH following castration of the juvenile male rat; (2) melatonin may suppress LH synthesis in intact animals; (3) melatonin is unable to suppress LH secretion in castrated 21 day male rats; (4) melatonin does not effect FSH secretion in either intact or orchietomized juvenile rats.

- 82 *Erythrocyte Na Flux in Cushing's Disease.* G. GALL, J. MELBY and R. KLEIN, Boston Univ. Sch. of Med.

This study was designed to establish whether cortisol which increases renal Na retention and K excretion also has the same effect as aldosterone on Na transport across other cell membranes. At last years meetings we reported increased erythrocyte Na flux rates in patients with primary aldosteronism. Since then we demonstrated that these patients have increased Na+K ATPase activity and that aldosterone increases active Na transport *in vitro*. Erythrocyte ghost Na flux, intracellular H₂O, Na, and K, and Na+K dependent ATPase were measured in control subjects and patients with primary hyperaldosteronism and Cushing's disease. Mean $\pm$ SEM are given in the table.

	Active trans	Passive leak	ATPase Na+K Dep.
Control	0.41 ± 0.02	0.21 ± 0.01	0.054 ± 0.004
Aldost	$0.80 \pm 0.15^*$	0.23 ± 0.02	$0.093 \pm 0.008^*$
Cushings	$1.46 \pm 0.20^*$	0.24 ± 0.01	0.126^*

	H ₂ O _i	Na _i	K _i
	Kg RBC		
Control	664 ± 2.47	8.9 ± 0.30	97.2 ± 0.92
Aldost	659 ± 6.90	10.5 ± 0.74	96.5 ± 5.1
Cushings	665	6.5	99.0

* Indicates difference from control was $p < 0.01$.

When 10^{-6} M aldosterone and 10^{-7} M cortisol were added *in vitro* to normal erythrocyte ghosts Na efflux rates increased by 37% and 76%, without change in the passive leak. Thus aldosterone and cortisol had similar effects on Na flux and Na+K dependent ATPase activity, at secretory rates approximately 10^3 greater for cortisol than for aldosterone.

- 83 *The Development and Characterization of Diabetes Mellitus in Cystic Fibrosis.* DOUGLAS S. HOLSCLOW, THEODORE AVRUSKIN, STEWART J. SOELDNER and HARRY SHWACHMAN, Harvard Med. Sch., Children's Hosp. Med. Center, Dept. of Ped. and Med., Boston, Mass.

The association of cystic fibrosis with chemical glucose intolerance and overt diabetes mellitus is becoming increasingly more common. It is not yet clear whether this results from genetic intertwining or pancreatic islet cell destruction or both. 200 children with cystic fibrosis over 7 years of age were screened for glucose intolerance. Factors as sex, age, severity of cystic fibrosis and family history of diabetes mellitus were analyzed. 48 of the 200 were classified as glucose intolerant (1-h blood sugar over 175 mg%). 3-h oral glucose tolerance tests were performed on these 48. Significant impairment of glucose tolerance was demonstrated in 85% (values greater than 97th percentile of normal established by PICKENS *et al.*). No relation was established as to sex, age or ideal body weight. Two and one half years later, these 48 were re-studied. Six had developed overt insulin requiring diabetes mellitus. These were evaluated by blood glucose measurements and serum immunoreactive insulin and growth hormone assays following oral and intravenous glucose, intravenous tolbutamide and intramuscular glucagon tolerance tests. Findings compatible with a severe insulin deficient state were obtained.

- 84 *A Role for Cyclic AMP in the Alpha Adrenergic Inhibition of Insulin Release by Hypoxia.* DAVID BAUM and DANIEL PORTE, Jr., Univ. of Wash. Sch. of Med., Seattle, Wash.