504 HY INTERMEDIATE PHENOTYPE: A POSSIBLE CAUSE OF TESTICULAR DYSGENESIS. Thomas A. Wilson and Stephen S. Wachtel. SUNY at Stony Brook and New York Hospital-Cornell Medical Center, Divisions of Pediatric Endocrinology, Stony Brook and New York. (Sponsored by John C. Partin).

The HY antigen is closely associated with the presence of testicular tissue which is thought to be induced during embryogenesis by the interaction of HY and its receptor. In coded samples, we examined the HY antigen in peripheral lymphocytes from a patient with male pseudohermaphroditism and dysgenetic testes whose karyotype in lymphocytes and gonadal fibroblasts was 46 XY. Results are as follows (mean + SD):

HY Antibody dilutions:	1.2	1:4	1:8	(N)
Unabsorbed:	49.5+4.8	47.9+5.7	50.8+6	(8)
Control female:	51.9+6.1	48.6+3.9	48.8+5.8	(10)
Control male:	32.4+5.8	31.7+4.9	31.1+4.5	(10)
Patient:	40.0+6.7	38.1+6.5	39.1+7.8	(7)
Mother:	51.1+5.4	50.4+4.3	49+4.1	(4)
Father:	35.4+4.2	34.3+1.9	34.3+3.4	(4)

The patient's lymphocytes exhibited an HY phenotype intermediate between that of the normal male and normal female and between that of the patient's mother and father. These results indicate a correlation between HY phenotype and abnormal testicular development and suggests the possibility that a partial deficiency of HY may have been responsible for the testicular dysgenesis in this patient.

505 THE ADRENAL SUPPRESSIVE EFFECT OF HYDROCORTISONE IN CONGENITAL ADRENAL HYPERPLASIA (CAH) IS NOT AFFECTED BY VARYING THE DOSE SCHEDULE. J. Winterer, G.P. Chrousos, D.L. Loriaux, and G.B. Cutler, Jr. (Spon. by David Nelson) DEB, NICHD, Bethesda, MD 20205

Conventional therapy of CAH does not yet achieve normal adult height (NEJM 299:1392, '78). We postulated that standard hydrocortisone (F) dose schedules might yield periods of undertreatment or overtreatment throughout the circadian cycle, and that an optimal dose schedule might improve the results of treatment. We therefore evaluated the adrenal suppressive effect of 5 dose schedules of F in 6 patients with 21-hydroxylase deficiency. The dose was 12.5/m^2/d, given in a random sequence for 6 weeks according to each of the following schedules: I, 3/3(AM); II, 2/3 (AM), 1/3(HS); III, 1/3 (AM), 1/3 (NOON), 1/3 (HS); IV, 1/3 (AM), 2/3 (HS); V, 3/3 (HS). We collected blood Q2Hx24 hrs during the last day of each schedule to measure plasma 17 hydroxyprogesterone (17OHP) and F. We collected a 24-hr urine to measure pregnanetriol (PTRIOL), 17 ketosteroids (17KS) and urine free F (UFC) (mean ± SEM):

	I	II	III	IV	V
17OHP (ng/dl)	4114 +1315	4775 +1901	4201 +1363	5606 +2427	3120 +1341
PTRIOL (mg/d)	16+6	13+6	16+6	16+6	18+6
17KS (mg/d)	15+5	15+4	12+3	16+5	16+4

None of the schedules caused a significant improvement of plasma 17OHP or its urinary metabolites. We conclude that the total daily dose of hydrocortisone appears to determine the degree of adrenal suppression irrespective of the dose schedule.

† **506** QUALITATIVE DIFFERENCES IN GLUCOSE HOMEOSTASIS BETWEEN OBESITY OF PRADER-WILLI SYNDROME (PWS) AND NORMAL CHILDHOOD OBESITY (NL-OB). W.B. Zipf, T.H. O'Dorisio, S. Cataland, Ohio State University College of Medicine, Columbus.

Obesity is associated with insulin resistance and glucose intolerance. Our studies of Nl-ob and obese PWS children suggest another factor contributes to pathology of glucose homeostasis, in addition to increased adipose tissue. We studied 32 Nl-ob children (age 3-18 yrs) and 18 PWS children (age 6-19 yrs) given a mixed liquid "milkshake" meal (4 Kcal/kg). Serum levels of pancreatic polypeptide (PP), glucose (G), insulin (I) and gastric inhibitory polypeptide (GIP) were obtained at 0,15,30,45,60,90, 120,180 min. There was no significant difference between weights of Nl-ob pts. (172±32%; mean±SD) and PWS pts. were 205±61% of ideal body weight for height.

Group	N	Peak G mg/dl	Peak I µIU/ml	Peak GIP pg/ml	Peak PP pg/ml
Nl-ob	32	111±2	114±10	2252±10	244±56
PWS	18	102±3*	75±12*	1613±197*	111±35*

(Values are mean±SE; * Significantly less than Nl-ob)

The PWS children with hyperphagia induced obesity had better glucose homeostasis and less insulin resistance than did the age matched, equally obese, normal children. Insulin resistance and glucose intolerance are not a uniform consequence of obesity; a reason for individual differences in susceptibility is not known. The differences between our groups may reflect a relative absence of an insulin hypersecretory-insulin resistance factor in the PWS group. Our data support the hypothesis that GIP hypersecretion may be causal. The PP deficiency may play a secondary role.

507 RESPONSE OF FETAL VASOACTIVE MEDIATORS TO INDUCED HYPOTENSION. Alan Zubrow, Salha Daniel, Raymond Stark, Kazim Husain, Stanley James. Harlem Hosp. & Babies Hosp. Columbia Univ., Coll. P&S, Depts. of Ped. & Anes., N. Y.

To characterize the fetal response to change in blood pressure vasoactive mediators were measured during and after 60 min of sodium nitroprusside induced hypotension. Fetal arterial blood pressure was lowered by 10-20% in seven chronically catheterized fetal lambs (116-135 d). Fetal pHa (7.39±0.01), $paCO_2$ (40.7± 1.1 torr), PaO_2 (22.1±0.9 torr), Osm (297.3±3 mosm/kg), and Na (144.5±1.4 meq/l remained unchanged. Results (mean ±SEM, analyzed using ANOVA) on log plasma vasopressin (V), renin activity (PRA), epinephrine (E) and norepinephrine (NE) were:

Time (min)	0	30	60	90	F	P
VP (pg/ml)	0.94 ±0.21	33.7 ±16.6	291 ±131	62.6 ±27.8	11.7	<.001
PRA (ng/ml/h)	3.66 ±0.77	18.9 ± 6.0	21.6 ± 8.0	12.2 ± 3.7	6.8	<.01
E (pg/ml)	19 ±3	342 ±228	277 ±123	41 ±14	3.2	<.04
NE (pg/ml)	155 ±57	468 ±193	467 ±115	144 ±43	3.3	<.04

These experiments demonstrate that the fetus responds promptly to a hypotensive stress by secreting vasoactive mediators. We speculate that vasopressin and renin play an important role in the maintenance of fetal blood pressure, catecholamines playing a secondary role.

EPIDEMIOLOGY

† **508** ASEPTIC MENINGITIS IN JEFFERSON COUNTY, ALABAMA. Michael J. Barrett, Raymond A. Strikas, Martha F. Rogers, Charles Rabkin, W. James Alexander. (Spon. by G.P. Oakley) Centers for Disease Control, Atlanta.

During the period June 1-October 15, 1983, an epidemic of 241 cases of aseptic meningitis occurred among Jefferson county residents (35.9 cases per 100,000 population). The mean age of the patients was 15.1 years and 54% were male. The attack rate in blacks was 1.7 times greater than whites (1.29-2.23; 95% C.L.). Enteroviruses were isolated from 12 of the 21 patients studied, including Coxsackie B5 (9 cases), Coxsackie B2 (1), ECHO 30 (1), and ECHO 16 (1). The unusually high attack rate in children under six months of age (50 cases; 9.3/1,000) is consistent with a recent report suggesting that the incidence in this age group is much greater than previously suspected. A case-control study, which included 44 of the 50 patients under six months of age and 88 controls matched for sex, race, and date of birth, revealed significantly more case families (13/44) with members who had an enterovirus-like illness during the month before hospitalization than control families (7/88) during the same time period (O.R.=4.8; 1.60-14.3, 95% C.L.). No significant differences were found between cases and controls with respect to breastfeeding, prematurity, family size, or recent history of receiving TOPV. The latter finding does not support published suggestions that recent TOPV administration protects against nonpolio enteroviral disease by interference phenomena in the gastrointestinal tract.

509 A POSSIBLE EXPLANATION FOR SOME CASES OF THE "SUDDEN INFANT DEATH SYNDROME (SIDS)". Millard Bass, Richard Kravath, and Leonard Glass. Department of Pediatrics, SUNY, Downstate Medical Center, Brooklyn, New York.

Investigations by a physician trained in forensic pediatrics were made following six consecutive SIDS episodes in order to test the hypothesis that there is a causal relationship between home thermal environment and SIDS. In four cases, autopsy had been performed, and SIDS given as a cause of death on the death certificate. In addition to measurements of temperature at various sites in the area where the infant had died, a detailed history was taken, and the scene of the death recreated. Environmental factors, such as the type of bedcovers, condition of the crib, and source and condition of the heat supply were noted. In each of the cases, asphyxia was the apparent cause of death. These include: 1)noxious gases in a tightly sealed basement apartment with a faulty heater; 2)face down smothering in a spongy pillow that had wrapped around the face; 3)three heavy blankets covering an infant in a body cast who was sleeping in a prone position; 4)three cases of mothers sleeping in the same bed as their infants, in which death was associated with maternal overlay.

This study suggests that a significant number of infant deaths attributed to SIDS may have a definable cause which can be uncovered by careful investigation, and that the incidence figures given for unexplained infant death may be questioned.