229 FATAL PLACENTAL INFARCTION, A PROSPECTIVE STUDY. Richard L. Naeye (Spon. by Nicholas M. Nelson) Pennsylvania State University College of Medicine, M.S. Hershey Medical Center, Department of Pathology, Hershey, Pa.

Placental infarction fatal to the fetus or neonate was analyzed in a prospective study of 59,379 pregnancies. 31,494 well preserved placentas and approximately 60,000,000 pieces of data were analyzed using a log-linear model analysis of contingency tables to determine the significance of the data. 108 fatal cases were attributed to placental infarction when over 25% of the placenta was infarcted and there was no other explanation for death. 96 were stillborn and all 108 had aspirated squames as evidence of antenatal hypoxia. The disorder had a frequency of 2.3/1000 live births. Its frequency was directly correlated with blood pressures in the gravida. This association was augmented by other features of pre-eclampsia and by work during pregnancy. Fatal infarcts were increased 5 fold when abruptio placentae was present and 2 fold when the gravida's hemoglobin was over 12 gm%. The disorder was more often fatal to males than to females and was strongly associated with suboptimal weight gain by the gravida suggesting that gestational undernutrition may have contributed to its genesis. The neonates were growth retarded with organ abnormalities characteristic of undernutrition. The disorder was more frequent in gravida with prior abortions, fetal deaths and preterm deliveries effects enhanced by hypertension. The fatal disorder was 72% more common in gravida who made 0-2 prenatal medical visits than in those who made 5 or more such visits. (Supported by U.S.P.H.S. contract NOl-NS-3-2311).

230 MATERNO-FETAL TRANSFER AND UPTAKE OF 2-DEOXY-GLUCOSE (DG) AND α-AMINOISOBUTYRIC ACID (AIB) IN INTRAUTERINE GROWTH RETARDATION (IUGR) ASSOCIATED WITH RESTRICTED UTERINE BLOOD SUPPLY. M. Nitzan, S. Orloff, and J.D. Schulman, NICHD, NIH, Bethesda, Maryland 20014

The mechanism by which restriction of uterine blood flow causes IUGR is not clearly established. We induced IUGR in rat fetuses by ligation at 18 days gestation of the artery supplying one uterine horn. Two days later, the pregnant rats received intravenously 25 μC H^3-DG (10 C/mM) and 1 μC C^{14}-AIB (9 mC/mM) per 100 gm. After 60 minutes IUGR and control (unligated horn) fetuses and placentas were delivered by C-section, and radioactivity determined (dual channel) in homogenates of placenta, fetus, liver, and brain, and in fetal and maternal plasma. Plasma concentrations and fetal/maternal ratios of both AIB and DG averaged 35% less in IUGR. Uptake of AIB per gram tissue by placenta, whole fetus, brain, and liver was 34, 44, 37 and 22% smaller respectively in IUGR. Uptake of DG per gram tissue by placenta, whole fetus, and brain was reduced 14, 15, and 16% respectively in IUGR. (All differences, $p<.05$). In contrast, liver uptake of DG averaged 17% greater in IUGR ($p=0.2$). The ratio of DG in fetal liver/fetal plasma, and in fetal brain/fetal plasma, was greater in IUGR than control (both $p<.05$). We conclude that in this IUGR model: there is reduction in transfer of DG and AIB into most fetal tissues; reduced growth *in utero* can reasonably be explained by diminished supply of amino acids and glucose; the IUGR fetal liver adapts glucose uptake more effectively than does fetal brain to reduced glucose supply.

231 DEVELOPMENT OF ORGAN BLOOD FLOWS IN THE FETAL AND NEONATAL BABOON. John B. Paton, David E. Fisher and C.W. de Lannoy. Pritzker School of Medicine, University of Chicago, Michael Reese Hospital and Medical Center, Department of Pediatrics, Chicago.

Subhuman primates are frequently used as models of human perinatal physiology and pathology. In order to obtain normal data for organ growth and blood flow 65 measurements were made on baboons from 115 days gestation (term 184 days) to age 8 weeks during acute surgical experiments using the radioactive microsphere method to estimate cardiac output and organ blood flow.

From 115 days to term, while body weight triples, biventricular cardiac output per kg body weight increases by about 50% -- a level that is maintained postnatally from birth to 8 weeks while body weight increases about 50%. Brain weight increases by about 150% during the last third of pregnancy while blood flow increases almost 6-fold. There is a significant decrease in cerebral blood flow in the transition from fetus to neonate but increased blood oxygen content maintains oxygen delivery at the prepartum level. Postnatally there is a 25% increase in brain weight and a comparable increase in blood flow with all of the increase in blood flow distributed to the cerebral cortex. In contrast, renal growth and blood flow over the age-span studied parallel changes in total body growth accounting for about 0.6% of body weight and about 4% of biventricular cardiac output.

232 MATERNAL ACIDOSIS AND FETAL GROWTH RETARDATION. Peggy Rapoport, Yoshio Miyazaki, David Bolam, and Charles L. Paxson Jr., U. of Nebraska Coll. of Med. Omaha, (Spons. by G.C. Rosenquist).

Experience with infants born to mothers suffering from ketoacidosis has suggested that acidosis may be involved in fetal growth retardation (FGR). These studies were designed to determine the effect of acidosis on fetal substrate availability as reflected in altered uterine carbohydrate metabolism. Five pregnant ewes of 90-120 days gestation were chronically prepared by surgical implantation of bilateral electromagnetic uterine artery flow transducers and femoral and uterine vein catheters, and unilateral femoral and cystic artery catheters. Following a 4 day recovery period with optimum maternal nutrition, ammonium chloride solution was continuously infused to produce systemic maternal metabolic acidosis. Whole blood samples for glucose assay and acid-base studies were drawn at hourly intervals for 1-5 hrs in 5 ewes and hourly lactate assay in 1 ewe. Uterine glucose uptake and lactate production were calculated by the Fick equation. A total of 29 infusion studies were completed.

The infusions produced no significant alterations in maternal glucose concentrations or uterine blood flow. Samples drawn after 4 hr. of acidosis demonstrated a significant decrease in maternal pH ($p<.100$), and uterine glucose uptake decreased by 55% ($p<.05$). Lactate production also decreased. We conclude that short term acidosis may decrease fetal substrate availability and that acidosis may play a significant role in the production of fetal growth retardation.

233 THE EFFECT OF SYSTEMIC INFUSIONS OF DEHYDROISOANDROSTERONE (D) ON THE DISTRIBUTION OF UTERINE BLOOD FLOW (UBF) IN THE OVINE PREGNANCY. Charles R. Rosenfeld, Richard J. Worley, Norman F. Gant (Spon. by Chester W. Fink), Southwestern Medical School, Depts. of Ped., Ob-Gyn, Dallas, Texas

Although estrogen is a potent vasodilator of the vascular beds of the ovine uterus, precursors of ovine estrogen remain unknown. Studies in our laboratories have shown increases in UBF and plasma estrone (E1) and estradiol (E2) after systemic infusions of D in the pregnant ewe. To investigate the distribution of UBF after the infusion of 6 mg D, 4 pregnant ewes, 105 to 128 days of gestation, were studied with microspheres. Serial blood samples from 3 ewes showed increases at 15 min in E1 from 27.3 ± 2.23 (mean ± SE) to 117 ± 13.2 pg/ml and in E2 from 34.3 ± 4.91 to 71.7 ± 5.21 pg/ml ($p \leq 0.05$). At 140 min neither UBF nor placental blood flow was significantly altered. Endometrial blood flow increased from 176 ± 24 to 242 ± 32 ml/min (39%, $p < 0.005$) and myometrial blood flow rose from 35 ± 6.1 to 51 ± 8.4 ml/min (45%, $p < 0.01$). Mammary gland blood flow also rose. Of particular interest were the responses in cervical blood flow, rising from 3.18 ± 0.6 to 15.6 ± 2.0 ml/min (441%, $p < 0.005$), and vaginal blood flow from 0.175 ± 0.03 to 0.992 ± 0.15 ml/min·gm (476%, $p < 0.01$). These studies suggest that D may be an important estrogen precursor and the ovine estrogen surge observed at term may prepare the mammary, uterus, cervix and vagina for the process of parturition. Furthermore, the increase in UBF following an infusion of D reflects increased perfusion of non-placental tissues, supporting earlier observations that exogenous estrogen administration might have no beneficial effects for the fetus.

234 COMPUTERIZED TOMOGRAPHIC FINDINGS IN BACTERIAL MENINGITIS. A. David Rothner; Gerald Erenberg; and Meredith Weinstein. (Spons. by William Michener). The Cleveland Clinic. Sections of Child Neurology and Neuroradiology. Cleveland, Ohio.

Despite a decrease in the mortality from bacterial meningitis, a high incidence of residua is still encountered including seizures (sz.), mental retardation (M.R.) and palsies. The C.T. findings in 5 patients with residua of bacterial meningitis are reviewed.

P.C. had pneumococcal meningitis at 10 months. Residua included quadrispasicity, szs, and M.R. C.T. scan revealed ventricular enlargement and no cortical mantle. Arteriography revealed vascular occlusions. C.C. developed H flu meningitis at 11 months. Residua included quadrispasicity and szs. C.T. scan showed cortical atrophy and a right frontal infarct. D.S. had pneumococcal meningitis at 10 months. Residua included M.R. and szs. C.T. scan showed ventricular enlargement. F.K. had H flu meningitis at 11 months. Residua included M.R., a right hemiparesis and szs. C.T. scan showed a left porencephalic cyst. N.N. had H flu meningitis at 2 months. No organism was recovered. Residua included a right hemiparesis. C.T. scan showed subdural effusions.

C.T. scanning can be a useful procedure during the acute illness and during recovery. It is recommended in all patients demonstrating neurologic residua. A prospective study delineating the incidence of C.T. abnormalites in a series of patients with meningitis is presently underway.