249 ANTENATAL STEROIDS AND IMMEDIATE NEONATAL GLUCOSE LEVELS ANDREW ELIMIAN[1], ANTHONY ROYEK[1], J. GERALD QUIRK[1]; [1]State University of New York at Stony Brook, Dept of Obstetrics, Gynecology and Reproductive Medicine, Stony Brook, NY

OBJECTIVE: Antenatal steroids results in temporary elevation in maternal glucose levels. However, it is not known if recent exposure to antenatal steroids results in increased glucose levels in the preterm neonate. The purpose of this study was to compare the glucose levels in preterm neonates recently exposed to antenatal steroids with an unexposed group.

STUDY DESIGN: Consecutive neonates born at 23 to 34 weeks' gestation between 1/1/98 and 4/30/01, who were exposed to one dose of 12 mg betamethasome prior to delivery were compared to a steroid unexposed group for immediate neonatal glucose levels. Student T- and Fisher exact tests were used for statistical analysis.

RESULTS: 94 neonates were exposed to one dose of 12 mg betamethasome prior to delivery while 122 neonates were steroid unexposed. The mean gestational age and birthweight at delivery were significantly lower among the steroid exposed group compared to the unexposed group (28.4 ± 2.9 vs 31.4 ± 4.0 wks, $P = .0001$) and (1172 ± 364 vs 1368 ± 364 g, $P = .0001$) respectively. In spite of these differences in GA and BW, 33% of the steroid exposed neonates had blood glucose level above 80 mg/dL compared with 19.7% of the unexposed group ($P = .028$). 6.4% of the steroid exposed group had blood glucose above 125 mg/dL compared with 1.6% of the unexposed group ($P = .08$).

CONCLUSION: Recent exposure to antenatal steroids results in significant elevation in glucose levels in the preterm neonate.

250 DURATION OF MAGNESIUM SULFATE EXPOSURE AND PERINATAL OUTCOME ANDREW ELIMIAN[1], PAUL OGBURN[1], J. GERALD QUIRK[1], [1]State University of New York at Stony Brook, Dept of Obstetrics, Gynecology and Reproductive Medicine, Stony Brook, NY

OBJECTIVE: To determine the relationship, if any, between duration of magnesium sulfate exposure as a tocolytic agent and perinatal morbidity and mortality.

STUDY DESIGN: We studied neonates at our institution who were born between 23 and 34 weeks' gestation following preterm labor with intact membranes and preterm premature rupture of membranes from 1/1/98 through 4/30/2001. The population was stratified by exposure to tocolytic Magnesium (Mg) sulfate for 24 hours or less versus over 24 hours and compared by various perinatal outcome variables. Student t test, χ, Fisher exact test, and logistic regression were used for analysis.

RESULTS: 79 neonates were exposed to Mg for over 24 h while 110 neonates were exposed for 24 h or less. There were no significant differences between groups with regards to GA (wks) (27.9 ± 3.1 vs 28.4 ± 2.8, $P = .11$), BW (g) (1122 ± 379 vs 1202 ± 372, $P = .75$), PTL (60 [76%] vs 81 [74%], $P = .74$), PROM (19 [24%] vs 29 [26%], $P = .74$). In addition, the two groups did not differ significantly with regards to exposure to steroids, antibiotics and rate of histologic chorioamnionitis. Furthermore the rates of RDS (71% vs 73%, $P = .91$), Apgar <7 (12.7% vs 19%, $P = .32$), surfactant use (53% vs 46%, $P = .31$), neonatal sepsis (23% vs 23%, $P = 1.0$), PDA (24% vs 21%, $P = .72$), NEC (2.5% vs 4.5%, $P = .70$), IVH/PVL (14% vs 15%, $P = .93$) and neonatal death 10.5% vs 9.3%, $P = .81$) did not differ significantly between the two groups. However, the incidence of clinical chorioamnionitis was significantly higher in the group exposed to magnesium for over 24 h compared to those exposed for 24 h or less (22% vs 8.2%, $P = .005$).

CONCLUSION: Perinatal outcomes are largely similar in women exposed to tocolytic magnesium sulfate for over 24 hours compared to those receiving magnesium for 24 hours or less. The exception was increased rate of clinical chorioamnionitis in those receiving magnesium for over 24 hours.

251 EARLY FETAL EXPOSURE TO LONG TERM INDOMETHACIN THERAPY TO PREVENT PRETERM DELIVERY: NEONATAL OUTCOME MICHAEL V. MUENCH[1], CHRISTOPHER R. HARMAN[1], AHMET A. BASCHAT[1], LINDSEY S. ALGER[1], CARL P. WEINER[1], [1]University of Maryland at Baltimore, Obstetrics, Gynecology and Reproductive Sciences, Baltimore, MD

OBJECTIVE: To answer the question: Is long-term, early onset, fetal exposure to indomethacin safe in the prevention of preterm delivery?

STUDY DESIGN: Case control study of all fetuses exposed to long-term (≥2 weeks) indomethacin therapy (LTIT) for cervical shortening and/or dilatation. Entry criteria: Singleton or twin pregnancy, indomethacin started <24 weeks gestation by ultrasound-confirmed dates, maintained at 25 mg PO QID or greater for at least 2 weeks. The treatment group was compared with randomly selected controls (no indomethacin, not delivered for maternal reasons) and matched (by gestational age within 1 week) from our concurrent neonatal database. Outcomes were abstracted from the medical record. The LTIT protocol included 100 mg po load, 50 mg po for 24-48 hours, maintenance 25 mg po qid. All fetuses were monitored serially for cardiac strain, abnormalities of ductus arteriosus flow, biometry and oligohydraminos until LTIT was stopped. By protocol no patient had LTIT >32 weeks.

RESULTS: There were 27 cases and 60 matched controls. Mean gestational age at onset of therapy was 22.0 weeks. Duration of treatment was 6.4 weeks (range 2-14). Fetal ductal constriction with right heart strain and oligohydraminos (maximum vertical pocket <2 cm) occurred in 2 fetuses >27 weeks. In both, all fetal effects resolved after with 50% dose reduction. There was no significant difference in birth weight, 5-minute Apgar, days intubated, IVH, NEC, PVL, neonatal sepsis, late neonatal oxygen requirement, days in the neonatal ICU, neonatal death, endomyometritis, or chorioamnionitis. Patent ductus arteriosus was significantly more common in the control group (chi square $P < .001$), attributed only to indomethacin use, by logistic regression.

CONCLUSION: Early gestational exposure to long-term indomethacin therapy does not place the fetus at risk when monitored appropriately. The reduction in the incidence of PDA after birth may be related to fetal indomethacin exposure.

252 THE EFFECT OF IN UTERO EXPOSURE TO INDOMETHACIN ON THE NEED FOR SURGICAL CLOSURE OF A PATENT DUCTUS ARTERIOSUS IN THE NEWBORN VR SUAREZ[1], LL THOMPSON[2], V JAIN[3], G OLSON[1], M BELFORT[3], GDV HANKINS[4], GR SAADE[1]; [1]University of Texas Medical Branch at Galveston, Obstetrics and Gynecology, Galveston, TX; [2]University of Texas Medical Branch at Galveston, Pediatrics/Neonatology, Galveston, TX; [3]Utah Valley Regional Medical Center, Provo, UT; [4]University of Texas Medical Branch, Obstetrics and Gynecology, Galveston, TX

OBJECTIVE: Animal studies show that fetal exposure to indomethacin leads to structural changes in the ductus arteriosus (DA) that may prevent its postnatal closure. Our objective was to determine the effect of in utero exposure to indomethacin on postnatal need for surgical closure of a patent DA.

STUDY DESIGN: Neonatal intensive care and maternal logs were used to identify infants who were diagnosed with patent DA and delivered at <32 weeks gestation. Perinatal and neonatal variables were compared between newborns who required surgical closure of the patent DA after failed medical management (cases) and those who did not (controls). Statistical analysis was performed using Student's t, Mann-Whitney, Chi-square and Multiple Logistic Regression tests as appropriate.

RESULTS: Seventy-seven neonates were included, 8 of which failed medical therapy and required surgical intervention. There were no significant differences between cases and controls for maternal demographics, gestational age, birth weight, and route of delivery. In utero exposure to indomethacin was more common in neonates requiring surgery versus those that did not (62.5% vs 21.7%; odds ratio = 4.75; 95% CI [1.3 - 18.1], $P = .02$). The association remained significant, even after controlling for confounders (odds ratio = 3.30; 95% CI [1.3 - 8.1], $P = .01$). In utero exposure to indomethacin for more than 72 hours was more common in neonates requiring surgery versus those that did not (50.0% vs 8.7%; odds ratio = 6.7; 95% CI [2.0 - 22.6], $P = .008$).

CONCLUSION: Failure of medical management and need for closure of patent DA does appear to be increased by in utero indomethacin exposure. Additional studies are needed to determine if indomethacin dose or length of exposure modify this risk. These findings should be considered into the overall context of the risks versus benefits of tocolysis.