257 **APPLICATION OF ELECTRICAL INHIBITORY PULSES (EI) TO THE *UTERUS OF TERM DELIVERING* RATS IN VIVO PROMOTES QUIESCENCE AND INCREASES DELIVERY TIMES AND BIRTH INTERVALS IN THE IN VIVO RAT UTERUS DELIVERING AT TERM** JEFFREY KARSDON[1], SHAO-QING SHI[2], WILLIAM MANER[2], GEORGE SAADE[2], ROBERT GARFIELD[2]; [1]Unaffiliated, Baldwin Harbor, NY; [2]University of Texas Medical Branch, Obstetrics and Gynecology, Galveston, TX

**OBJECTIVE:** Parturition depends on uterine contractions which are mechanical events resulting from underlying myometrial electrical activity. Previous studies showed EI, an electrical current injected into the myometrium, inhibits the contracting uterus during pregnancy. Our aim was to determine if EI could inhibit parturition as determined by a decrease in contractility and a suppression of delivery of fetuses in vivo in the rat.

**STUDY DESIGN:** Timed-pregnant Sprague-Dawley rats on gestational day 21 or 22 were used for all studies. An array of 5 stainless steel electrodes were sutured along one of the bicornuate uterine horns of study rats and intrauterine pressure (IUP) and uterine electromyographic (EMG) activity were measured with an intraperitoneal radiotelemetric transmitter. An on-line computer with a data acquisition system collected the area under the contraction curve (AUC). After delivery of the first pup, EI was initiated via the array of electrodes with a constant electrical current of square wave pulses at 1-5 mA, 28 ms/pulse, 30 Hz and was maintained intermittently using a feedback loop. The delivery times (time interval between pups delivered) were analyzed (mean ± SE) for 4 control (n = 25) and 3 study rats (n = 14). Delivery times were also recorded in noninstrumented and non-EI treated rats.

**RESULTS:** EI significantly decreased IUP and EMG activity, and increased delivery times of subsequent pups from the study horn only. Twelve hours after delivery of the first pup, the average number of pups delivered were 10.25 and 5.00, and their delivery times were 8.96 ± 4.48 and 22.29 ± 31.81 minutes for control and EI respectively ($P>.05$).

**CONCLUSION:** EI is known to inhibit IUP and uterine EMG activity in vivo, and now is shown to increase delivery times in rats. This effect is localized i.e. not systemic, and may find clinical use in for prevention of preterm birth.

258 **IMPAIRED CLEARANCE OF SERUM MAGNESIUM IN THE VERY LOW BIRTH WEIGHT (VLBW, <1500 G) NEONATE AS COMPARED TO ITS MOTHER** ROBERT MITTENDORF[1], PETER PRYDE[2], KWANG-SUN LEE[3], RICHARD BESINGER[1], WILLIAM MACMILLAN[1], ROBERTA KARLMAN[1], PATRICIA HEYWOOD[1], SUSAN JANECZEK[1], JOHN GIANOPOULOS[1]; [1]Loyola University (Chicago), Obstetrics and Gynecology, Maywood, IL; [2]University of Wisconsin, Madison, Obstetrics and Gynecology, Madison, WI; [3]University of Chicago, Pediatrics, Chicago, IL

**OBJECTIVE:** The goal of this study is to determine whether the magnesium clearance rate of the VLBW neonate during the first day of life is decreased or impaired as compared to its mother.

**STUDY DESIGN:** Analyzing retrospective data collected at the University of Chicago Children's Hospital since 1994, we identified neonates (n = 49) who had elevated serum total magnesium (TMg) levels (>2.5 mg/dL) on the day of delivery, and on whom serum TMg levels were available for the next day of life. Comparing these two levels, we were able to compute the mean clearance rate of serum TMg during the first day of neonatal life.

**RESULTS:** Among these 49 children, the mean serum TMg level was 4.4 mg/dL on the day of delivery, and the mean clearance rate for serum TMg during the first day of life was 0.79 mg/dL. For VLBW neonates (n = 19), the mean serum TMg clearance rate during the first day of life was 0.29 mg/dL. When comparing these empiric determinations with our previously published research (Pediatr Res 49(4), 407A, 2001) on the maternal clearance rate of serum TMg (1.04 mg/dL per day [.012 mmol/L per hour, ionized magnesium]) among a group of women who had been exposed to tocolytic magnesium sulfate within 100 hours of delivery, we found that compared to their mothers, VLBW neonates had a significantly decreased daily clearance rate of serum TMg (one sample Student t test, $P<.001$).

**CONCLUSION:** The finding of impaired magnesium clearance in VLBW neonates may be relevant to the search for mechanisms to explain the recent observations of the Neonatal Network (NICHD) demonstrating a significant association between tocolytic magnesium sulfate exposure and both (1) periventricular leucomalacia, and (2) cerebral palsy.

259 **MECHANISMS OF MORTALITY IN THE MAGNESIUM AND NEUROLOGIC ENDPOINTS TRIAL (MAGNET TRIAL): FETAL INFLAMMATORY RESPONSE SYNDROME (FIRS)** ROBERT MITTENDORF[1], SUSAN JANECZEK[2], WILLIAM MACMILLAN[2], JOHN GIANOPOULOS[2], RICHARD BESINGER[2], ROBERTA KARLMAN[2], PATRICIA HEYWOOD[2], KWANG-SUN LEE[3], PETER PRYDE[4]; [1]Loyola University (Chicago), Obstetrics and Gynecology, Maywood, IL; [2]Loyola University (Chicago), Obstetrics and Gynecology, Maywood, IL; [3]University of Chicago, Pediatrics, Chicago, IL; [4]University of Wisconsin, Madison, Obstetrics and Gynecology, Madison, WI

**OBJECTIVE:** To determine whether the co-morbid presence of FIRS could account for some of the excess mortality among children who were exposed to magnesium sulfate (MgSO4) in the MagNET Trial.

**STUDY DESIGN:** In a clinical trial, mothers in preterm labor were randomized to tocolytic or preventive MgSO4, other tocolytic, or saline control. At delivery, we obtained umbilical cord blood to determine plasma interleukin-6 (IL-6) levels using the standard commercial enzyme-linked immunoassay (Endogen, Cambridge, Massachusetts). Placentas and umbilical cords underwent primary and secondary histopathologic examination for chorioamnionitis and funisitis. Using these empiric data, we defined FIRS as being present at delivery if: (a) plasma IL-6 levels exceeded 10 pg/ml, or (b) funisitis was present, or (c) both findings were present.

**RESULTS:** In our study, 82 fetuses were exposed in utero to MgSO4. Elevated IL-6 levels were found in 34% of 58 evaluable cord specimens, and funisitis was diagnosed in 15% of 73 available placentas. FIRS data are available for 8 of 9 children who died following randomization and exposure to MgSO4. Of the children who died, 13% (1/8) had FIRS, as defined; whereas 28% (21/74) of MgSO4 survivors had FIRS (Fisher exact test, $P = .68$). Although the difference is not significant, in these data, FIRS is less prevalent, not more prevalent, among the children who died. In a multivariable logistic regression, including the FIRS variable, very low birth weight (<1500 g), use of MgSO4 as a tocolytic or preventive, and fetal plurality (twins), there is no statistical evidence to suggest that these findings are confounded.

**CONCLUSION:** Our data do not support the hypothesis that co-morbidity with FIRS may account for the previously reported excess total pediatric mortality associated with tocolytic magnesium sulfate.

260 **MATERNAL AND FETAL PLATELET FUNCTION AFTER MATERNAL EXPOSURE TO MAGNESIUM SULFATE** ELEANOR RHEE MD[1], ANDRA JAMES MD[1], CHARLES WALKER BS[2], THOMAS ORTEL MD[2]; [1]Duke University, Obstetrics and Gynecology, Durham, NC; [2]Duke University, Department of Medicine, Durham, NC

**OBJECTIVE:** Magnesium has been shown to have antithrombotic properties. Few studies, however, have evaluated platelet function in magnesium-exposed pregnant women and no studies have evaluated platelet function in exposed neonates. The purpose of this study was to evaluate platelet function in mothers and neonates exposed to magnesium.

**STUDY DESIGN:** Magnesium-exposed mothers and controls were identified while in labor. After delivery, two 5-ml 3.2% sodium citrate tubes were collected by venipuncture from each mother and the umbilical cord of each neonate. Specimens were processed with two different agonists, collagen and ADP, using a whole blood impedance aggregometer (Chrono-Log Corp., *Havertown*, PA). *Impedance*, which is directly proportional to the amount of aggregation, was measured in ohms. Median values were obtained from exposed mothers and neonates and compared with controls. Statistical analysis was performed using the Mann Whitney U Test. (SAS, Carey, NC).

**RESULTS:** Specimens were successfully processed on 11 mothers and 10 neonates exposed to magnesium and 12 control mothers and neonates. The mean gestational age was 37 weeks (range: 33-41) for cases and 39 weeks (range: 36-42) for controls. In all cases, the indication for magnesium was preeclampsia. Although platelet aggregation was not significantly different between magnesium-exposed mothers and controls, in neonates, ADP-induced aggregation was markedly impaired in the magnesium-exposed group. See Table 1 and Table 2 below.

**CONCLUSION:** In-utero magnesium exposure is associated with impaired ADP-induced platelet aggregation in neonates. This may have implications for neonatal hemostasis.

**Table 1**

**Median platelet aggregation measurements in ohms for mothers**

| | MAGNESIUM-EXPOSED (N = 11) | CONTROLS (N = 12) | *P* VALUE |
|---|---|---|---|
| ADP | 8.0 | 6.3 | .88 |
| Collagen | 29.0 | 25.8 | .07 |

**Table 2**

**Median platelet aggregation measurements in ohms for neonates**

| | MAGNESIUM (N = 10) | CONTROLS (N = 12) | *P* VALUE |
|---|---|---|---|
| ADP | 0 | 3.0 | <.01 |
| Collagen | 21.8 | 20.0 | .29 |