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Magnesium Sulfate Decreases Maternal Blood Pressure but Not Uterine Blood Flow during Epidural Anesthesia in Gravid Ewes

Robert D. Vincent, Jr., M.D., David H. Chestnut, M.D.,† Susan L. Sipes, M.D.,‡ Carl P. Weiner, M.D.,§ Craig S. DeBruyn,¶ Shari A. Bleuer, B.S.¶*

The purpose of this study was to determine whether administration of magnesium sulfate decreased maternal blood pressure during epidural anesthesia in gravid ewes. Twenty-two experiments were performed in 11 chronically instrumented animals between 0.8 and 0.9 of timed gestation. The experimental sequence included: 1) T = 0: magnesium sulfate 4 g intravenously over 5 min followed by an infusion of magnesium sulfate at 4 g/h, *or* normal saline iv followed by an infusion of normal saline alone; 2) T = 135 min: 500 ml normal saline intravenously over 12 min; and 3) T = 150 min: epidural administration of 2% lidocaine. The initial bolus of magnesium sulfate slightly decreased maternal mean arterial pressure (MAP) but increased uterine artery blood flow (UBF). The increase in UBF was accompanied by an increase in fetal Pa_{O_2} at 145 min in the magnesium sulfate group but not in the control group. At 165 min (*i.e.*, 15 min after the epidural injection of lidocaine), epidural lidocaine resulted in a median sensory level of T-10 in the magnesium sulfate group and T-11 in the control group. During epidural anesthesia, maternal MAP was lower ($P = 0.001$) in the magnesium sulfate group than in the control group. At 165 min, maternal MAP was 18 ± 3% below baseline ($P = 0.0001$) in the magnesium sulfate group but did not differ significantly from baseline in the control group. Maternal cardiac output and UBF did not differ from baseline after epidural injection of lidocaine in either group. Over time, maternal and fetal Pa_{CO_2} were greater, and fetal arterial *p*H was lower, in the magnesium sulfate group than in the control group. Fetal Pa_{O_2} and base excess were not significantly below baseline at any time in either group. We conclude that infusion of magnesium sulfate slightly decreased maternal blood pressure during epidural lidocaine anesthesia in gravid ewes. However, magnesium sulfate did not decrease UBF or fetal Pa_{O_2} during epidural lidocaine anesthesia. (Key words: Anesthesia: obstetric. Anesthetic techniques: epidural. Anesthetics, local: lidocaine. Ions: magnesium. Pharmacology, tocolytic agents: magnesium sulfate. Pregnancy: preterm labor.)

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PARTURIENTS RECEIVING MAGNESIUM SULFATE frequently become candidates for epidural anesthesia. Examples include preterm patients with failed tocolysis and patients with preeclampsia. Magnesium decreases baseline tension in vascular smooth muscle[1] and attenuates the vasoconstrictor response to vasopressor agents *in vivo*[2,3] and *in vitro*.[1,4] Some have suggested that magnesium sulfate should be discontinued before induction of epidural anesthesia to decrease the risk of maternal hypotension.[5] But other anesthesiologists successfully give epidural anesthesia to preeclamptic women who are receiving magnesium sulfate for seizure prophylaxis.

Chestnut *et al.*[6] noted that magnesium sulfate, but not ritodrine, worsened maternal hypotension during hemorrhage in pregnant sheep. We speculated that administration of magnesium sulfate might also exaggerate hypotension during epidural anesthesia. The purpose of this study was to determine whether magnesium sulfate worsens maternal hypotension during epidural anesthesia in gravid ewes.

Materials and Methods

The protocol was approved by the University of Iowa Animal Care Committee. Mixed-breed ewes were obtained from a commercial breeder at approximately 118 days of timed gestation (term = 145 days). Each animal was fasted for 36 h before surgery. At 120 days of gestation, induction of general anesthesia was accomplished with sodium thiopental (8–12 mg/kg). After tracheal intubation, anesthesia was maintained with 50% nitrous oxide, 50% oxygen, and 1–1.5% halothane. Mechanical ventilation was maintained throughout surgery. With sterile technique, a laparotomy and hysterotomy were performed, and catheters (polyethylene-90) were inserted into the fetal descending aorta *via* each femoral artery.

* Research Fellow, Department of Anesthesia.

† Associate Professor, Departments of Anesthesia and Obstetrics and Gynecology.

‡ Fellow, Department of Obstetrics and Gynecology.

§ Professor, Department of Obstetrics and Gynecology.

¶ Research Assistant, Department of Anesthesia.

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Address correspondence to Dr. Vincent: Department of Anesthesia, The University of Iowa, Iowa City, Iowa 52242. Reprints will not be available.

Fenestrated high pressure tubing (MX 566, Medex, Hilliard, OH) was secured to the fetal hind limb to monitor intraamniotic pressure. After the hysterotomy and laparotomy incisions were closed, a left paramedian incision was made. The left uterine artery was isolated *via* a retroperitoneal approach, and an electromagnetic flow probe (Dienco, Los Angeles, CA) was placed around the artery. Catheters (polyethylene-240) were then inserted into the maternal descending aorta and inferior vena cava *via* the left mammary artery and vein, respectively. All catheters were tunneled subcutaneously and exteriorized through a small incision in the left flank. A single-orifice, 19-G epidural catheter (Portex, Wilmington, MA) was inserted percutaneously 5 cm into the epidural space at the lumbosacral junction, and the catheter was secured to the back. Finally, an 8.5-Fr introducer (AK-09800, Arrow, Reading, PA) was placed percutaneously into the right jugular vein. Eight milliliters 2% lidocaine were injected through the epidural catheter before the completion of surgery. After surgery the sensory level of anesthesia was determined.

After surgery each animal was kept in an approved cage in a restricted area, fed a balanced diet, and allowed a recovery period of at least 3 days. Procaine penicillin G 500,000 units and dihydrostreptomycin 625 mg (Distrycillin®, Solvay, Princeton, NJ) were given to the mother intramuscularly the day of surgery and daily for 3 days after surgery. Gentamicin 80 mg was given to the mother intravenously during surgery and on the day of each experiment. Also, gentamicin 40 mg was given *via* the amniotic catheter during surgery and on the day of each experiment.

Each experiment was performed with the animal standing supported by a canvas sling, within an approved transport cart. The canvas sling allowed the animals to remain upright despite the occurrence of hind limb weakness during epidural anesthesia. (Earlier we had performed a pilot study in which the animals were restrained in the lateral decubitus, but the animals developed agitation and tachycardia.)

Before the first experiment in each animal, a pulmonary artery catheter (93A-131H-7F or 93A-831H-7.5F, American Edwards, Santa Ana, CA) was inserted through the jugular vein introducer. Maternal arterial blood pressure, central venous pressure, pulmonary artery pressure, and fetal arterial blood pressure were measured continuously with disposable strain gauge pressure transducers (46951-02, Abbott Critical Care Systems, North Chicago, IL) *via* Coulbourn amplifiers (S72-25, Coulbourn Instruments, Lehigh Valley, PA). Fetal pressures were corrected by subtraction of simultaneous intraamniotic pressure. Mean arterial blood pressure (MAP) was computed arithmetically. The maternal heart rate (HR) and fetal HR were computed from the arterial waveforms. Uterine artery blood flow (UBF) was measured continuously with a quantitative electromagnetic flowmeter (RF-2500, Dienco, Los Angeles, CA). Arterial and venous pressures, HR, and UBF were recorded at 10-s intervals with a computer-based system and customized software (Alternatives Unlimited, Des Moines, IA).

Cardiac output measurements were made in triplicate with 10 ml iced saline and a thermodilution cardiac output computer (9520A, Edwards Laboratories, Santa Ana, CA). Maternal and fetal Pa_{O_2} and Pa_{CO_2} and *p*H values were determined with an Instrumentation Laboratory (1302, Leighton, MA) blood gas analyzer. All values were corrected for temperature (39.5° C).

The experimental sequence was as follows:

1. Forty minutes were taken for the animal to acclimate to the laboratory environment.
2. Twenty minutes were used to take baseline measurements.
3. At T = 0 each animal received magnesium sulfate 4 g intravenously, or normal saline over 5 min, followed by an infusion of magnesium sulfate (4 g/h) or normal saline alone. The total volume of normal saline was 100 ml/h in each experiment.
4. At 135 min each animal received 500 ml normal saline intravenously over 12 min.
5. At 150 min each animal received 8–12 ml 2% lidocaine *via* the epidural catheter. The goal was to produce a T-10 sensory level. The dose was based on the sensory level obtained at the time of surgery. Within an individual animal, the dose was identical for the two experiments.
6. Beginning at 165 min (*i.e.*, 15 min after the epidural injection of lidocaine) and continuing every 5 min through 180 min, the sensory level of anesthesia was determined with a curved hemostat. (We did not pinch the skin of the sheep. Rather, we used the hemostat in a manner similar to the way one would use a needle to assess the sensory level.[7])
7. At 210 min the infusion of magnesium sulfate or normal saline was discontinued.
8. Hemodynamic measurements were continued through 240 min.

Maternal serum magnesium concentrations were determined at baseline, 145 min, and 210 min, using a bichromatic colorimetric method (Lancer®, Rapid Stat, St. Louis, MO).

Twenty-two experiments were performed in 11 animals. Both experiments were performed in each animal in random order, but only one experiment was done each day.

Statistical analysis was by repeated measures analysis of variance. This analysis included *t* tests for comparison of individual measurements. Bonferroni adjustment was

performed as appropriate. $P < 0.05$ was considered significant. Data are given as mean ± SEM.

Results

The mean (± SEM) weight of the animals was 61.5 ± 1.8 kg. The two groups were similar with regard to baseline maternal and fetal hemodynamic, blood gas, and acid–base measurements (table 1).

Magnesium sulfate increased the maternal serum magnesium concentration to 5.5 ± 0.2 mg/dl at 145 min and 6.1 ± 0.4 mg/dl at 210 min (table 2). (These concentrations are within the therapeutic range for tocolysis and seizure prophylaxis in pregnant women.[8,9])

At 165 min, epidural injection of lidocaine (9.6 ± 0.4 ml) resulted in a median sensory level of T-10 in the magnesium sulfate group and T-11 in the control group.

Across all times maternal HR was slightly higher ($P = 0.01$) in the magnesium sulfate group than in the control group (data not shown). However, maternal HR did not significantly differ from baseline at any time in either group.

Magnesium sulfate decreased maternal MAP 7 ± 2% below baseline ($P = 0.0001$) at 5 min (fig. 1). At 150 min, just after the bolus infusion of normal saline, maternal MAP did not differ from baseline in the magnesium sulfate group but was increased 9 ± 1% above baseline ($P = 0.0001$) in the control group. After epidural lidocaine, maternal MAP measurements were lower ($P = 0.0001$) in the magnesium sulfate group than in the control group. At 165 min (*i.e.*, 15 min after the epidural injection of lidocaine) maternal MAP was 18 ± 3% below baseline ($P = 0.0001$) in the magnesium sulfate group but was only 5 ± 4% below baseline (P = nonsignificant [NS]) in the control group. At 240 min (*i.e.*, 30 min after the infusion of magnesium sulfate was discontinued), maternal MAP was 4 ± 4% below baseline (P = NS) in the magnesium sulfate group and 3 ± 3% below baseline (P = NS) in the control group (data not shown).

Maternal systemic vascular resistance (SVR) was below baseline ($P < 0.003$) at 150, 165, and 180 min in the magnesium sulfate group (fig. 1). In contrast, SVR did

TABLE 1. Baseline Maternal and Fetal Hemodynamic, Blood Gas, and Acid–Base Measurements

	$MgSO_4$ (n = 11)	NS (n = 11)
Maternal		
Heart rate (beats per min)	116 ± 5	109 ± 2
Mean arterial pressure (mmHg)	94 ± 2	95 ± 2
Cardiac output (l/min)	9.9 ± 0.4	10.0 ± 0.3
Uterine blood flow (ml/min)	555 ± 98	652 ± 124
Pulmonary capillary wedge pressure (mmHg)	10.6 ± 1.3	9.5 ± 1.3
Central venous pressure (mmHg)	7.0 ± 1.0	5.7 ± 1.4
*p*H	7.45 ± 0.01	7.44 ± 0.02
P_{O_2} (mmHg)	106 ± 4	106 ± 2
P_{CO_2} (mmHg)	37 ± 1	36 ± 1
Fetal		
Heart rate (beats per min)	166 ± 4	163 ± 5
Mean arterial pressure (mmHg)	45 ± 2	45 ± 2
*p*H	7.35 ± 0.01	7.34 ± 0.01
Base excess (mEq/L)	+2.2 ± 0.6	+2.0 ± 0.7
P_{O_2} (mmHg)	21 ± 2	20 ± 1
P_{CO_2} (mmHg)	49 ± 2	50 ± 1

All values are expressed as mean ± SEM.
There were no significant differences between the two groups.

TABLE 2. Maternal Arterial Serum Magnesium Concentrations

	$MgSO_4$ (n = 11)	NS (n = 11)
Baseline	1.8 ± 0.1	1.9 ± 0.1
145 min	5.5 ± 0.2	1.8 ± 0.1
210 min	6.1 ± 0.4	1.9 ± 0.1

All values are expressed as mean ± SEM. Concentrations are mg/dl.

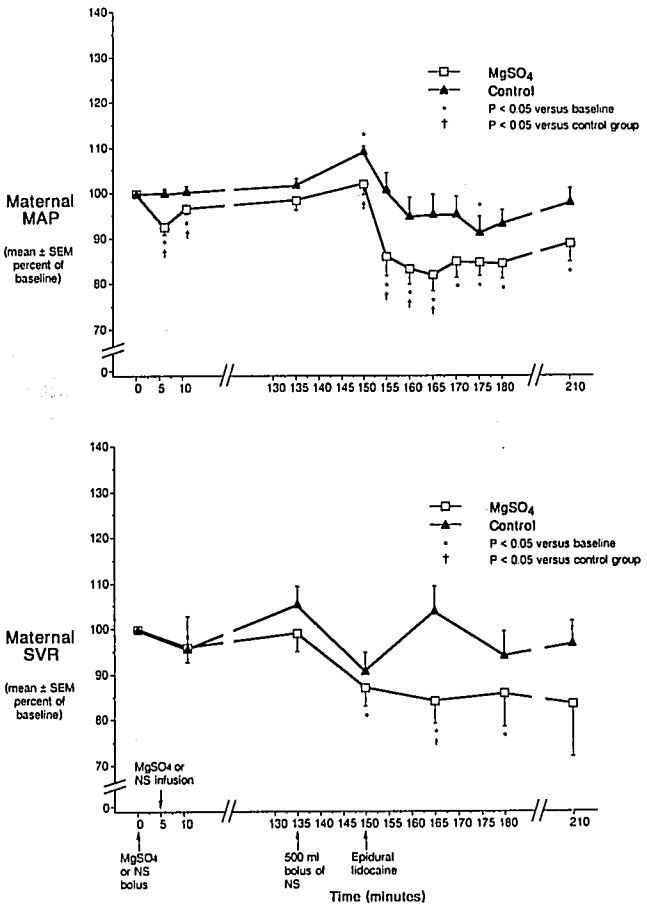

FIG. 1. Maternal mean arterial pressure (MAP) and systemic vascular resistance (SVR) responses over time.

not significantly differ from baseline at any time in the control group.

The bolus infusion of normal saline increased maternal cardiac output at 150 min in both groups ($P \leq 0.0004$; fig. 2). However, after epidural lidocaine, maternal cardiac output was similar to baseline at each measurement in both groups.

At 10 min, UBF was increased 12 ± 3% above baseline ($P = 0.0002$) in the magnesium sulfate group (fig. 2). At 150 min, UBF was 32 ± 6% above baseline ($P = 0.0001$) in the magnesium sulfate group, but did not differ from baseline in the control group. UBF was similar to baseline at all measurements after epidural lidocaine in both groups.

Fetal HR and fetal MAP did not differ significantly from baseline at any time in either group (data not shown).

Across time, maternal Pa_{CO_2} was slightly higher ($P = 0.0004$) in the magnesium sulfate group than in the control group (fig. 3). Also, fetal Pa_{CO_2} was slightly higher ($P = 0.0001$) in the magnesium sulfate group than in the control group over time (fig. 3).

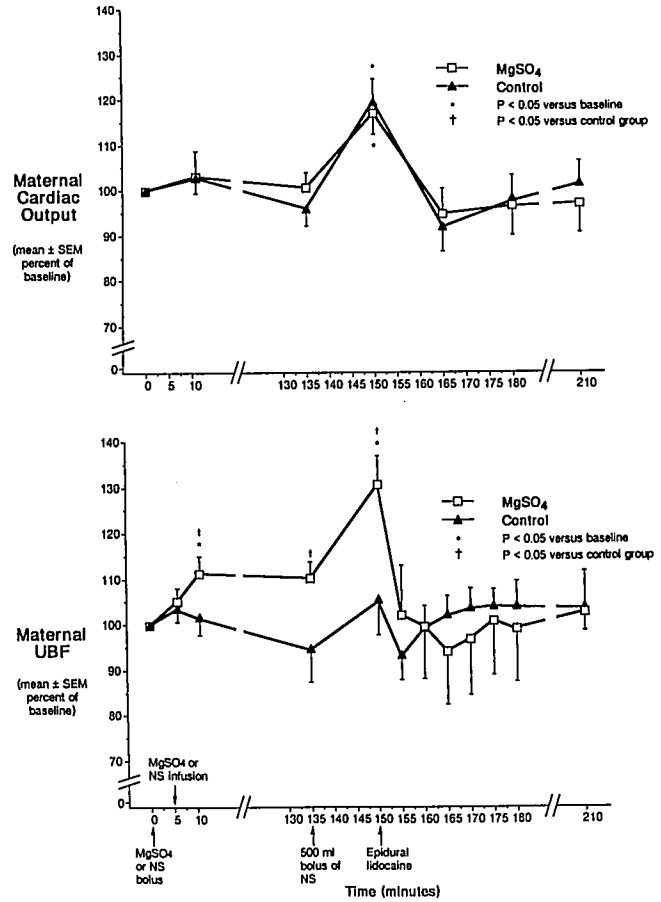

FIG. 2. Maternal cardiac output (CO) and uterine blood flow (UBF) responses over time.

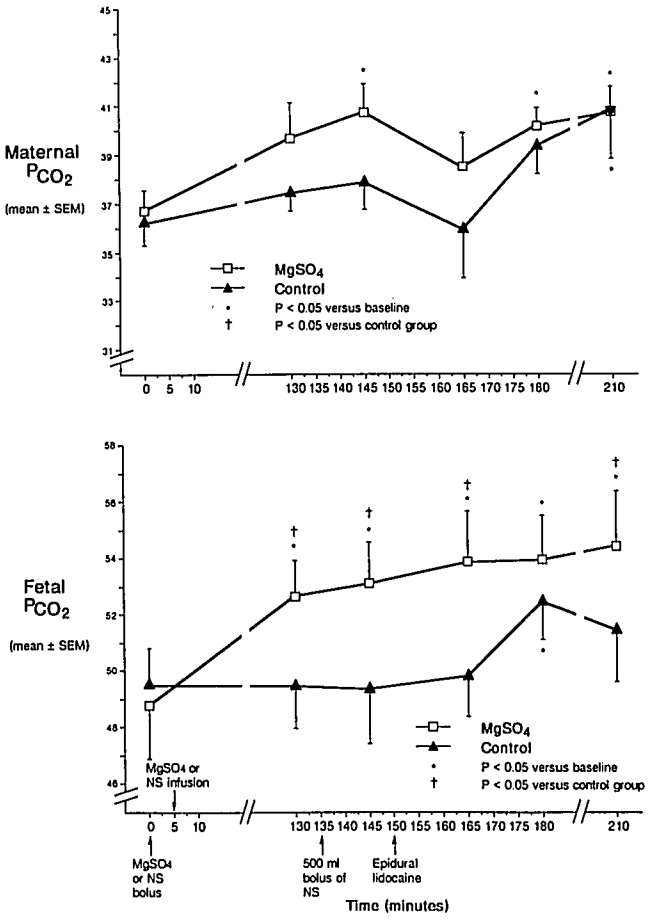

FIG. 3. Maternal and fetal arterial blood P_{CO_2} measurements over time.

Fetal arterial *p*H was slightly lower ($P = 0.0003$) in the magnesium sulfate group than in the control group over time (fig. 4). Epidural lidocaine did not significantly change fetal arterial *p*H in either group when compared with fetal arterial *p*H measurements just before epidural lidocaine.

Fetal Pa_{O_2} was above baseline ($P = 0.007$) at 145 min in the magnesium sulfate group (fig. 4) but not in the control group. Fetal Pa_{O_2} did not significantly differ from baseline after epidural lidocaine in either group. Fetal arterial base excess did not differ from baseline at any time in either group (data not shown).

Discussion

Bolus administration of magnesium sulfate typically causes a small decrease in arterial pressure in laboratory animals and in preeclamptic patients.[10–13] Typically the decrease in arterial pressure is not maintained with continuous infusion of magnesium sulfate.[14] Similarly, in the current study, magnesium sulfate produced a small, transient decrease in maternal MAP just after bolus injection.

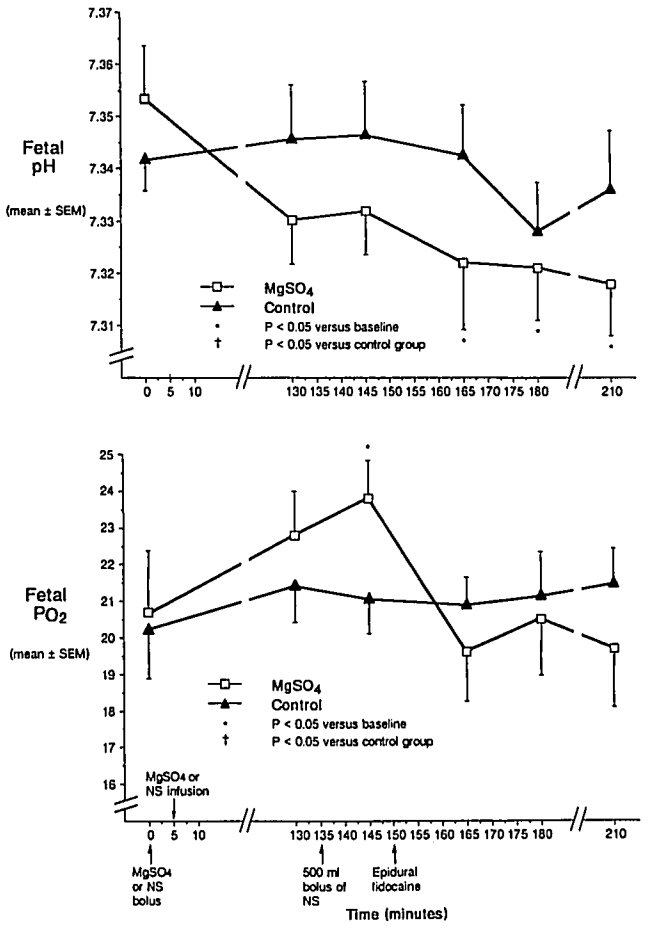

FIG. 4. Fetal arterial blood *p*H and P_{O_2} measurements over time.

Magnesium antagonizes the activity of calcium in vascular smooth muscle.[1,15] Magnesium also decreases the release of norepinephrine from the adrenal medulla[16,17] and increases the uptake of norepinephrine in adrenergic nerve terminals.[18] In addition, magnesium sulfate attenuates the vasoconstrictor response to epinephrine, norepinephrine, and angiotensin II *in vivo*[2,3] and *in vitro*.[1,4] Chestnut *et al.*[6] noted that magnesium sulfate, but not ritodrine, worsened maternal hypotension during hemorrhage in pregnant sheep. Thus, we hypothesized that magnesium sulfate might interfere with compensatory adaptations that occur during epidural anesthesia.

During epidural anesthesia, vasoconstriction occurs in the areas of the body with intact sympathetic innervation. This reflex vasoconstriction helps minimize the cardiovascular changes resulting from vasodilation below the level of anesthesia. Baron *et al.*[19] demonstrated that a T-11 sensory level from epidural anesthesia increased forearm vascular resistance without altering mean arterial pressure (MAP) or HR in human patients. Likewise, using ^{99m}Tc-labeled erythrocytes, Arndt *et al.*[20] demonstrated that T-4 epidural anesthesia caused vasoconstriction in the thorax, upper limbs, and the splanchnic regions. Low-pressure baroreceptors located in the pulmonary vessels, right atrium, and left atrium, and carotid baroreceptors mediate this compensatory increase in vascular tone above the level of sympathetic denervation.[21,22] In the current study, maternal MAP was lower during epidural lidocaine anesthesia in the magnesium sulfate group than in the control group. This suggests that hypermagnesemia attenuates the increase in vascular tone that occurs above the level of anesthesia in response to vasodilation below the level of anesthesia.

This study confirms earlier observations[23] that in the absence of hypotension, epidural anesthesia does not decrease UBF in gravid ewes. However, Greiss and Crandell[24] observed that UBF decreases in direct proportion to decreases in MAP caused by spinal anesthesia. In the current study, epidural anesthesia did not decrease UBF below baseline measurements despite the occurrence of modest hypotension in the magnesium sulfate group. Others have observed that magnesium sulfate slightly increases UBF in gravid ewes.[14,25] This effect seemed to protect UBF during hypotension from epidural anesthesia in the current study. We acknowledge that we measured total UBF, and that the observed changes do not necessarily reflect changes in placental perfusion.[26] However, we note that changes in fetal Pa_{O_2} seemed to parallel changes in UBF in both groups (figs. 2 and 4).

Although UBF, fetal Pa_{O_2}, and base excess were similar in the two groups during epidural lidocaine anesthesia, fetal arterial *p*H was slightly lower over time in the magnesium sulfate group. The higher maternal Pa_{CO_2} measurements in the magnesium sulfate group suggest that slight maternal hypoventilation occurred in that group. Carbon dioxide freely crosses the placenta, and increased maternal Pa_{CO_2} results in parallel increases in fetal Pa_{CO_2}.[27] Therefore, small increases in maternal and fetal Pa_{CO_2}, and not impaired oxygen delivery to the fetus, resulted in slightly lower fetal arterial *p*H measurements in the magnesium sulfate group. These findings are consistent with those of Ayromlooi *et al.*,[25] who also noted increased maternal and fetal Pa_{CO_2} measurements during magnesium sulfate infusion in gravid ewes. Likewise, magnesium sulfate tends to decrease maternal respiratory rate in human subjects.[28]

One should be cautious before extrapolating data from laboratory animals to clinical practice. First, the animals in this experiment were not in labor, and they were standing, not recumbent, during epidural anesthesia. Second, the lower extremity vascular bed in sheep represents a smaller proportion of the total vasculature than in humans. Thus, the current study may underestimate the effect of lumbar sympathetic blockade on vascular resistance and arterial pressure in humans. Third, although the lower thoracic sensory level in this study approximated

that which is adequate for labor analgesia, it represents a less extensive block than that achieved for cesarean section. Finally, we acknowledge that in our study the infusion of magnesium sulfate was continued *during* epidural anesthesia. It is unlikely that women receiving magnesium sulfate for tocolysis would continue to receive magnesium sulfate during epidural anesthesia. However, preeclamptic women who receive magnesium sulfate for seizure prophylaxis typically continue to receive magnesium sulfate during anesthesia.

We conclude that magnesium sulfate slightly worsened maternal hypotension during epidural lidocaine anesthesia in gravid ewes. However, magnesium sulfate did not decrease UBF or fetal oxygenation during epidural lidocaine anesthesia. If applicable to humans, magnesium sulfate may increase the likelihood of modest hypotension during epidural anesthesia in normotensive pregnant patients, but this hypotension may not be associated with increased fetal risk.

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