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Does Ritodrine Worsen Maternal Hypotension during Epidural Anesthesia in Gravid Ewes?

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The purpose of this study was to determine whether prior administration of ritodrine worsens maternal hypotension during epidural anesthesia in gravid ewes. Twenty-four experiments were performed in nine chronically instrumented animals between 0.8 and 0.9 of timed gestation. The experimental sequence included the following: 1) at time-zero, intravenous (iv) administration of ritodrine, 0.004 $mg \cdot kg^{-1} \cdot min^{-1}$, or normal saline (NS) for 2 h; 2) at 120 min discontinuation of ritodrine, and administration of a 500 ml iv bolus of NS over 15 min; and 3) at 135 min epidural injection of 2% lidocaine or NS. There were three groups of experiments: 1) iv ritodrine–epidural lidocaine (n = 9); 2) iv NS–epidural lidocaine (n = 8); and 3) iv ritodrine–epidural NS (n = 7). Epidural injection of lidocaine resulted in a median sensory level of T9 in both the ritodrine–lidocaine and NS–lidocaine groups. At 165 min (*i.e.*, 30 min after the epidural injection of lidocaine), maternal mean arterial pressure was 19 ± 3% below baseline ($P = 0.0001$) in the ritodrine–lidocaine group and 22 ± 7% below baseline ($P = 0.0001$) in the NS–lidocaine group (NS between groups). At 120 min ritodrine had increased maternal cardiac output 45 ± 6% above baseline ($P = 0.0001$) in the ritodrine–lidocaine group and 39 ± 6% above baseline ($P = 0.0001$) in the ritodrine–NS group. Cardiac output remained above baseline ($P < 0.01$) after epidural injection of lidocaine in the ritodrine–lidocaine group. In contrast, in the NS–lidocaine group cardiac output was 13 ± 5% below baseline ($P = 0.005$) at 150 min. Fetal arterial *p*H did not change significantly in either the ritodrine–lidocaine or ritodrine–NS group. In contrast, in the NS–lidocaine group fetal arterial *p*H was decreased ($P < 0.02$) at 210 min. Thus, prior administration of ritodrine did not adversely affect the maternal hemodynamic response to epidural lidocaine anesthesia in gravid ewes. (Key words: Anesthesia: obstetric. Anesthetics, local: lidocaine. Anesthetic techniques: epidural. Pregnancy: preterm labor. Sympathetic nervous system, β-sympathomimetic agents: ritodrine.)

OBSTETRICIANS often give a β-sympathomimetic agent to treat preterm labor or acute fetal distress. Unfortunately, tocolytic therapy is not uniformly successful, and parturients often require anesthesia after administration of a β-sympathomimetic agent. Shin and Kim¶ suggested that maternal hypotension is more common when induction of epidural anesthesia occurs within 30 min of discontinuation of ritodrine compared with a delay of greater than 30 min. Therefore, some anesthesiologists avoid regional anesthesia in patients who have recently received a β-sympathomimetic agent. The purpose of the present study was to determine whether ritodrine 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 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 40% nitrous oxide, 60% oxygen, and 1–1.5% halothane. Mechanical ventilation was maintained throughout surgery. Using sterile technique, a laparotomy and hysterotomy were performed, and catheters (polyethylene-90) were inserted into the fetal descending aorta *via* each femoral artery and into the amniotic cavity. 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, California) 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, Massachusetts) 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 (AK09800, Arrow, Reading, Pennsylvania) was placed percutaneously into the right jugular vein.

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¶ Shin YK, Kim YD: Anesthetic considerations in patients receiving ritodrine therapy for preterm labor (abstract). Anesth Analg 65(suppl): 140, 1986.

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 5 days. Procaine penicillin G 500,000 units and dihydrostreptomycin 625 mg (Combiotic®, Pfizer, New York, New York) were given to the mother intramuscularly before surgery and daily for 3 days after surgery. Gentamicin 80 mg was given to the mother intravenously on the day of each experiment, and 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 hindlimb weakness during epidural anesthesia. (We had performed a pilot study in which the animals were restrained in the lateral decubitus position, but the animals developed agitation and tachycardia.)

Before the first experiment in each animal, a pulmonary artery catheter (#93A-131H-7F, American Edwards Laboratories, Santa Ana, California) was inserted through the jugular vein introducer. At the end of each experiment, the catheter was withdrawn into the superior vena cava, and sterility was maintained with an 80-cm sheath. Maternal arterial blood pressure, central venous pressure, pulmonary artery pressure, and fetal arterial blood pressure were measured continuously *via* disposable strain gauge pressure transducers (#46951-02, Abbott Critical Care Systems, North Chicago, Illinois). 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, California). Arterial and venous pressures, HR, and UBF were recorded at 10-s intervals using a customized data acquisition system (Coulbourn Instruments, Lehigh Valley, Pennsylvania) interfaced with a personal computer (PCXT, IBM, Rochester, Minnesota).

Cardiac output measurements were made in triplicate with 10 ml of iced saline and a thermodilution cardiac output computer (#9520, Edwards Laboratories, Santa Ana, California). Maternal and fetal arterial blood gas and *p*H values were determined using an Instrumentation Laboratory (#1302, Leighton, Massachusetts) blood gas analyzer. All values were corrected for temperature (39.5° C).

The experimental sequence included the following:

1. One hour for baseline measurements.
2. At time-zero iv administration of ritodrine, 0.004 $mg \cdot kg^{-1} \cdot min^{-1}$, or normal saline (NS) for 2 h. This dose of ritodrine was within the range used clinically (*i.e.*, approximately 0.001–0.005 $mg \cdot kg^{-1} \cdot min^{-1}$). The total volume of crystalloid was 100 ml/h in each group.
3. At 120 min the infusion of ritodrine was discontinued. Each animal then received a 500-ml iv bolus of NS over 15 min. (We gave a bolus of NS in anticipation of the sympathetic block that occurs during epidural anesthesia. Similarly, in clinical practice the anesthesiologist typically gives a bolus of crystalloid before epidural anesthesia.)
4. At 135 min each animal received 8 ml of 2% lidocaine or NS *via* the epidural catheter. Each animal continued to receive NS at 100 ml/h. If hypotension occurred, we did not increase the rate of crystalloid administration or give a vasopressor agent.
5. At 150 min (*i.e.*, 15 min after the epidural injection of lidocaine) a curved hemostat was used to determine the sensory level of anesthesia. (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.)
6. Hemodynamic measurements were continued through 210 min.

Twenty-four experiments were performed in nine animals. There were three groups of experiments: 1) iv ritodrine–epidural lidocaine (n = 9); 2) iv NS–epidural lidocaine (n = 8); and 3) iv ritodrine–epidural NS (n = 7). Only one experiment was performed per day, and each animal rested at least 48 h between experiments. Experiments were performed in random order. (We intended to perform all three experiments in each animal. However, catheter occlusion or fetal death precluded the performance of all three experiments in two of the animals.)

Statistical analysis was by three-factor analysis of variance, followed by *t* tests for individual measurements. $P < 0.05$ was considered significant.

Results

The mean (±SEM) weight of the animals was 61.6 ± 2.3 kg. The three groups were similar with regard to baseline maternal and fetal hemodynamic, acid–base, and blood gas measurements (table 1).

Epidural injection of lidocaine resulted in a median sensory level of T9 in both the ritodrine–lidocaine and NS–lidocaine groups. Epidural injection of NS did not result in a detectable level of sensory anesthesia.

At 120 min ritodrine had increased maternal HR 47 ± 8% above baseline ($P = 0.0001$) in the ritodrine–lidocaine group and 37 ± 6% above baseline ($P = 0.0001$) in the ritodrine–NS group (NS between groups) (fig. 1). At 135 min, just before the epidural injection of lidocaine or NS, maternal HR remained 30 ± 6% above baseline ($P = 0.0001$) in the ritodrine–lidocaine group and 22

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

	Ritodrine–Lidocaine (n = 9)	NS–Lidocaine (n = 8)	Ritodrine–NS (n = 7)
Maternal			
Heart rate (beats/min)	123 ± 4	115 ± 3	128 ± 3
Mean arterial pressure (mmHg)	94 ± 3	94 ± 1	95 ± 2
Cardiac output (l/min)	9.6 ± 0.6	9.4 ± 0.8	9.7 ± 0.6
Uterine blood flow (ml/min)	715 ± 123	818 ± 149	754 ± 117
Pulmonary capillary wedge pressure (mmHg)	7.4 ± 1.9	9.1 ± 1.7	10.3 ± 0.9
Central venous pressure (mmHg)	1.8 ± 1.8	2.9 ± 1.4	4.8 ± 1.0
*p*H	7.45 ± 0.01	7.45 ± 0.01	7.46 ± 0.01
P_{O_2} (mmHg)	99 ± 2	100 ± 3	108 ± 3
P_{CO_2} (mmHg)	36 ± 1	37 ± 1	35 ± 1
Fetal			
Heart rate (beats/min)	165 ± 3	170 ± 5	161 ± 9
Mean arterial pressure (mmHg)	45 ± 1	43 ± 1	47 ± 2
*p*H	7.35 ± 0.01	7.35 ± 0.01	7.35 ± 0.01
P_{O_2} (mmHg)	21 ± 1	19 ± 1	21 ± 1
P_{CO_2} (mmHg)	48 ± 1	49 ± 1	50 ± 2

Values are mean ± SEM.

± 4% above baseline ($P = 0.0001$) in the ritodrine–NS group (NS between groups). Maternal HR remained above baseline ($P < 0.05$) through 160 min in the ritodrine–lidocaine group and 165 min in the ritodrine–NS group. In contrast, in the NS–lidocaine group maternal HR was below baseline ($P < 0.005$) at 165 and 210 min. Specifically, at 165 min maternal HR was 16 ± 8% below baseline ($P < 0.005$).

At 120 min ritodrine had decreased maternal MAP 8 ± 3% below baseline ($P < 0.05$) in the ritodrine–lidocaine group and 9 ± 2% below baseline ($P < 0.05$) in the ritodrine–NS group (fig. 2). There was no significant difference between the ritodrine–lidocaine and the NS–lidocaine groups in the maternal MAP response to epidural lidocaine anesthesia. Epidural lidocaine decreased maternal MAP ($P = 0.0001$) at each measurement between 140 and 210 min in the ritodrine–lidocaine group and between 145 and 210 min in the NS–lidocaine group. At 165 min (*i.e.*, 30 min after the epidural injection of lidocaine) maternal MAP was 19 ± 3% below baseline ($P = 0.0001$) in the ritodrine–lidocaine group and 22 ± 7% below baseline ($P = 0.0001$) in the NS–lidocaine group. Epidural injection of NS did not change maternal MAP in the ritodrine–NS group.

At 120 min ritodrine had increased maternal cardiac output 45 ± 6% above baseline ($P = 0.0001$) in the ritodrine–lidocaine group and 39 ± 6% above baseline ($P = 0.0001$) in the ritodrine–NS group (fig. 3). Cardiac output remained above baseline ($P < 0.01$) at each measurement between 120 and 210 min in both the ritodrine–lidocaine and ritodrine–NS groups. In contrast, at 150 min (*i.e.*, 15 min after the epidural injection of lidocaine) cardiac output was 13 ± 5% below baseline ($P = 0.005$) in the NS–lidocaine group. Furthermore, cardiac output was higher ($P < 0.01$) in the ritodrine–lidocaine group than in the NS–lidocaine group at each measurement between 120 and 210 min.

At 120 min, ritodrine had decreased UBF 15 ± 4% ($P < 0.002$) in the ritodrine–lidocaine group and 22 ± 2% ($P < 0.002$) in the ritodrine–NS group (NS between

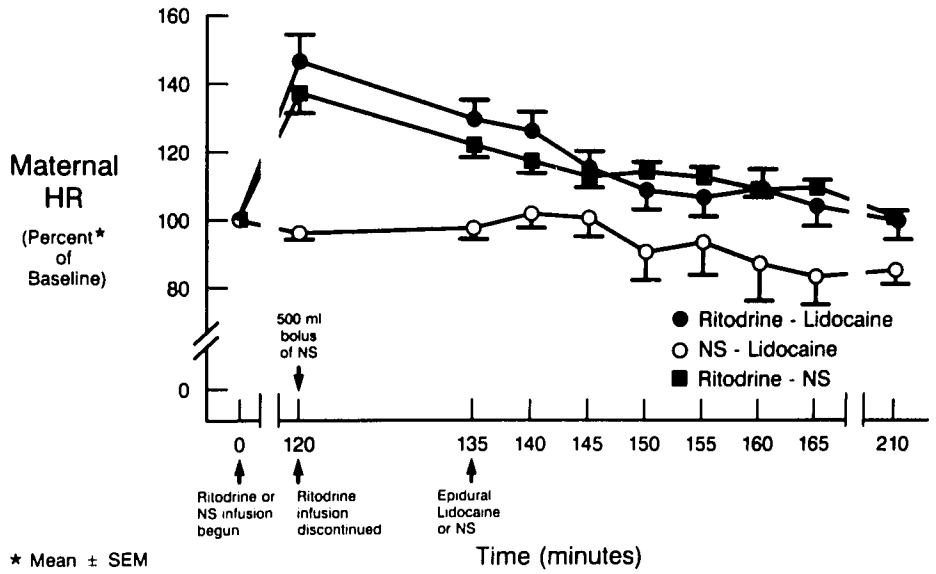

FIG. 1. Maternal heart rate (HR) responses over time. Maternal HR was above baseline ($P < 0.05$) at each measurement between 120 and 160 min in the ritodrine–lidocaine group and between 120 and 165 min in the ritodrine–NS group. In contrast, in the NS–lidocaine group maternal HR was below baseline ($P < 0.005$) at 165 and 210 min.

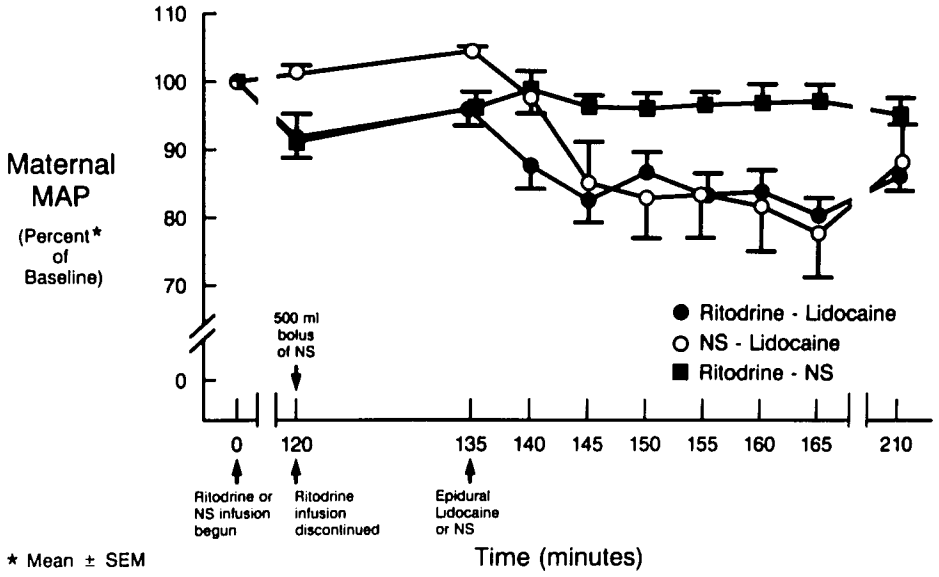

FIG. 2. Maternal mean arterial pressure (MAP) responses over time. Ritodrine decreased maternal MAP ($P < 0.05$) at 120 min in both the ritodrine–lidocaine and ritodrine–NS groups. Epidural lidocaine decreased maternal MAP ($P = 0.0001$) at each measurement between 140 and 210 min in the ritodrine–lidocaine group and between 145 and 210 min in the NS–lidocaine group.

groups) (fig. 4). UBF did not change significantly during the first 30 min after epidural injection of lidocaine (*i.e.*, 135–165 min) in either the ritodrine–lidocaine or the NS–lidocaine group. However, at 210 min UBF was above baseline ($P < 0.001$) in both the ritodrine–lidocaine and ritodrine–NS groups but not in the NS–lidocaine group.

Fetal HR and fetal MAP did not change significantly from baseline in any group (data not shown).

A small but significant decrease ($P < 0.001$) in maternal arterial *p*H occurred over time in each of the three groups (fig. 5). Maternal arterial P_{O_2} and P_{CO_2} did not change significantly in any group (data not shown).

Fetal arterial *p*H did not change significantly in either the ritodrine–lidocaine or ritodrine–NS group (fig. 6). In contrast, fetal arterial *p*H was decreased ($P < 0.02$) at 210 min in the NS–lidocaine group. Fetal arterial P_{O_2} and P_{CO_2} did not change significantly in any group (data not shown).

Discussion

Preterm labor and delivery is by far the most common cause of perinatal morbidity and mortality in the United States.[1] Obstetricians often give a β-sympathomimetic agent (*e.g.*, ritodrine, terbutaline) for tocolysis (*i.e.*, the treatment of preterm labor by inhibition of uterine muscle contractions). Ritodrine is the only agent specifically approved by the U. S. Food and Drug Administration

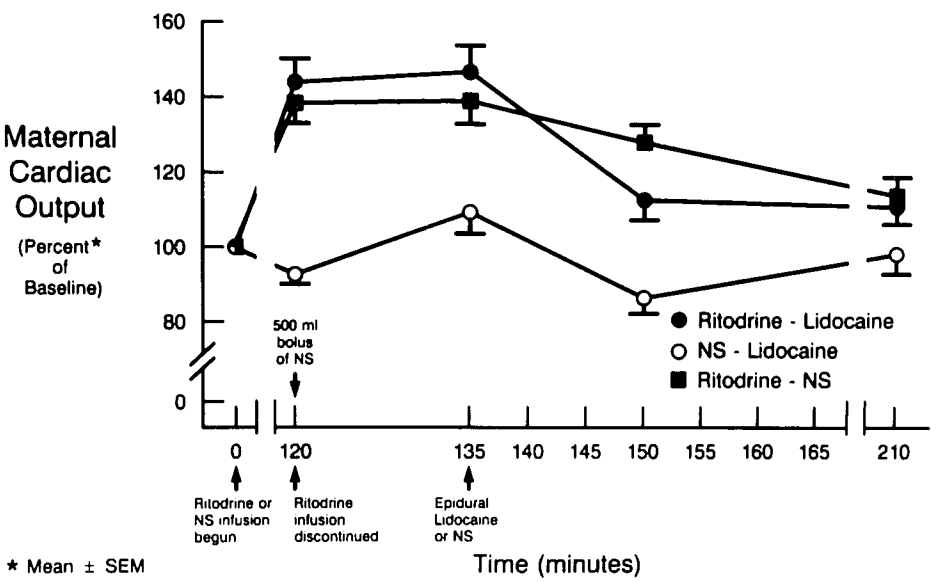

FIG. 3. Maternal cardiac output responses over time. Cardiac output was above baseline ($P < 0.01$) at each measurement between 120 and 210 min in both the ritodrine–lidocaine and ritodrine–NS groups. In contrast, in the NS–lidocaine group cardiac output was below baseline ($P = 0.005$) at 150 min.

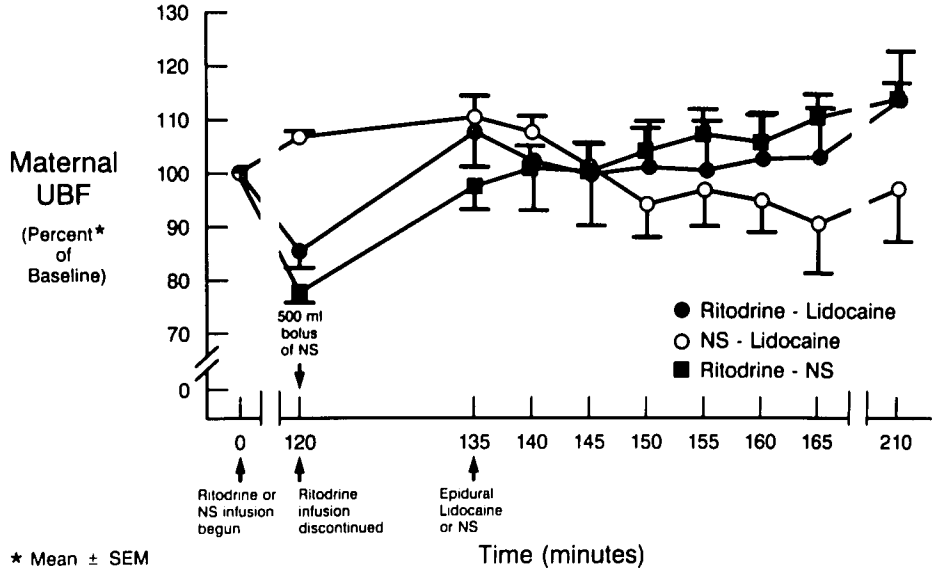

FIG. 4. Maternal uterine artery blood flow (UBF) responses over time. Ritodrine decreased UBF ($P < 0.002$) at 120 min in both the ritodrine–lidocaine and ritodrine–NS groups. UBF did not change significantly between 135 and 165 min in either the ritodrine–lidocaine or the NS–lidocaine group. However, at 210 min UBF was above baseline ($P < 0.001$) in both the ritodrine–lidocaine and ritodrine–NS groups but not in the NS–lidocaine group.

for tocolysis. Although ritodrine and terbutaline are relatively selective for the β_2 receptor (*e.g.*, uterine smooth muscle), β_1-receptor stimulation does occur, resulting in increased maternal HR and systolic arterial pressure, decreased diastolic arterial pressure, and unchanged or decreased MAP.[2–5]

There are at least three situations in which obstetric patients require anesthesia during or after infusion of a β-sympathomimetic tocolytic agent. First, despite use of tocolytic agents, preterm delivery still occurs. These parturients may desire epidural analgesia for relief of pain during labor and vaginal delivery, or they may require regional or general anesthesia for operative delivery. Second, some obstetricians advocate the infusion of ritodrine or terbutaline before and during performance of cervical cerclage. Third, obstetricians recently have recommended bolus injection of a tocolytic agent for treatment of fetal distress associated with uterine activity.[6,7] The goal is to improve uteroplacental blood flow by abolishing uterine contractions and facilitate fetal resuscitation *in utero*. Although this may allow obstetricians to forego operative delivery in some patients, the remaining women will require emergency induction of anesthesia for cesarean section.

Unfortunately, the half-lives of the β-sympathomimetic tocolytic agents in pregnant women are prolonged. Kuhnert *et al.*[8] noted that distribution phase and equilibrium phase half-lives for ritodrine in pregnant women are

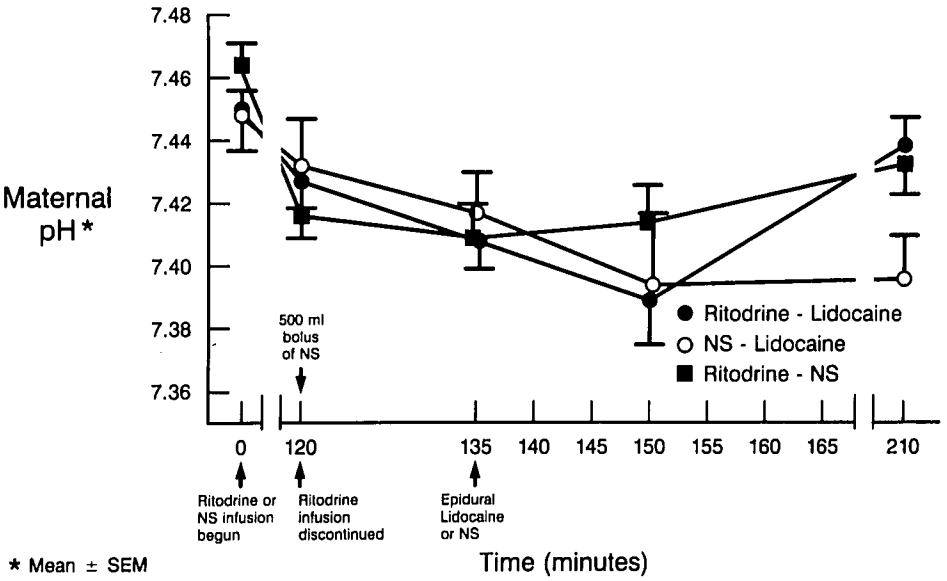

FIG. 5. Maternal arterial *p*H measurements over time. A small but significant decrease ($P < 0.001$) occurred over time in each of the three groups.

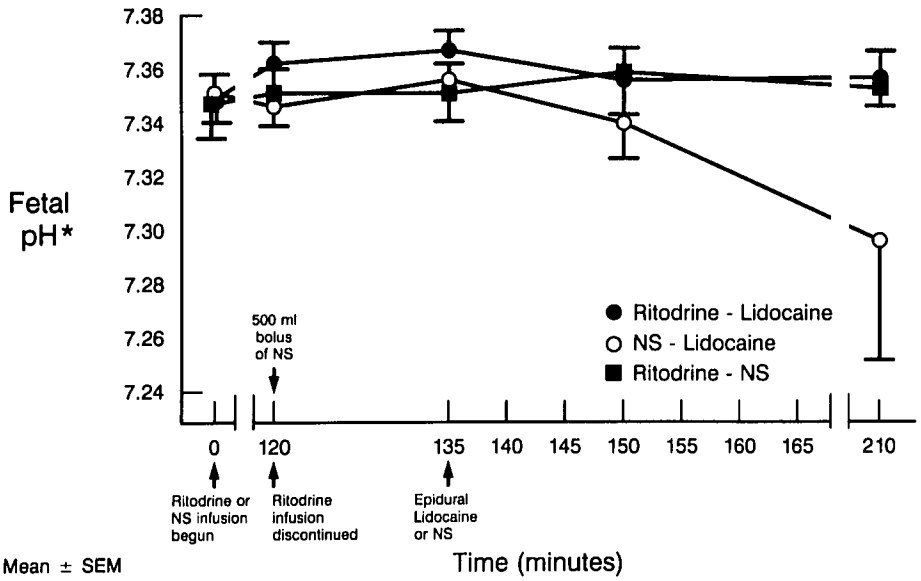

FIG. 6. Fetal arterial *p*H measurements over time. Fetal *p*H was decreased ($P < 0.02$) at 210 min in the NS–lidocaine group.

32 ± 21 min and 17 ± 10 h, respectively. The cardiovascular effects of ritodrine and other β-sympathomimetic agents also persist after their discontinuation.[2,4,¶] There are no prospective studies regarding anesthetic management after administration of a β-sympathomimetic tocolytic agent. The literature is limited to case reports,[9–14] retrospective studies published in abstract and letter form,[15,¶] and review articles and textbook chapters. Shin and Kim¶ observed that maternal hypotension occurred in two of three women who received epidural anesthesia within 30 min of discontinuation of ritodrine *versus* one of eight women in whom there was a delay of more than 30 min ($P = 0.15$). They recommended "that anesthesia be deferred at least 30 min following discontinuation of ritodrine . . . provided that such a delay does not jeopardize the fetus."¶ Abouleish[16] stated that regional anesthesia may be administered if the β-sympathomimetic agent has been discontinued for at least 2 h before cesarean section.

The aforementioned contentions notwithstanding, women with failed tocolysis typically require induction of anesthesia on an emergency basis. A delay may compromise fetal well-being. A proscription against regional anesthesia deprives the parturient of enjoying the emotional experience associated with birth and subjects her to the risks of general anesthesia. The majority of anesthetic deaths in obstetric patients occur with general, not regional, anesthesia and result primarily from failure to intubate the trachea and/or aspiration of gastric contents.[17] Thus, there should be careful scrutiny of admonitions to avoid regional anesthesia in patients with failed tocolysis. Heretofore there has been no controlled study of hemodynamic responses to epidural, spinal, or general anesthesia after infusion of a β-sympathomimetic tocolytic agent.

We recently reported that administration of ritodrine did not worsen maternal hypotension during hemorrhage in gravid ewes.[18] In that study we speculated that ritodrine's inotropic and chronotropic activity[2–5] helped maintain maternal cardiac output and MAP during hemorrhage. In the present study prior administration of ritodrine did not worsen maternal hypotension during epidural lidocaine anesthesia in gravid ewes. One should be cautious before extrapolating data from animals to clinical practice. First, we acknowledge the difference in species. Second, the animals in this study were standing, not recumbent, during epidural anesthesia. Third, the animals were not in labor. Fourth, although the thoracic sensory level in this study approximated that which is achieved clinically for labor analgesia, it represented a less extensive block than that which is achieved for cesarean section. However, we have successfully administered epidural anesthesia to patients who recently received a β-sympathomimetic agent, provided that tachycardia was not excessive. In the present study maternal HR remained 30% above baseline at the time that epidural lidocaine was given in the ritodrine–lidocaine group. Nonetheless, there was no significant difference in the maternal MAP response to epidural lidocaine between the ritodrine–lidocaine and NS–lidocaine groups. Furthermore, maternal cardiac output was consistently greater in the ritodrine–lidocaine group than in the NS–lidocaine group, and fetal arterial *p*H was decreased only in the NS–lidocaine group.

We conclude that prior administration of ritodrine did not worsen maternal hypotension during epidural lidocaine anesthesia in gravid ewes. Rather, the present study

suggests that the inotropic and chronotropic activity of ritodrine helped maintain maternal cardiac output and UBF during epidural anesthesia. We suggest that there should be reevaluation of admonitions against the use of epidural anesthesia in patients who have recently received a β-sympathomimetic agent.

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References

1. Gonik B, Creasy RK: Preterm labor: Its diagnosis and management. Am J Obstet Gynecol 154:3–8, 1986
2. Barden TP: Effect of ritodrine on human uterine motility and cardiovascular responses in term labor and the early postpartum state. Am J Obstet Gynecol 112:645–652, 1972
3. Bieniarz J, Ivankovich A, Scommegna A: Cardiac output during ritodrine treatment in premature labor. Am J Obstet Gynecol 118:910–920, 1974
4. Ehrenkranz RA, Walker AM, Oakes GK, McLaughlin MK, Chez RA: Effect of ritodrine infusion on uterine and umbilical blood flow in pregnant sheep. Am J Obstet Gynecol 126:343–349, 1976
5. Hosenpud JD, Morton MJ, O'Grady JP: Cardiac stimulation during ritodrine hydrochloride tocolytic therapy. Obstet Gynecol 62: 52–58, 1983
6. Arias F: Intrauterine resuscitation with terbutaline. Am J Obstet Gynecol 131:39–43, 1978
7. Caritis SN, Lin LS, Wong LK: Evaluation of the pharmacodynamics and pharmacokinetics of ritodrine when administered as a loading dose: On establishing a potentially useful drug administration regimen in cases of fetal distress. Am J Obstet Gynecol 152: 1026–1031, 1985
8. Kuhnert BR, Gross TL, Kuhnert PM, Erhard P, Brashar WT: Ritodrine pharmacokinetics. Clin Pharmacol Ther 40:656–664, 1986
9. Knight RJ: Labour retarded with β-agonist drugs. A therapeutic problem in emergency anesthesia. Anaesthesia 32:639–641, 1977
10. Schoenfeld A, Joel-Cohen SJ, Duparc H, Levy E: Emergency obstetric anaesthesia and the use of β_2-sympathomimetic drugs. Br J Anaesth 50:969–971, 1978
11. Crowhurst JA: Salbutamol, obstetrics and anaesthesia: A review and case discussion. Anaesth Intensive Care 8:39–43, 1980
12. Ravindran R, Viegas OJ, Padilla LM, LaBlonde P: Anesthetic considerations in pregnant patients receiving terbutaline therapy. Anesth Analg 59:391–392, 1980
13. Simpson JI, Giffin JP: A glycopyrrolate–ritodrine drug–drug interaction. Can J Anaesth 35:187–189, 1988
14. Shin YK, Kim YD: Ventricular tachyarrhythmias during cesarean section after ritodrine therapy: Interaction with anesthetics. South Med J 81:528–530, 1988
15. Suppan P: Tocolysis and anaesthesia for caesarean section (letter). Br J Anaesth 54:1007, 1982
16. Abouleish E: Preterm labor, Common Problems in Obstetric Anesthesia. Edited by Datta SJ, Ostheimer GW. Chicago, Year Book, 1987, pp 279–285
17. Morgan M: Anaesthetic contribution to maternal mortality. Br J Anaesth 59:842–855, 1987
18. Chestnut DH, Thompson CS, McLaughlin GL, Weiner CP: Does the intravenous infusion of ritodrine or magnesium sulfate alter the hemodynamic response to hemorrhage in gravid ewes? Am J Obstet Gynecol 159:1467–1473, 1988