

Effect of oxytocin treatment in sows on umbilical cord morphology, meconium staining, and neonatal mortality of piglets

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Objective—To evaluate the effect of 2 oxytocin products administered to sows at the onset of fetal expulsion on the integrity of umbilical cords, meconium staining, and piglet mortality.

Animals—2099 neonatal pigs.

Procedure—180 parturient sows were randomly assigned to 3 stratified groups of 60 sows each. Two groups of sows were injected IM at the onset of fetal expulsion with 1 of 2 oxytocin commercial products (20, 40, or 50 U for sows weighing 120 to 150 kg, 151 to 250 kg, or \geq 251 kg, respectively). Control sows were treated IM with saline (0.9% NaCl) solution. Farrowing time, expulsion intervals, and numbers of stillborn and liveborn piglets were recorded for each sow. Piglets were evaluated for inspiratory effort, heart rates, and degree of meconium staining of skin (nonstained, and moderately or severely stained). Umbilical cords were classified as normal in appearance, edematous, congested, hemorrhagic, or ruptured.

Results—Oxytocin-treated sows had a significant decrease in farrowing time and expulsion intervals and also had a significantly higher number of stillborn piglets per litter, compared with control sows. The number of piglets per litter with ruptured and hemorrhagic umbilical cords was significantly greater in oxytocin-treated sows, compared with control sows. In near-death stillborn piglets, oxytocin treatment significantly decreased inspiratory efforts at birth and increased the rate and severity of meconium staining, compared with saline treatment.

Conclusions and Clinical Relevance—Oxytocin given to sows at the onset of fetal expulsion significantly increases the rate of fetal distress, anoxia, and intrapartum death in piglets. (*Am J Vet Res* 2002;63:1571–1574)

Despite current advances in drug treatment and obstetric interventions, stillbirths remain a major problem in intensive pig farming and account

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for 5 to 10% of herd mortality.¹⁻⁵ According to the time of death, stillbirths are subdivided into 2 distinct types. In type-I stillbirths, death occurs before parturition (prepartum or antepartum), and the cause of fetal death is generally attributed to intrauterine infection. In type-II stillbirths, death occurs during parturition (intrapartum), and it is generally associated with a noninfectious cause such as anoxia and dystocia.^{6,7}

Birth order of piglets and the time interval between the expulsion of 2 successive piglets are also important factors in stillbirths in pigs.^{7,9} According to 1 report, approximately 80% of intrapartum deaths occur in piglets born in the last third of a litter.⁸ Intrapartum anoxia or asphyxia during farrowing or immediately after birth are the most important events leading to type-II stillbirths in pigs.^{8,10,11} An increase in tensile stress on the umbilical cord during parturition often results in cord injury, an increase in the risk of intrapartum anoxia, and a high rate of perinatal mortality.^{8,12}

When fetal anoxia occurs, blood redistribution in the fetus results in an increase in intestinal peristalsis, relaxation of the anal sphincter, and defecation of meconium into the amniotic fluid.^{6,13} Following severe anoxia, hypoxemic fetuses gasp violently inside of the amniotic sac, causing the passage (inhalation) of amniotic fluid and meconium into the lungs. Newborns with severe airway obstruction may die at birth or survive and develop meconium aspiration syndrome.¹⁴ Morbidity and mortality attributable to meconium aspiration, and its impact on neonates, has been well documented in babies but remains still unsettled in domestic and laboratory animals.¹⁴⁻¹⁶ In human obstetrics, the degree of meconium staining in the amniotic fluid and in the skin is used as a visual predictor of intrapartum anoxia and impending meconium aspiration syndrome in newborn babies.^{17,18} This procedure has rarely been used in the study of neonates in veterinary medicine even though several reports have indicated that calves, lambs, and foals born with meconium-stained skin are weaker and more susceptible to perinatal death than nonstained neonates.¹⁹ The relationships between meconium passage and intrapartum anoxia, and the staining of the skin and umbilical cord damage, has never properly been investigated in pigs.

Intrapartum anoxia and aspiration of amniotic fluid have been linked to an increase rate of type-II stillbirths and a decrease rate of postnatal survival in piglets.^{8,20} It remains unknown, however, whether uterotrophic drugs such as oxytocin, which is com-

monly used in parturient sows, have an effect on the rate of type-II stillbirths, umbilical cord damage, magnitude of meconium expulsion, and frequency of meconium aspiration syndrome in piglets. Furthermore, in the study of human neonates, it has been reported that high blood concentrations of oxytocin increases the risk for umbilical cord disorders, hypoxia, and meconium aspiration syndrome.²¹ This issue is particularly intriguing for veterinarians, because although oxytocin is commonly used to induce farrowing and reduce the parturition time in sows, this hormone has also been associated with dystocia in sows and an increase need for manual assistance.²²⁻²⁴ The purpose of the study reported here was to evaluate the effect of 2 oxytocin products administered to sows at the onset of fetal expulsion on the integrity of umbilical cords, meconium staining, and piglet mortality.

Materials and Methods

Animals—Our study was conducted from June to October 1998 in a 900-sow pig farm located in Tamaulipas, Mexico. Sows were Yorkshire X Landrace crossbreds (n = 180) of mixed parity, all of which were in close proximity to farrowing. Sows did not receive any treatment to synchronize or induce farrowing. Four to 6 days before the expected date of parturition, sows were moved and kept individually housed in plastic-floor farrowing pens. To avoid confounding effects on piglet survival, no obstetric interventions were done during farrowing.

Procedures—Sows were injected IM in the neck with oxytocin or saline (0.9% NaCl) solution immediately after the first piglet was born. The dose of oxytocin was calculated by body weight as follows: sows weighing 120 to 150 kg were given 20 U of oxytocin, sows weighing 151 to 250 kg were given 40 U, and those weighing more than 251 kg received 50 U. Sows were continuously monitored beginning at 72 hours prior to the expected delivery time and during parturition. Newborn piglets were classified as live or stillborn. Live piglets were monitored for 72 hours. The farrowing time, expulsion interval between piglets, birth order of piglets, sow parity, and health status of piglets were also recorded. At birth, piglets were designated as nonstained or meconium-stained according to the degree (moderate or severe) of skin discoloration. Stillborn piglets were assessed as having antepartum (type-I stillbirths) or intrapartum (type-II stillbirths) deaths following the criteria reported by others.^{6,20,23,26} In short, type-I stillbirths included fetuses that died before the end of gestation and had autolysis, subcutaneous edema, or emphysema. Type-II stillbirths included fresh fetuses that lacked autolytic changes, but may have had gross evidence of ascites and hydrothorax or petechia. Dead piglets with partially inflated lungs were considered type-II stillbirths.

Intrapartum stillborn piglets were grouped according to birth order as follows: first to fourth piglets of litter, fifth to eighth piglets of litter, and all remaining piglets of litter. Umbilical cords were grossly examined at birth and classified as normal or ruptured.

According to circulatory abnormalities in the umbilical cord, changes were classified as normal, edematous, congested, or hemorrhagic. In intrapartum stillborn piglets (ie, those that died during or immediately following parturition), heart rates (beats/min) were measured by use of a stethoscope, and inspiratory efforts were assessed by simple observation of thoracic movements.

Experimental design—Sows were randomly assigned to 3 experimental groups. Each experimental group included 60

sows of mixed parity. Sows in the control group received an IM injection of saline solution. There were 2 oxytocin treatment groups; sows in each treatment group received 1 of 2 commercially available oxytocin^{a,b} products IM.

Statistical analyses—Data are expressed as mean \pm SEM. The effect of treatment on the number of liveborn and stillborn piglets, farrowing times, expulsion intervals, meconium staining, umbilical cord morphology, inspiratory effort, and heart rates were analyzed by use of a 1-way ANOVA and the Tukey-B test. The effect of treatment on intrapartum death by order of birth was tested using the χ^2 test.²⁷ Differences were considered significant at values of $P < 0.05$.

Results

Treatment with oxytocin did not have an effect on the mean number of liveborn piglets per litter. The frequency of antepartum fetal deaths ranged from 0.13 to 0.15 piglets/litter in all groups, with no significant differences between control and oxytocin-treated groups. In contrast, the number of intrapartum deaths was significantly ($P = 0.009$) higher in piglets of oxytocin-treated sows (Table 1), compared with control sows. There was no significant difference in the number of intrapartum stillborn piglets per litter between treatments with the 2 types oxytocin products. Oxytocin administered to sows immediately after the birth of the first piglet significantly ($P < 0.001$) decreased the overall farrowing time and interval expulsion time between piglets. No difference in the interval expulsion time was detected between sows injected with the 2 types of oxytocin products.

According to the birth order of piglets, 9% of stillbirths in control sows occurred in piglets 1 to 4, 16% in piglets 5 to 8, and 75% in piglets ≥ 9 . In contrast, more than 80% of stillbirths in oxytocin-treated sows occurred in piglets 1 to 4 of the birth order ($P < 0.001$; Table 2). The incidence and severity of meconium-stained skin was significantly ($P < 0.001$) higher in piglets of oxytocin-treated sows, compared with control sows (Table 3). There was no difference in the number of stillborn piglets with moderately stained skin between oxytocin-treated and control sows.

Treatment with oxytocin caused a significant ($P < 0.001$) increase in the number of stillborn piglets per litter with umbilical cord hemorrhage and rupture

Table 1—Mean (\pm SEM) farrowing variables in saline (0.9% NaCl) solution-treated (control) sows and oxytocin-treated sows

Variables	Sow group		
	Control (n = 60)	Oxytocin-treated (n = 60)	Oxytocin-treated (n = 60)
No. of live births (piglets/litter)	10.41 \pm 0.16	10.53 \pm 0.16	10.56 \pm 0.17
No. of AP stillbirths (piglets/litter)	0.15 \pm 0.05	0.13 \pm 0.05	0.15 \pm 0.05
No. of IP stillbirths (piglets/litter)	0.73 \pm 0.13 ^a	1.16 \pm 0.09 ^b	1.16 \pm 0.11 ^b
Farrowing time (min)	316.68 \pm 9.70 ^a	166.31 \pm 4.86 ^b	173.98 \pm 4.56 ^b
Interval expulsion time (min)	28.54 \pm 0.63 ^a	14.02 \pm 0.34 ^b	14.72 \pm 0.38 ^b

^{a,b}Within a row, values with different superscript letters are significantly ($P < 0.05$) different.
AP = Antepartum. IP = Intrapartum.

(Table 4). In oxytocin-treated sows, all intrapartum stillborn piglets with a ruptured umbilical cord (100%) and 63 to 71% of stillborn piglets with umbilical hemorrhage were born in the first third of the litter. These findings were significantly ($P < 0.001$) different when compared with those of control sows. Inspiratory effort by near-death stillborn piglets was significantly ($P < 0.007$) decreased in piglets of oxytocin-treated sows, compared with control sows. Interestingly, the decrease in the frequency of inspiratory effort of near-death stillborn piglets was also significantly ($P = 0.012$) different between the 2 groups of oxytocin-treated sows. Near-death stillborn piglets of oxytocin-treated sows had a significantly ($P = 0.007$) decreased heart rate at birth, compared with control sows.

Discussion

As it may be expected, the farrowing time in the control sows was considerably longer than that observed

Table 2—Number (%) of intrapartum stillbirths of control and oxytocin-treated sows

Intrapartum stillbirths (No.)	Sow group		
	Control (n = 60)	Oxytocin-treated (n = 60)	Oxytocin-treated (n = 60)
Total piglets	44	71	70
Sows	22 (36.6%)	50 (83.3%)	48 (80%)
Birth order piglets 1 to 4	4 (9.1%)	58 (81.7%)*	62 (88.6%)*
Birth order piglets 5 to 8	7 (15.9%)	5 (7.0%)	2 (2.8%)
Birth order piglets ≥ 9	33 (75%)	8 (11.3%)*	6 (8.6%)*

*Significantly ($P < 0.05$) different compared with control sows.

Table 3—Mean (\pm SEM) number of meconium-stained liveborn and stillborn piglets per litter of control and oxytocin-treated sows

Piglets		Sow group		
		Control (n = 60)	Oxytocin-treated (n = 60)	Oxytocin-treated (n = 60)
Birth status	Grade of stain			
Liveborn	Moderate	0.28 \pm 0.07 ^a	0.60 \pm 0.07 ^b	0.80 \pm 0.09 ^c
Liveborn	Severe	0.08 \pm 0.03 ^a	0.45 \pm 0.07 ^b	0.50 \pm 0.09 ^b
Stillborn	Moderate	0.63 \pm 0.11	0.75 \pm 0.08	0.63 \pm 0.08
Stillborn	Severe	0.10 \pm 0.00 ^a	0.43 \pm 0.08 ^b	0.60 \pm 0.09 ^c

^{a,b,c}Within a row, values with different superscript letters are significantly ($P < 0.05$) different.

Table 4—Mean (\pm SEM) number of intrapartum stillborn piglets (ie, those that died during or immediately following parturition) per litter with various variables born to control and oxytocin-treated sows

Variables (stillborn piglets/litter)	Sow group		
	Control (n = 60)	Oxytocin-treated (n = 60)	Oxytocin-treated (n = 60)
Umbilical cord abnormalities			
Edematous	0.26 \pm 0.06 ^a	0.13 \pm 0.04 ^{ab}	0.08 \pm 0.03 ^b
Congested	0.25 \pm 0.05 ^a	0.11 \pm 0.04 ^{ab}	0.08 \pm 0.03 ^b
Hemorrhagic	0.05 \pm 0.02 ^a	0.36 \pm 0.06 ^b	0.21 \pm 0.05 ^b
Ruptured	0.15 \pm 0.05 ^a	0.55 \pm 0.09 ^b	0.73 \pm 0.10 ^b
Inspiratory effort	0.35 \pm 0.08 ^a	0.10 \pm 0.03 ^a	0.00 \pm 0.00 ^c
Detectable heart rate	0.50 \pm 0.10 ^a	0.16 \pm 0.05 ^a	0.08 \pm 0.03 ^b

^{a,b,c}Within a row, values with different superscript letters are significantly ($P < 0.05$) different.

in oxytocin-treated sows. This observation is consistent with the literature and simply reflects the farrowing acceleration induced by uterotrophic drugs such as oxytocin.^{1,12,28} Also, the shorter expulsion intervals between piglets of oxytocin-treated sows supports the view that this hormone administered after the birth of the first piglet facilitates expulsion of subsequent litter mates through an increase in uterine contractions.²⁹ The dose and timing of oxytocin injection did not induce uterine inertia, as has been reported when a comparable dose of the hormone is given along with prostaglandins.^{22,24}

The mean expulsion time interval between piglets farrowed by control sows was notably longer than the times reported for clinically normal sows by other researchers.^{5,8,9,30,31} The reason for this discrepancy between nontreated clinically normal sows is not clear, but it may be related to the confounding effect of husbandry practices in Mexico versus the United States and Canada, as well as the stressor effect of sow restraining used in our study.¹¹ Also, the continuous presence of 1 or more people monitoring the parturition in the farrowing barn may have been disruptive and may have influenced the length of parturition. In contrast, the presence of the observer in the barn was not considered to be an important source of error in heart rate monitoring, because the newborn piglets were lying down on the floor motionless for several minutes and did not require any type of manipulation.

Results of our investigation indicate that oxytocin causes important umbilical cord abnormalities. For instance, the percentage of piglets born with ruptured umbilical cords in the 2 groups of oxytocin-treated sows was significantly higher (55 and 73%) than the 15% found in control sows. According to data from other investigators, up to 33% of piglets in normal deliveries may be born with ruptured umbilical cord.³⁰ Results of our investigation indicate that administration of oxytocin decreases the birth intervals, but notably increases the risk for trauma and rupture of the umbilical cord.

Because the incidence of hemorrhagic and ruptured umbilical cords in oxytocin-treated sows was significantly higher and closely paralleled the percentage of intrapartum stillborn piglets, it is reasonable to suggest that umbilical injury caused by oxytocin resulted in asphyxia and death in most nonviable piglets. This view is supported by other studies^{5,6,8,25} that have reported that as many as 94% of intrapartum stillborn piglets have evidence of ruptured umbilical cords. The cause for umbilical damage during parturition is not well understood, but uterine contractions may cause compression when the cord becomes trapped between the fetal body and maternal pelvis.^{6,7} It is perhaps reasonable to hypothesize that piglet mortality and postnatal viability may relate to the time when rupture or damage to the umbilical cord occurs. For instance, prepartum rupture would likely result in imminent death, whereas rupture in the late stages of parturition may cause nonfatal anoxia, as well as meconium defecation, staining of the skin, and meconium aspiration syndrome.

An increase in tensile stress may have also contributed to umbilical cord hemorrhage and rupture in oxytocin-treated sows. It has been suggested that the

oxytocin-induced muscle contractions during expulsion may increase pressure on umbilical cords, decrease blood flow to the fetus, and cause anoxic death.^{6,7} It is then possible that intrapartum death, at least for some of the piglets of oxytocin-treated sows, may have resulted from an increase in tensile pressure on the umbilical cords.

The highest frequency of intrapartum deaths (75%) in piglets born in the last third of the litter in the control sows coincides with 80% reported in other studies.⁸ Conversely, the highest numbers of intrapartum deaths (81 to 88%) were observed in the first third of the litter in oxytocin-treated sows.

Results of our investigation indicate that injection of oxytocin results in an increase in the incidence of meconium-stained intrapartum stillborn piglets and liveborn piglets. On the basis of information in the current literature,^{32,33} it is presumed that oxytocin increases the risk of anoxia and predisposes to meconium defecation during parturition with the concurrent staining of the skin. Development of atelectasis or hyperinflation has been shown in the lungs of pigs experimentally inoculated with meconium.^{34,35}

Results of our study also indicate that inspiratory effort and heart rate in near-death stillborn piglets were significantly lower in oxytocin-treated sows, compared with control sows. This finding is consistent with the literature suggesting depression of the respiratory centers as part of the general effects of anoxia.¹³ Obstruction of bronchi and bronchioles with meconium could also contribute to abnormal respiratory and cardiac rates.^{6,36} The effect of lower doses of oxytocin or the administration of this hormone at different stages of parturition when additional piglets have been born requires future attention.

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^bOxipar, Boehringer Ingelheim Vetmedica Inc, Guadalajara, Mexico.

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