Fetal Acidosis from Obstetric Interventions During the First Vaginal Delivery

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SUMMARY

Objective: The aim of this study was to analyze the blood gas values of umbilical cord blood in newborns of first vaginal deliveries with or without obstetric interventions.

Materials and Methods: In a prospective descriptive study conducted during the 6-month period from August 2003 through February 2004 at a university hospital, we analyzed the umbilical cord blood gas results of 80 term newborns delivered vaginally from healthy nulliparous women. Multivariate logistic analysis was used to evaluate the associations between fetal acidosis (pH < 7.20) and any obstetric interventions.

Results: The mean of umbilical cord blood arterial pH was 7.26 (standard deviation, 0.072). After controlling for the confounding factors with multivariate logistic regression, fetal acidosis (pH < 7.20) was found to be significantly associated with oxytocin augmentation (odds ratio [OR], 16.48; 95% confidence interval [CI], 1.21–226.1) and vacuum extraction (OR, 10.76; 95% CI, 1.025–112.9). In contrast, there was no significant relationship between fetal acidosis with episiotomy (OR, 1.096; 95% CI, 0.07–16.6) or epidural anesthesia (OR, 0.074; 95% CI, 0.003–2.09).

Conclusion: Oxytocin augmentation and vacuum extraction were significantly related to low cord arterial pH values (pH < 7.20), but there were no adverse effects to the newborns of first vaginal deliveries. [*Taiwan J Obstet Gynecol* 2008;47(4):397–401]

Key Words: fetal acidosis, first vaginal delivery, low cord arterial pH, obstetric interventions, umbilical blood gas analysis

Introduction

Traditionally, obstetric interventions, including episiotomy, vacuum extraction, epidural anesthesia, uterine stimulation and augmentation, are used at the time of labor and delivery. These procedures have substantial influences on the perinatal outcomes. The measurement of umbilical cord blood gas values is a better indicator of perinatal asphyxia than Apgar scores, because Apgar scoring can be influenced by prematurity, maternal sedation with opioids, aspiration of mucus or meconium [1]. The measurement of acid–base status of cord blood after delivery was suggested for intrapartum fetal surveillance by Spencer and Ward in 1993 [2]. They also recommended that cord blood gas and pH analyses should be used in neonates with low Apgar scores to distinguish metabolic acidemia from hypoxia or other causes that might result in low Apgar scores.

The aim of the study was to determine whether any obstetric interventions would cause fetal acidosis (cord arterial pH < 7.20) by measuring the umbilical cord blood pH of newborns of first vaginal deliveries.

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Materials and Methods

A total of 80 women at the time of their first vaginal deliveries were recruited at a university hospital from August 2003 through February 2004 after obtaining

informed consent that was approved by the institutional review board of the human investigation committee at the university hospital. Umbilical cord blood gas results of every term newborn delivered vaginally from these healthy nulliparous women were analyzed. The inclusion criteria were: term singleton vaginal delivery; absence of congenital fetal anomalies; mother with no previous maternal diseases (diabetes, hypertension, and known chronic diseases); absence of preeclampsia; absence of eclampsia; and absence of antepartum hemorrhage.

Data, including maternal age, gestational age, maternal height and weight, duration of second-stage labor, Apgar scores at time of delivery, and the use of oxytocin augmentation and epidural anesthesia, were collected from the patient charts. Oxytocin augmentation was performed when uterine activity did not reach 200 Montevideo units, with the cervix dilated to 4 cm or more. Oxytocin started at about 0.5 mU/min, and the dose rose every 30 minutes to a maximum of 8 mU/min or until there were three regular contractions every minute. Vacuum extraction was performed when the second stage lasted over 1 hour. Epidural anesthesia was performed upon the patient's request. All patients were constantly monitored for uterine contractions and fetal heart beats during the second stage of labor. A 10–20 cm segment of umbilical cord was clamped immediately after each delivery in this study, because delayed clamping of the umbilical cord by 20–30 seconds can alter both the P_{CO_2} and pH [3]. Separate specimens of blood (at least 1 mL) from the umbilical artery and umbilical vein in isolated umbilical cord segments were drawn into prepared plastic syringes that were rinsed with 1,000 mL of heparin solution. The needles were capped and placed into a plastic cup containing crushed ice, and sent to the laboratory immediately for analysis.

The blood samples reached the laboratory for analysis within 60 minutes. A clamped segment of cord is stable for blood gas assessment for 60 minutes after delivery at room temperature [4]. Using a Bayer Rapidlab 248 blood gas analyzer (Bayer Corp., Elkhart, IN, USA), we measured the pH, P_{CO_2}, P_{O_2} and calculated bicarbonate (HCO_3), as well as base deficit from these measurements. We defined fetal acidosis as umbilical arterial blood pH < 7.20. A second-stage labor duration of more than 2 hours was considered prolonged. Obstetric interventions included at least one or more of the following: episiotomy, oxytocin augmentation, vacuum extraction, and epidural anesthesia.

Statistical analysis was performed using SPSS version 9.0 (SPSS Inc., Chicago, IL, USA) for Windows. The sample size calculation was based on detecting a 0.03 difference between the mean umbilical arterial blood pH of the group with obstetric interventions and that of the group without obstetric interventions. The calculated number of 72 in each group, with a two-tailed α of 0.05, provided 90% power to detect these differences. Logistic regression analysis was used for multivariate analysis that was performed to evaluate the associations between fetal acidosis and any obstetric interventions. Student's *t* test was used for independent continuous variables of umbilical blood pH between the obstetric intervention group and the non-obstetric intervention group. A p value < 0.05 was considered statistically significant.

Results

The demographics and characteristics of all women are shown in Table 1. The mean umbilical arterial blood pH was 7.26 (standard deviation [SD], 0.072), P_{CO_2} was 52.9 (SD, 9.23), HCO_3 was 23.2 (SD, 2.57), and base deficit was −4.37 (SD, 3.04). The mean umbilical venous blood pH was 7.31 (SD, 0.071), P_{CO_2} was 41.65 (SD, 7.94), HCO_3 was 20.6 (SD, 2.89), and base deficit was −5.42 (SD, 3.25). The mean umbilical arterial blood pH of the non-obstetric intervention group (n = 16) was 7.289 (SD, 0.039) and that of

Table 1. Descriptive statistics for the study group (n = 80)

Variables	Mean	SD	Range
Maternal age (yr)	27.0	5.17	15–37
Maternal weight (kg)	63.7	5.88	52–76
Maternal height (cm)	158.8	4.70	152–171
Maternal BMI (kg/m^2)	25.29	2.51	21–32
Gestational age (wk)	38.5	1.33	36–41
Duration of second stage (min)	30.9	20.6	4–85
Fetal cephalic circumference (cm)	32.8	1.24	30–36
Birth fetal weight (g)	2,947	351.8	2,450–3,720

SD = standard deviation; BMI = body mass index.

the obstetric intervention group ($n=64$) was 7.253 (SD, 0.076) (Table 2). The mean pH from the two groups was analyzed using the Student's t test and revealed no statistical difference ($p=0.075$).

Sixty women underwent episiotomy and 26 women underwent vacuum extraction deliveries. Twenty-six women used oxytocin for labor augmentation, all of whom received high-dose (2–6 mU/min) therapy. Ten women underwent epidural anesthesia. All 80 women experienced a second-stage labor duration of < 2 hours. After controlling for confounding factors with multivariate logistic regression, the results showed that: oxytocin augmentation had an odds ratio (OR) of 16.48 and a 95% confidence interval (CI) of 1.21–226.1, vacuum extraction had an OR of 10.76 and a 95% CI of 1.025–112.9, episiotomy had an OR of 1.096 and a 95% CI of 0.07–16.6, and epidural anesthesia had an OR of 0.074 and a 95% CI of 0.003–2.09. Fetal acidosis (cord arterial pH < 7.20) was significantly associated with oxytocin augmentation and vacuum extraction (Table 3).

Eighteen newborns were delivered with umbilical arterial blood pH < 7.20, four of whom had fetal heart rate deceleration (< 60 beats/min) detected cardiotocographically during the second stage of labor. None of the newborns had arterial pH < 7.00. Among the 18 newborns with pH < 7.20, the lowest cord arterial pH value of 7.08 was found in one of the four newborns with heart rate deceleration. Six of the 18 newborns with umbilical arterial blood pH < 7.20 had neonatal hyperbilirubinemia and recovered uneventfully after phototherapy.

Table 2. Umbilical arterial pH values in obstetric and non-obstetric interventions

Intervention	Umbilical arterial pH, mean (SD)	p
Obstetric* ($n=64$)	7.253 (0.076)	
Non-obstetric ($n=16$)	7.289 (0.039)	0.075

**Includes at least one or more of the following: episiotomy, oxytocin augmentation, vacuum extraction, and epidural anesthesia. SD = standard deviation.*

Table 3. Multivariate logistic regression analysis of fetal acidosis (pH < 7.20)

Variable	Odds ratio	95% confidence interval
Episiotomy	1.096	0.07–16.6
Oxytocin augmentation	16.48*	1.21–226.1
Vacuum extraction	10.76*	1.025–112.9
Epidural anesthesia	0.074	0.003–2.09

**p < 0.05.*

Sixty-two of the 80 newborns had umbilical arterial blood pH > 7.20, four of whom were diagnosed with meconium aspiration syndrome (MAS). The pH of the four newborns with MAS were 7.223, 7.224, 7.235, and 7.298. None of the four infants had severe hypoxia. They received ventilatory support in an optimal thermal environment and recovered uneventfully without any complications. Six of 62 newborns with umbilical arterial blood pH > 7.20 had neonatal hyperbilirubinemia and recovered after phototherapy. No deaths, morbidities or neurologic deficits were noted in any of the infants at 3 years of follow-up.

Discussion

Fetal oxygenation and umbilical blood pH usually decline during the course of normal labor [5]. Recent reviews has conclusively determined the mean value of umbilical arterial blood pH to be greater than 7.25 for normal term newborns [6–8]. Classically, a cord arterial pH cut-off value of less than 7.20 has been used for defining pathologic fetal acidemia. Goldaber et al [9] found that most fetuses would tolerate intrapartum acidemia with a pH as low as 7.00 without neurologic impairment, and there were no deaths or neurologic dysfunction at a pH cut-off value of more than 7.00. Thus, they concluded that a more realistic pH cut-off for pathologic fetal academia would appear to be less than 7.00 [9]. As the American College of Obstetricians and Gynecologists emphasized in 1994, acidemia alone is not sufficient to lead to hypoxic brain injury unless all of the following are present: (1) umbilical artery metabolic or mixed respiratory–metabolic acidemia with pH < 7.00; (2) a persistent Apgar score of 0–3 for more than 5 minutes; (3) neonatal neurologic sequelae, such as seizures, coma or hypotonia; and (4) multiorgan system dysfunction [2]. In the labor ward of our hospital, 80 nulliparous women consented to participate in this study, and the mean umbilical arterial blood pH for newborns was 7.26 (SD, 0.072), which was similar to the results of Luttkus and co-workers [10] who found that normally delivered fetuses had pH > 7.20 or Apgar score at 5 minutes greater than 7. No newborns with pH < 7.00 were found in our study, and we did not find any neurologic deficits in the delivered newborns.

Previous publications have indicated that obstetric interventions as performed in our study may not pose any threat to the newborns [11–16]. However, no specific data on the relationship between low arterial pH (pH < 7.20) and obstetric interventions has been reported. Some studies revealed that no significant

association between umbilical arterial blood pH and fetal acidosis was found with either the use or nonuse of high-dose oxytocin augmentation [11,12]. Throp et al [11] reported that the mean arterial pH value in the no-oxytocin group was 7.24 ± 0.07, which was not significantly different from that of the oxytocin-augmented group, i.e. 7.23 ± 0.07, for term primigravid women with singleton, vertex presentations. They concluded that there were no adverse effects of high-dose oxytocin augmentation or active management of labor on umbilical cord blood gas values at delivery [11]. Morel et al [13] found that the gas evaluation of umbilical blood in 3,321 women vaginally delivering for the first time demonstrated an increase in the incidence of meconium aspiration and low cord arterial pH with the use of oxytocin augmentation in the presence of thick meconium. The findings indicated that oxytocin was safe for labor augmentation but must be carefully used in the presence of thick meconium. In the study by Vintzileos et al [14] comparing newborns delivered by elective vacuum extraction with those who underwent normal vaginal delivery, the data revealed a decline in umbilical arterial pH and an increase in umbilical arterial blood carbon dioxide, and there was also no perinatal morbidity or deaths. Bodner-Adler et al [15] reported that epidural analgesia showed a statistically significant prolonged second stage of labor ($p = 0.0001$), episiotomy ($p = 0.0001$), and labor augmented with oxytocin ($p = 0.001$); however, there were no statistically significant differences in neonatal outcomes determined by Apgar scores ($p = 0.84$) or cord pH ($p = 0.23$). Nikkola et al [16] evaluated the usefulness of intravenous patient-controlled fentanyl for labor analgesia versus epidural analgesia. They found out that all the newborns were healthy; Apgar scores and pH were normal. Neurologic scoring was similar in the patient-controlled fentanyl and epidural analgesia groups. Umbilical cord fentanyl concentrations were low or beyond the detection limits. They concluded that intravenous fentanyl with appropriate monitoring could be used for labor analgesia as an alternative to epidural analgesia [16].

We used multivariate logistic regression for controlling the confounding bias and to verify the relationship between obstetric interventions and fetal acidosis. We found that low cord arterial pH (pH < 7.20) was significantly associated with oxytocin augmentation and vacuum extraction. The newborns with pH < 7.20 in our study did not present with any neurologic deficits during hospitalization or perinatally, and no neurologic deficits were noted at 3 years of follow-up. Fetal acidosis with pH < 7.20 was an indication of low blood pH levels, but fetuses can probably tolerate this acidic condition. They did not have any neurologic sequelae unless there were other hypoxic events accompanying at the time of delivery.

Meconium in the amniotic fluid may be a pathologic explanation in response to hypoxia and signals fetal compromise. Ramin et al [17] measured umbilical artery blood gases in 7,816 term pregnancies with meconium in the amniotic fluid. They reported that 69 (1%) infants developed MAS, and 31 (45%) of them had fetal acidemia at birth. However, Blackwell et al [18] found that 60% of infants diagnosed as having MAS had umbilical artery blood pH of 7.20 or greater, suggesting that this syndrome was unrelated to the neonatal condition at delivery. Our findings of four newborn patients with MAS with cord arterial pH > 7.20 were similar to the findings of Blackwell et al. Liu and Harrington [19] identified the risk factors for MAS in newborns born through meconium-stained amniotic fluid. Twenty-four infants had respiratory symptoms consistent with MAS; 11 of the 24 infants required ventilatory support, and one required extracorporeal membrane oxygenation. They concluded that in meconium deliveries, infants with thick meconium, fetal distress and Apgar scores of < 7 at 1 and 5 minutes were at high risk for developing respiratory symptoms with MAS [17]. In our study, the most likely reason for MAS in the four infants was the presence of the thick meconium.

Kilpatrick and Laros [20] reported that the mean length of the second stage of labor for nulliparas without anesthesia was 54 minutes, and those with anesthesia was 79 minutes. In our study, we found that the mean duration of the second stage was 30.9 minutes. The reasons for the short mean length might be due to: the limited study numbers ($n = 80$), the fact that most women had normal body mass index (without obstructive outlet) and the patients only had vaginal deliveries, and non-cesarean deliveries. The longest duration of the second stage of labor in our study was 85 minutes. There were no cases of prolonged second stage (> 2 hours) in our study. Kuo et al [21] reported in 1996 that the mean of the prolonged second stage was 156 ± 28.3 minutes ($n = 165$, nulliparous = 148) and that of the normal second stage was 29.5 ± 25.4 minutes ($n = 1,750$, nulliparous = 656) for Taiwanese women. Regarding patients without prolonged second stage, the mean length in our study (30.9 ± 20.6 minutes) was similar to that reported by Kuo et al [21]. They also found that infants born after a prolonged second stage did not have an increased incidence of cord arterial pH < 7.20 [21].

In conclusion, obstetric interventions may confer risk for low cord arterial pH values, but no adverse effects to the newborns of first vaginal deliveries were

noted. Spontaneous vaginal delivery is a natural birth process and any obstetric interventions should be undertaken as sparingly as possible.

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