The umbilical cord is the fetal lifeline to the placenta. Measurements of umbilical cord blood gas values can help clinicians determine if infant compromise resulted from an asphyxial event—and, if so, whether this event was acute, prolonged, or occurred before presentation in labor.

# How and when umbilical cord gas analysis can justify your obstetric management

Summer cases illustrate how umbilical cord gas values can provide insight into a newborn's status

#### Michael G. Ross, MD, MPH

mbilical cord blood (cord) gas values can aid both in understanding the cause of an infant's acidosis and in providing reassurance that acute acidosis or asphyxia is not responsible for a compromised infant with a low Apgar score. Together with other clinical measurements (including fetal heart rate [FHR] tracings, Apgar scores, newborn nucleated red cell counts, and neonatal imaging), cord gas analysis can be remarkably helpful in determining the cause for a depressed newborn. It can help us determine, for example, if infant compromise was a result of an asphyxial event, and we often can differentiate whether the event was acute, prolonged, or occurred prior to presentation in labor. We further can use cord gas values to assess whether a decision for operative intervention for nonreassuring fetal well-being was appropriate (see "Brain injury at birth: Cord gas values presented as



Dr. Ross is Distinguished Professor of Obstetrics and Gynecology and Public Health, Geffen School of Medicine at UCLA, Fielding School of Public Health at UCLA, Department of Obstetrics and Gynecology, Harbor/UCLA Medical Center, Torrance, California.

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evidence at trial" on page 42). In addition, cord gas analysis can complement methods for determining fetal acidosis changes during labor, enabling improved assessment of FHR tracings.<sup>1–3</sup>

I recommend checking umbilical cord blood gas values on all operative vaginal deliveries, cesarean deliveries for fetal concern, abnormal FHR patterns, clinical chorioamnionitis, multifetal gestations, premature deliveries, and all infants with low Apgar scores at 1 or 5 minutes. If you think you may need a cord gas analysis, go ahead and obtain it. Cord gas analysis often will aid in justifying your management or provide insight into the infant's status.

Controversy remains as to the benefit of universal cord gas analysis. Assuming a variable cost of \$15 for 2 (artery and vein) blood gas samples per neonate,<sup>4</sup> the annual cost in the United States would be approximately \$60 million. This would likely be cost effective as a result of medicolegal and educational benefits as well as potential improvements in perinatal outcome<sup>5</sup> and reductions in special care nursery admissions.<sup>4</sup>

**CASE1** A newborn with unexpected acidosis A 29-year-old woman (G2P1) at 38 weeks' gestation was admitted to the hospital following an office visit during which oligohydramnios



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	Umbilical artery	Umbilical vein
рН	7.18–7.38	7.25–7.45
Po2	5.6–30.4 mm Hg	17.4–41.0 mm Hg

32.4-66.0 mm Hg

4.79 (3.46) mmol/L

TABLE 1 Normal values for fetal umbilical cord gases<sup>6,7</sup>

Ranges based on mean ±2 SD.

BD<sub>ECF</sub>, mean (SD)

Pco,

(amniotic fluid index, 3.5 cm) was found. The patient had a history of a prior cesarean delivery for failure to progress, and she desired a repeat cesarean delivery. Fetal monitoring revealed a heart rate of 140 beats per minute with moderate variability and uterine contractions every 3 to 5 minutes associated with moderate variable decelerations. A decision was made to proceed with the surgery. Blood samples were drawn for laboratory analysis, monitoring was discontinued, and the patient was taken to the operating room. An epidural anesthetic was placed and the cesarean delivery proceeded.

27.0–49.4 mm Hg

~4.0 (3.5) mmol/L

On uterine incision, there was no evidence of abruption or uterine rupture, but thick meconium-stained amniotic fluid was observed. A depressed infant was delivered, the umbilical cord clamped, and the infant handed to the pediatric team. Cord samples were obtained and values from the umbilical artery were as follows: pH, 6.80;  $PCO_2$ , 120 mm Hg;  $PO_2$ , 6 mm Hg; and base deficit extracellular fluid ( $BD_{ECF}$ ), 13.8 mmol/L. Values from the umbilical vein were: pH, 7.32;  $PCO_2$ , 38 mm Hg;  $PO_2$ , 22 mm Hg; and  $BD_{ECF}$ , 5.8 mmol/L. The infant's Apgar scores were 1, 2, and 7 at 1, 5, and 10 minutes, respectively, and the infant demonstrated encephalopathy, requiring brain cooling.

What happened?

# Using cord gas values in practice

Before analyzing the circumstances in Case 1, it is important to consider several key questions, including:

- What are the normal levels of cord pH, O<sub>2</sub>, CO<sub>2</sub>, and base deficit (BD)?
- How does cord gas indicate what happened during labor?

• What are the preventable errors in cord gas sampling or interpretation?

For a review of fetal cord gas physiology, see "Physiology of fetal cord gases: The basics," on page 44.

**Normal values: The "20, 30, 40, 50 rule"** Among the values reported for umbilical blood gas, the pH,  $PCO_2$ , and  $PO_2$  are measured, whereas BD is calculated. The normal values for umbilical pH and blood gases are often included with laboratory results, although typically with a broad, overlapping range of values that may make it difficult to determine which is umbilical artery or vein (TABLE 1).<sup>67</sup>

I recommend using the "20, 30, 40, 50 rule" as a simple tool for remembering normal umbilical artery and vein  $PO_2$  and  $PCO_2$  values (TABLE 2):

- Po<sub>2</sub> values are lower than Pco<sub>2</sub> values; thus, the 20 and 30 represent Po<sub>2</sub> values
- as fetal umbilical artery Po<sub>2</sub> is lower than umbilical vein Po<sub>2</sub>, 20 mm Hg represents the umbilical artery and 30 mm Hg represents the vein
- PCO<sub>2</sub> values are higher in the umbilical artery than in the vein; thus, 50 mm Hg represents the umbilical artery and 40 mm Hg represents the umbilical vein.

Umbilical cord BD values change in relation to labor and FHR decelerations.<sup>8</sup> Prior to labor, the normal fetus has a slight degree of acidosis (BD, 2 mmol/L). During the latent phase of labor, fetal BD typically does not change. With the increased frequency of contractions, BD may increase 1 mmol/L for every 3 to 6 hours during the active phase and up to 1 mmol/L per hour during the second stage, depending on FHR responses. Thus,

# TABLE 2Fetal umbilical cord gases:The "20, 30, 40, 50 rule"

Value, mm Hg	Cord gas measured
20	Umbilical artery PO <sub>2</sub>
30	Umbilical vein PO <sub>2</sub>
40	Umbilical vein PCO <sub>2</sub>
50	Umbilical artery PCO <sub>2</sub>
	1



The pH,  $PCO_2$ , and  $PO_2$  in cord blood are measured, while BD is calculated

following vaginal delivery the average umbilical artery BD is approximately 5 mmol/L and the umbilical vein BD is approximately 4 mmol/L. As lactate crosses the placenta slowly, BD values are typically only 1 mmol/L less in the umbilical vein than in the artery, unless there has been an obstruction to placental flow (see Case 1).

For pH, the umbilical artery value is always lower than that of the vein, a result of both the higher umbilical artery  $PCO_2$  as well as the slightly higher levels of lactic acid before placental clearance. Fetal pH levels typically decrease during labor associated with the increased BD described above. However, short-term effects of increased  $CO_2$  (respiratory acidosis) or  $CO_2$  clearance may cause fluctuations in pH that do not correlate with the degree of metabolic acidosis.

## Possible causes of abnormal cord gas values

Because of the nearly fully saturated maternal hemoglobin under normal conditions, fetal arterial and venous Po, levels cannot be increased significantly above normal values. However, *reduced* fetal  $PO_2$  and *increased* fetal  $PCO_2$  may occur with poor gas exchange between the maternal and fetal compartments (eg, placental abruption) or maternal respiratory compromise.

In contrast, reduced fetal Pco<sub>2</sub> may occur under conditions of maternal hyperventilation and lower maternal Pco<sub>2</sub> values. Decreased pH levels may be due to respiratory or metabolic acidosis, the former of which is generally benign. Elevated BD typically is a result of fetal metabolic acidosis, and values approaching 12 mmol/L should be avoided, if possible, as this level may be associated with newborn neurologic injury.<sup>9</sup>

#### Effect of maternal oxygen administration on fetal oxygenation

Although maternal oxygen administration is commonly used during labor and delivery, controversy remains as to the benefit of oxygen supplementation.<sup>10</sup> In a normal mother with oxygen saturation above 95%,

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### Brain injury at birth: Cord gas values presented as evidence at trial

At 40 weeks' gestation, a woman presented to the hospital because of decreased fetal movement. On arrival, an external fetal heart-rate (FHR) monitor showed nonreassuring tracings, evidenced by absent to minimal variability and subtle decelerations occurring at 10- to 15-minute intervals. The on-call ObGyn requested induction of labor with oxytocin, and a lowdose infusion (1 mU/min) was initiated. An internal FHR monitor was then placed and late decelerations were observed with the first 2 induced contractions. The oxytocin infusion was discontinued and the ObGyn performed an emergency cesarean delivery. The infant's Apgar scores were 1, 2, and 2 at 1, 5, and 10 minutes, respectively. Cord samples were obtained and values from the umbilical artery were as follows: pH, 6.86; PCO<sub>2</sub>, 55 mm Hg; PO<sub>2</sub>, 6 mm Hg; and BD<sub>ECE</sub>, 21.1 mmol/L. Values from the umbilical vein were: pH, 6.94; PCO<sub>2</sub>, 45 mm Hg; PO<sub>2</sub>, 17 mm Hg; and BD<sub>ECF</sub>, 20.0 mmol/L. The infant was later diagnosed with a hypoxic brain injury resulting in cerebral palsy. At trial years later, the boy had cognitive and physical limitations and required 24-hour care.

The parents claimed that the ObGyn should have performed a cesarean delivery earlier when the external FHR monitor showed nonreassuring tracings.

The hospital and physician claimed that, while tracings were consistently nonreassuring, they were stable. They maintained that the child's brain damage was not due to a delivery delay, as the severe level of acidosis in both the umbilical artery and vein could not be a result of the few heart rate decelerations during the 2-hour period of monitoring prior to delivery. They argued that the clinical picture indicated a pre-hospital hypoxic event associated with decreased fetal movement.

A defense verdict was returned.

#### Case assessment

Cord gas results, together with other measures (eg, infant nucleated red blood cells, brain imaging) can aid the ObGyn in medicolegal cases. However, they are not always protective of adverse judgment.

**TRACK** While maternal oxygen supplementation for normal mothers may have nominal benefit for compromised fetuses, it may have

marked benefit when

maternal Po, is low

FAST

the administration of oxygen will increase maternal arterial  $PO_2$  levels and thus *dissolved* oxygen. Because maternal hemoglobin is normally almost fully saturated at room air  $PO_2$  levels, there is little change in the *bound* oxygen and thus little change in the maternal arterial  $O_2$  content or maternal uterine venous  $PO_2$  levels. As fetal umbilical vein  $PO_2$  levels equilibrate to maternal uterine vein  $PO_2$  levels, there is minimal change in fetal oxygenation.

However, maternal oxygen supplementation may have marked benefit in cases in which maternal arterial  $Po_2$  is low (respiratory compromise). In this case, the steep fetal oxygen saturation curve may produce a large increase in fetal umbilical vein oxygen content. Thus, **strongly consider oxygen supplementation for mothers with impaired cardiorespiratory function**, and recognize that maternal oxygen supplementation for normal mothers may result in nominal benefit for compromised fetuses.

### How did the Case 1 circumstances lead to newborn acidosis?

Most noticeable in this case is the large difference in BD between the umbilical artery and vein and the high  $PCo_2$  in the artery. Under conditions without interruption of fetal placental flow, either the umbilical artery and/or vein will provide a similar assessment of fetal or newborn metabolic acidosis (that is, BD).

Whereas BD normally is only about 1 mmol/L greater in the umbilical artery versus in the vein, occasionally the arterial value is markedly greater than the vein value. This can occur when there is a cessation of blood flow through the placenta, as a result of complete umbilical cord obstruction, or when there is a uterine abruption. In these situations, the umbilical vein (which has not had blood flow) represents the fetal status prior to the occlusion event. In contrast, despite bradycardia, fetal heart pulsations mix blood within the umbilical artery and therefore the artery generally represents the fetal status at the time of birth. In response to complete cord occlusion, fetal BD increases by approximately 1 mmol/L every 2 minutes. Consequently, an 8 mmol/L difference in BD between the umbilical artery and vein is consistent with a 16-minute period of umbilical occlusion or placental abruption. Also in response to complete umbilical cord occlusion, PCO<sub>2</sub> values rise by approximately 7 mm Hg per minute of the occlusion, although this may not be linear at higher levels. Thus, **the BD difference suggests there was likely a complete cord occlusion for the 16 minutes prior to birth.** 

The umbilical vein BD is also elevated for early labor. This value suggests that repetitive, intermittent cord occlusions (evident on the initial fetal monitor tracing) likely resulted in this moderate acidosis prior to the complete cord occlusion in the final 16 minutes.

Thus, BD and  $Pco_2$  levels can be used to time the onset of umbilical cord occlusion or abruption. Since pH is an inverse logarithmic function, it cannot be used to time the onset or duration of cord occlusion. Remember that BD values should be adjusted for extracellular fluid under conditions of markedly elevated Pco<sub>2</sub>.

### CASE 2 An infant with unusual umbilical artery values

An infant born via vacuum delivery for a prolonged second stage of labor had 1- and 5-minute Apgar scores of 8 and 9, respectively. Cord gas values were obtained, and analysis revealed that for the umbilical artery, the pH was 7.29;  $PCO_2$ , 20 mm Hg; and  $PO_2$ , 60 mm Hg. For the umbilical vein, the pH was 7.32;  $PCO_2$ , 38 mm Hg; and  $PO_2$ , 22 mm Hg.

The resident asked, "How is the  $PO_2$  higher in the artery than in the vein?"

## The curious Case 2 values suggest an air bubble

Although it is possible that the aberrant values in Case 2 could have resulted from switching the artery and vein samples, the pH is lower in the artery, and both the artery  $Po_2$  and  $PCO_2$  levels do not appear physiologic. The likely explanation for these values is that an air bubble was contained in the syringe.

Since normal room air  $(21\% O_2)$  has a PO<sub>2</sub> of 159 mm Hg and a PCO<sub>2</sub> of less than 1 mm Hg, exposure of cord blood gases to air bubbles will significantly increase the PO<sub>2</sub> and markedly reduce the PCO<sub>2</sub> values of the sample. Take care to avoid air bubbles in the syringes used to obtain samples for analysis.

### CASE 3 A vigorous baby with significant acidosis

A baby with 1- and 5-minute Apgar scores of 9 and 9 was delivered by cesarean and remained vigorous. Umbilical cord analysis revealed an umbilical artery pH level of 7.15, with normal  $Po_2$ and  $Pco_2$  values. What could be the explanation?

#### Was there a collection error in Case 3?

On occasion, a falsely low pH level and, thus, a falsely elevated BD may result from excessive heparin in the collection syringe. Heparin is acidotic and should be used only to coat the syringe. Although syringes in current use are often pre-heparinized, if one is drawing up heparin into the syringe, it should be coated and then fully expelled.

#### Umbilical cord sampling: Procedures and equipment

Many issues remain regarding the optimal storage of cord samples. Ideally, a doubly clamped section of the cord promptly should be sampled into glass syringes that can be placed on ice and rapidly measured for cord values.

Stability of umbilical cord samples within the cord is within 20 to 30 minutes. Delayed sampling of clamped cord sections generally has minimal effect on pH and Pco, values.11 The BD does not change to a clinically significant degree over 15 to 30 minutes despite the cord specimen remaining at room temperature. However, one report demonstrated an increase in lactate and BD by 20 minutes under these conditions: this likely was a result of metabolism from endothelial or blood cells.12 I therefore recommend that clamped cord be sampled as soon as is feasible and ideally not beyond 20 to 30 minutes. Plastic syringes can introduce interference. Several studies have demonstrated



BD and  $PCO_2$ levels can be used to time the onset of umbilical cord occulsion or abruption

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### Physiology of fetal cord gases: The basics

A review of basic fetal cord gas physiology will assist in understanding how values are interpreted.

#### Umbilical cord O, and CO,

Fetal cord gas values result from the rapid transfer of gases and the slow clearance of acid across the placenta. Approximately 10% of maternal blood flow supplies the uteroplacental circulation, with the nearterm placenta receiving approximately 70% of the uterine blood flow.1 Of the oxygen delivered, a surprising 50% provides for placental metabolism and 50% for the fetus. On the fetal side, 40% of fetal cardiac output supplies the umbilical circulation. Oxygen and carbon dioxide pass readily across the placental layers; exchange is limited by the amount of blood flow on both the maternal and the fetal side (flow limited). In the human placenta, maternal blood and fetal blood effectively travel in the same direction (concurrent exchange); thus, umbilical vein O<sub>2</sub> and CO<sub>2</sub> equilibrate with that in the maternal uterine vein.

Most of the  $O_2$  in fetal blood is carried by hemoglobin. Because of the markedly greater affinity of fetal hemoglobin for  $O_2$ , the saturation curve is shifted to the left, resulting in increased hemoglobin saturation at the relatively low levels of fetal  $PO_2$ . This greater affinity for oxygen results from the unique fetal hemoglobin gamma ( $\gamma$ ) subunit, as compared with the adult beta (B) subunit. Fetal hemoglobin has a reduced interaction with 2,3-bisphosphoglycerate, which itself decreases the affinity of adult hemoglobin for oxygen.

The majority of  $CO_2$  (85%) is carried as part of the bicarbonate buffer system. Fetal  $CO_2$  is converted into carbonic acid  $(H_2CO_3)$  in the red cell and dissociates into hydrogen (H<sup>+</sup>) and bicarbonate (HCO<sub>3</sub><sup>-</sup>) ions, which diffuse out of the cell. When fetal blood reaches the placenta, this process is reversed and  $CO_2$  diffuses across the placenta to the maternal circulation. The production of H<sup>+</sup> ions from  $CO_2$  explains the development of *respiratory* acidosis from high PCO<sub>2</sub>. In contrast, anaerobic metabolism, which produces lactic acid, results in *metabolic* acidosis.

#### Difference between pH and BD

The pH is calculated as the inverse log of the H<sup>+</sup> ion concentration; thus, the pH falls as the H<sup>+</sup> ion concentration *exponentially* increases, whether due to respiratory or metabolic acidosis. To quantify the more important metabolic acidosis, we use BD, which is a measure of how much of bicarbonate buffer base has been used by (lactic) acid. The BD and the base excess (BE) may be used interchangeably, with BE representing a negative number. Although BD represents the metabolic component of acidosis, a correction may be required to account for high levels of fetal Pco, (see Case 1). In this situation, a more accurate measure is BD extracellular fluid (BD<sub>ECE</sub>).

Why not just use pH? There are 2 major limitations to using pH as a measure of fetal or newborn acidosis. First, pH may be influenced by both respiratory and metabolic alterations, although only metabolic acidosis is associated with fetal neurologic injury.<sup>2</sup> Furthermore, as pH is a log function, it does not change linearly with the amount of acid produced. In contrast to pH, BD is a measure of metabolic acidosis and changes in direct proportion to fetal acid production.

What about lactate? Measurements of lactate may also be included in blood gas analyses. Under hypoxic conditions, excess pyruvate is converted into lactate and released from the cell along with H<sup>+</sup>, resulting in acidosis. However, levels of umbilical cord lactate associated with neonatal hypoxic injury have not been established to the same degree as have pH or BD. Nevertheless, lactate has been measured in fetal scalp blood samples and offers the potential as a marker of fetal hypoxemia and acidosis.<sup>3</sup>

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Because maternal blood and fetal blood effectively travel in the same direction in the placenta, umbilical vein  $O_2$  and  $CO_2$  equilibrate with that in the maternal uterine vein that collection of samples in plastic may result in an increase in  $Po_2$  values, likely due to the high room air  $Po_2$  diffusing through the plastic to the blood sample.

**Use glass, and "ice" the sample if necessary.** Although it has been suggested that placing samples on ice minimizes metabolism, the cooled plastic may in fact be more susceptible to oxygen diffusion. Thus, unless samples will be analyzed promptly, it is best to use glass syringes on ice.<sup>13,14</sup>

#### What if the umbilical cord is torn?

Sometimes the umbilical cord is torn and discarded or cannot be accessed for other reasons. A sample can still be obtained, however, by aspirating the placental surface artery and vein vessels. Although there is some potential variance in pH, Po<sub>2</sub>, and Pco<sub>2</sub> levels, the BD values of placental vessels have a high correlation with those of umbilical vessels

and therefore can be used when the cord is not available.<sup>15</sup>

## How do you obtain cord analysis when delaying cord clamping?

The American College of Obstetricians and Gynecologists (ACOG) now advises delayed cord clamping in term and preterm deliveries, which raises the question of how you obtain a blood sample in this setting. Importantly, ACOG recommends delayed cord clamping only in vigorous infants,16 whereas potentially compromised infants should be transferred rapidly for newborn care. Although several studies have demonstrated some variation in cord gas values with delayed cord clamping,<sup>17-21</sup> clamping after pulsation has ceased or after the recommended 30 to 60 seconds following birth results in minimal change in BD values. Thus, do not hesitate to perform delayed cord clamping in vigorous infants. @

### FAST TRACK

If the umbilical cord is unavailable, blood samples for gas analysis can be obtained from placental surface vessels

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