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Interpreting Umbilical Cord Blood Gases: Cord Occlusion with Terminal Fetal Bradycardia: Part II

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Case 11: Umbilical Cord Occlusion with Terminal Fetal Bradycardia, Severe

From this point forward in these installments, all base deficits are extracellular fluid base deficits, and for consistency, all have been calculated using the equations supplied by CSLI (Clinical Laboratories Standard Institute). If your hospital already uses the CSLI equations, no adjustment is necessary.

$$BD(ecf) = -(HCO3^- - 24.8 + 16.2 \times (pH - 7.4))$$

$$Log(HCO3^-) = pH + Log(Pco_2) - 7.608$$

$$HCO3^- = 10^{(Log\ (HCO3-))}$$

Much earlier in my life, I could have handled these equations. Today I would not even try. However, my computer spits out the results without a moment of hesitation. If you choose to, you can use your computer in the same way.

The mother was a 39-year-old, gravida 2, para 1, aborta 0, with an intrauterine pregnancy at 38 0/7 weeks' gestation. Membranes ruptured spontaneously with egress of clear fluid. There were mild uterine contractions. On external monitor, the FHR was approximately 140 bpm with moderate variability. Pitocin was begun because of failure to progress. Deeper and more prolonged variable decelerations ensued and Pitocin was discontinued. Terbutaline was administered with initially good recovery. Shortly thereafter, the FHR suddenly fell into the 60s and an emergency cesarean delivery was performed. At delivery, the male infant weighing 2951 g had a single tight loop of umbilical cord encircling his neck. The cord was clamped, cut and removed from around the neck. Resuscitation included suctioning, stimulation, and very brief bag-mask positive pressure ventilation with 100% oxygen. Apgar scores were 3 and 9 at one and five minutes, respectively.

Cord blood gas results were as follows:

	Umbilical Vein	Umbilical Artery
pH	7.20	6.96
Pco_2 (mmHg) *(kPa)*	54 *7.20*	104 *13.87*
Po_2 (mmHg) *(kPa)*	35 *4.67*	35 *4.67*
BD (mmol/L)	7	9

At 20 minutes of age, an arterial blood gas in room air was:

	Infant's ABG
pH	7.27
Pco_2 (mmHg) *(kPa)*	56 *7.47*
Po_2 (mmHg) *(kPa)*	81 *10.80*
BD (mmol/L)	1

A central hematocrit was 58%; when repeated one hour later, it was 50%.

"The umbilical venous blood sample demonstrates a mild respiratory acidosis. The base deficit of seven is in the normal range. The umbilical arterial blood sample, on the other hand, is quite abnormal. The pH is severely depressed on the basis of a severe respiratory acidosis and a mild metabolic acidosis."

Interpretation

The umbilical venous blood sample demonstrates a mild respiratory acidosis. The base deficit of seven is in the normal range. The umbilical arterial blood sample, on the other hand, is quite abnormal. The pH is severely depressed on the basis of a severe respiratory acidosis and a mild metabolic acidosis. The Po_2 of 35 is high, suggesting contamination with an air bubble. Some blood gas analyzers no longer report results if they detect air bubbles. This is further supported by the fact that the Po_2 in the arterial sample is not lower than the Po_2 in the venous sample, thus breaking one of the "rules" of the relationship between these two sample sites. However, it is very clear from the large differences between the venous and arterial samples that both a vein and an artery are represented. If this sample was contaminated with an air bubble, as I think it was, the "true" Pco_2 would have been even higher and the "true" pH even lower.

The differences between the pH and Pco_2 values are very wide indeed. This finding supports the diagnosis of umbilical cord occlusion with terminal fetal bradycardia. The widened pH and Pco_2 differences are the result of partial restoration of umbilical arterial blood flow over a significant period of time. The umbilical venous pH is almost normal while the umbilical arterial pH is very low. Extreme differences between venous and arterial cord blood pH that are associated with variable decelerations and cord compression are not a new discovery; this observation dates back to at least 1977. (1) Umbilical venoarterial Pco_2 differences of greater than 25 mmHg in infants with an umbilical artery pH of less than 7.00 are associated with an increased incidence of neonatal seizures, hypoxic-ischemic encephalopathy, cardiopulmonary dysfunction, renal dysfunction, and abnormal development at discharge. (2)

Infants with evidence of cord compression and terminal bradycardia will become significantly hypovolemic (3) and anemic (4) With the umbilical vein occluded and restored umbilical artery blood flow, an excess of blood accumulates in the placenta, unavailable to the fetus. Following delivery, as the fetus either spontaneously reconstitutes a normal blood volume or the newborn is given volume expansion, the hematocrit will fall. In the infant described above, the central hematocrit fell from 58% to 50% over the first 80 minutes of life; complete equilibration may take longer. This suggests a loss of approximately 14% of the fetal blood volume into the placenta (50/58 = 0.86). This is not unexpected, as cord compression has been observed to result in an increase in villous blood volume, sometimes by more than 50%. (5)

The follow-up ABG at 20 minutes of age, taken directly from the infant, has normalized. This is a little surprising as many infants will have a worsened base deficit when retested shortly after delivery. However, considering that this infant was quite vigorous by five minutes of age, and had only a moderate metabolic acidosis in the umbilical artery sample, quick recovery may well have occurred. An infant with normal cardiopulmonary function can quickly metabolize lactic acid to bicarbonate.

Mercer et al (6) have suggested the possibility that milking of the umbilical cord before clamping may be an effective way to remedy fetal/neonatal hypovolemia, especially if resuscitation can be accomplished at the perineum. Their suggestion was specifically aimed at therapy for newborns following shoulder dystocia with vaginal delivery (see Case 17 in an upcoming installment), but the concept seems applicable to other newborns with significant hypovolemia from all causes. Hosono et al (7) have investigated milking the umbilical cord in premature infants. Hemoglobin was higher in the milked group by 2.4 g/dL. This procedure might well obscure the interpretation of the cord gas results; however, optimizing care of the newborn is everyone's first priority.

"Studies in this area are warranted. Improved communication between the obstetric and neonatal teams would be essential as a history of a vulnerable cord followed by a prolonged terminal bradycardia should raise suspicion of significant fetal/neonatal hypovolemia."

Although this particular infant was probably not depressed enough to require such therapy, other infants might be (see Cases 13-16 and 18 in future installments). Studies in this area are warranted. Improved communication between the obstetric and neonatal teams would be essential as a history of a vulnerable cord followed by a prolonged terminal bradycardia should raise suspicion of significant fetal/neonatal hypovolemia.

Key Points

- When umbilical cord occlusion with restoration of umbilical artery blood flow is prolonged, differences between umbilical venous and arterial blood gas values may be extreme.
- Cord occlusion with termination of both venous and arterial blood flow, but with temporary partial restoration of umbilical arterial blood flow (as typically occurs with cord occlusion and terminal fetal bradycardia), results in a net transfer of blood into the placenta. The greater the umbilical venoarterial pH difference, the more time in which umbilical artery blood flow continued in the absence of umbilical venous return. Also, the more likely the resultant fetal hypovolemia and subsequent neonatal anemia will be critical.

Case 12: True Knot in Cord

The mother was a 30-year-old, gravida 2, para 0, aborta 1 with an intrauterine pregnancy at 39 6/7 weeks' gestation. The pregnancy had been uneventful. Fetal activity was reported as normal and unchanged. The initial FHR was 140-150 bpm and had occasional accelerations. As labor progressed, variable decelerations appeared and became increasingly severe over time. Baseline beat-to-beat variability, originally moderate (normal), diminished and became absent. Seventeen hours after admission, a severe fetal bradycardia precipitated an emergency cesarean delivery under general anesthesia. At delivery, a tight true knot in the umbilical cord was found. Apgar scores were 1, 3 and 5 at one, five and 10 minutes, respectively. The infant was floppy and unresponsive. Resuscitation included positive pressure ventilation and intubation. Birth weight was 3370 g. The placenta weighed 624 g and revealed no histiologic abnormalities.

Cord blood gas results were as follows:

	Umbilical Vein	**Umbilical Artery**
pH	7.31	6.85
Pco_2 (mmHg) *(kPa)*	44 *5.87*	116 *15.47*
Po_2 (mmHg) *(kPa)*	33 *4.40*	15 *2.00*
BD (mmol/L)	4	13

A CBG blood gas at age 45 minutes was as follows:

	CBG
pH	7.00
Pco_2 (mmHg) *(kPa)*	67 *8.93*
Po_2 (mmHg) *(kPa)*	45 *6.00*
BD (mmol/L)	15

Bicarbonate and normal saline were administered IV. The infant remained floppy and non-responsive at 32 minutes of age. A WBC count and differential were as follows: 26.0K/mm^3 with 10% bands and 36% neutrophils. The hematocrit and platelet counts were 50% and 148K, respectively. Ampicillin and cefotaxime were started and discontinued after four days when the blood culture was reported as negative. A CT of the head at age two days was normal and no seizures were noted at any time during the six days of hospitalization. Kidney and liver function tests results were normal.

Interpretation

The umbilical venous blood sample is normal. The umbilical arterial blood sample is very abnormal. The pH is severely depressed on the basis of a combined severe respiratory acidosis and a moderate metabolic acidosis. Every clinician should be aware of the type of base deficit reported at their hospital. Extracellular fluid base deficit should be encouraged. The umbilical pH, Pco_2 and base deficit venoarterial differences are very wide. The follow-up blood gas demonstrates a much improved Pco_2 and a marginally higher base deficit. Most commonly, newborns who have markedly depressed one-minute Apgar scores, have some degree of acid washout (increased base deficit) on their first follow-up blood gas. However, by age 45 minutes the base deficit already may have improved from what it was earlier. It should be noted that in the face of poor perfusion, a CBG may be misleading. The inaccuracy tends to make the base deficit appear worse.

" The most likely pathophysiology is as follows: Initially, in terminal cord occlusion, both the umbilical vein and the umbilical arteries are occluded. Usually, however, the blood flow in the umbilical arteries is restored temporarily due to increasing fetal blood pressure."

There is nothing that distinguishes this set of cord gases from others with cord compression and terminal fetal bradycardia already presented. What is noteworthy is the cause of the abnormalities in blood gas values, namely the true knot in the cord. True knots in the umbilical cord have long been known as an occasional cause of stillbirth, but seldom seem to cause a problem during labor and delivery. (8) Although the placenta was said to have shown no histological abnormalities, one would expect to see a dilated umbilical vein on the placental side of the knot. Perhaps the knot in the cord was untied in the delivery room.

"A true knot has little impetus to tighten during labor and delivery unless the cord is anatomically critically short or made functionally short by being wrapped around a fetal body part."

Although the diagnosis seems straightforward, it is always possible that an unidentified occult cord prolapse caused the cord occlusion rather than the true knot in the cord. A true knot has little impetus to tighten during labor and delivery unless the cord is anatomically critically short or made functionally short by being wrapped around a fetal body part.

Key Point

- Although rare, a true knot in the umbilical cord may result in the classical cord blood gas findings associated with cord occlusion and terminal bradycardia, namely widened differences between umbilical venous and arterial pH, Pco_2 and base deficit.

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