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Interpreting Umbilical Cord Blood Gases: Section 9: Deciphering Multiple Issues

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Case 26: Cord Occlusion with Release Just Before Delivery

The mother was a 27-year-old, 226 lb, gravida 3, para 2, aborta 0, with an intrauterine pregnancy at 40 5/7 weeks gestation. The mother had two previous difficult vaginal deliveries. One required vacuum extraction (birth weight 3034 g) and the other suprapubic pressure for shoulder dystocia (birth weight 3289 g). One day before admission, the mother complained of decreased fetal activity; however, an NST was reactive. On admission, the FHR tracing had a baseline heart rate of 145 bpm with good variability. Over the next six hours, both late and variable decelerations occurred with increasing frequency.

"Thirty-five minutes before delivery, Tucker forceps were applied. The FHR declined slowly from 180 to 40 bpm. This was followed by a rapid increase to 140, a rapid decrease to 75, an unstable heart rate between 75 and 130, a return to 180, and finally, a deceleration to 80 bpm without variability."

Thirty-five minutes before delivery, Tucker forceps were applied. The FHR declined slowly from 180 to 40 bpm. This was followed by a rapid increase to 140, a rapid decrease to 75, an unstable heart rate between 75 and 130, a return to 180, and finally, a deceleration to 80 bpm without variability. Further attempts at vaginal delivery were abandoned, and an emergency cesarean section was ordered. Twenty-five minutes later, the infant was delivered. The FHR was 145 bpm three minutes before delivery, still without variability. Apgar scores were 2, 4, and 4 at one, five, and 10 minutes, respectively.

Cord blood gas results were as follows:

	Umbilical Vein	**Umbilical Artery**
pH	7.00	6.75
Pco_2 (mmHg) ***(kPa)***	71 *9.47*	132 *17.60*
Po_2 (mmHg) ***(kPa)***	35 *4.67*	5 *0.67*
BD (mmol/L)	14	17

At delivery, the umbilical cord was wrapped tightly around the shoulders "like suspenders." Thick meconium was present. Resuscitation included intubation, suctioning, positive pressure ventilation, and oxygen. Oxygen was increased to 100% when the baby was slow to respond. The Birth weight was 3318 g.

Follow-up arterial blood gases from the infant at 37 minutes of age were:

	Infant's ABG
pH	7.13
Pco_2 (mmHg) ***(kPa)***	37 *4.93*
Po_2 (mmHg) ***(kPa)***	48 *6.40*
BD (mmol/L)	17

A CBC at age 42 minutes had a hematocrit of 49.3% and a WBC count that was mildly elevated with a shift to the left. A follow-up WBC count at age 18 hours was normal; the hematocrit was 47.6%. The blood culture was negative.

The infant was found to have a right non-depressed parietal skull fracture, developed seizures that resolved, and had an MRI that was compatible with a hypoxic-ischemic event. Intracranial hemorrhage was absent. Subsequently, the infant was found to have cerebral palsy, spastic quadriparesis, and moderate to severe

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Interpretation

The umbilical venous gas has a low pH secondary to severe respiratory acidosis and moderate to severe metabolic acidosis. The umbilical arterial blood gas has a very low pH secondary to a combination of very severe respiratory acidosis and severe metabolic acidosis. The differences between the umbilical venous and arterial values are quite wide; the pH difference is 0.25 (7.00 minus 6.75), the Pco_2 difference is 61 mmHg (132 minus 71) (kPa difference of 8.13), and the base deficit difference is 3 mmol/L (17 versus 14).

In uteroplacental insufficiency, typically, both the umbilical venous and the umbilical arterial blood gas values are about equally abnormal. In umbilical cord occlusion with terminal fetal bradycardia, typically, the umbilical venous blood gas is normal or nearly so. Additionally, the differences between umbilical venous and arterial pH, Pco_2, and sometimes base deficit are widened.

In this case, the umbilical venous gas is far from normal, *and* the veno-arterial differences are marked, suggesting the possibility of initial uteroplacental insufficiency, followed by umbilical vein occlusion as the functionally shortened cord (umbilical cord wrapped tightly around the shoulders "like suspenders") was stretched during fetal descent.

"How, then, to account for the combined moderate venous respiratory and severe metabolic acidoses? Close to the time of delivery, there was probably a period in which the cord occlusion was released, and acid that had accumulated on the arterial side began to be transferred to the umbilical venous side before the cord was again occluded by clamping at birth."

Against the argument of uteroplacental insufficiency playing a role in the umbilical cord blood gas outcome is the umbilical venous Po_2 of 35 mmHg, a value too high to be associated with uteroplacental insufficiency but frequently associated with cord occlusion and terminal bradycardia with a brief preceding period of slowed umbilical venous blood flow allowing for improved downloading of oxygen from the mother to the fetus. (1) How, then, to account for the combined moderate venous respiratory and severe metabolic acidoses? Close to the time of delivery, there was probably a period in which the cord occlusion was released, and acid that had accumulated on the arterial side began to be transferred to the umbilical venous side before the cord was again occluded by clamping at birth. The most likely scenario would appear to be as follows: initial complete cord occlusion, reactive fetal hypertension overcoming occlusive forces with the restoration of umbilical arterial blood flow, and finally, restoration of umbilical venous blood flow with mixing of umbilical arterial blood (that has just passed through chorionic villi) and sequestered placental blood. This would result in umbilical venous cord gases that are no longer normal or near-normal but not as abnormal as the umbilical arterial sample. This theory is appealing as three minutes before delivery, the recorded FHR was 145 bpm with absent variability, and the heart rate at birth was over 100 bpm. Unless the mother was critically ill or had received an atropine-like medication, the poor variability on the fetal heart rate tracing precludes the possibility of the heart rate being maternal. What caused the occlusion to be relieved is unknown, but unless the occlusion were released, the FHR would have remained severely depressed. The initial hematocrit of 49.3% (which didn't change much over time) suggests that a significant portion of the blood transferred to the placenta had already been returned to the fetus.

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This case provides insight into the cord blood gases of a fetus/newborn with cord occlusion and terminal fetal bradycardia in whom the occluding forces are released prior to irreversible fetal bradycardia. When the period of umbilical venous occlusion with restored umbilical arterial blood flow is prolonged (much-widened pH and Pco_2 differences), apparently, it takes more than three minutes for the equilibration of umbilical venous and arterial blood gases.

Lastly, a diagnosis of septic shock (see Case 21) should be mentioned. This diagnosis as a cause of the cord gas values seems unlikely as umbilical cord metabolic acidosis had entirely normalized in less than two hours, the WBC count and differential normalized within 18 hours of birth, and the blood culture was negative.

Please note that the interpretation of this case in the 1st edition of this book was quite different (concomitant uteroplacental insufficiency and umbilical cord occlusion). Experience and continuing cogitation can make a difference.

Key Points

- In umbilical vein occlusion, the umbilical venous blood gas tends to be normal or near-normal. There are widened differences between venous and arterial pH, Pco_2, and sometimes base deficit.
- When severe cord occlusion is released before irreversible fetal bradycardia, which occurs just before birth, there will be

a resultant transfer of umbilical arterial abnormalities to the umbilical venous side. Umbilical arterial blood (that has just passed through chorionic villi) will mix with sequestered placental blood. This results in umbilical venous cord gases that are no longer normal or near-normal but not as abnormal as the umbilical arterial sample.

- If an infant has an adequate heart rate at birth, delayed cord clamping with the infant held below the level of the uterus may result in a substantial return of blood from the placenta to the newborn. In the absence of an adequate heart rate, stripping the cord may provide a similar benefit. This is not yet recommended therapy.

Case 27: Fetal Deterioration During Cordocentesis

The mother was a 22-year-old, gravida 3, para 2, aborta 0, with an intrauterine pregnancy at 31 3/7 weeks gestation with known Rh isoimmunization. (2) Early in pregnancy, the serum Rh titer was 1:64; at 23 weeks gestation, the Δ OD was mid-zone II. At 25 weeks gestation, the fetus developed ascites and pericardial effusion. Twenty-five mL of packed red blood cells (PRBCs) were transfused by cordocentesis.

Additional transfusions were given by cordocentesis at 27 and 29 weeks gestation. At 31 weeks gestation, cordocentesis was repeated under pancuronium immobilization. The pre-transfusion fetal hematocrit was 26%. After 55 mL of PRBCs had been infused through the umbilical vein, brief fetal bradycardia occurred. After recovery, another 20 mL of PRBCs were infused. The needle became dislodged, and a post-transfusion fetal hematocrit was not obtained. One hour later, the FHR monitor showed fetal tachycardia with poor variability. This was followed by a sudden deceleration to 50 bpm. An emergency cesarean delivery resulted in an infant with Apgar scores of 2, 6, and 7 at one, five, and 10 minutes, respectively.

Cord blood gas results were as follows:

	Umbilical Vein	Umbilical Artery
pH	7.04	7.26
Pco_2 (mmHg) ***(kPa)***	51 *6.80*	47 *6.27*
Po_2 (mmHg) ***(kPa)***	36 *4.80*	61 *8.13*
BD (mmol/L)	17	6

At age 36 minutes, the initial neonatal hematocrit was 39%. A follow-up hematocrit two hours later was 37%.

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Interpretation

This set of umbilical cord blood gas results presents a severe challenge in interpretation. One must rely heavily on previous knowledge and experience. The first step is to correctly deduce which sample should be labeled venous and which arterial. In this case, the easiest approach is to remember that the base deficits in the venous and arterial samples are usually approximately the same. Still, if one is significantly worse (i.e., a greater metabolic acidosis), it must be the umbilical artery specimen (see Relationship between umbilical venous and arterial blood gases, Section 2). A base deficit of 17 indicates a far worse metabolic acidosis than six. Therefore, the specimens must be mislabeled.

Cord blood gas results with correct labeling were:

	Umbilical Vein	Umbilical Artery
pH	7.26	7.04
Pco_2 (mmHg) ***(kPa)***	47 *6.27*	51 *6.80*
Po_2 (mmHg) ***(kPa)***	61 *8.13*	36 *4.80*
BD (mmol/L)	6	17

Now it can be seen that the umbilical venous pH, Pco_2, and base deficit are all normal. However, the umbilical venous Po_2 is quite high (normal, 17 to 41). The only reasonable explanation for this is exposure to an air bubble. Exposing the sample to an air bubble will not only increase the Po_2 but also will decrease the Pco_2 and increase the pH. The only parameter reported in the umbilical venous cord gas that is not affected by the air bubble is the base deficit. Changing the Pco_2, a "respiratory" event, has no effect on the metabolic component, the base deficit. We do not know how much exposure to an air bubble affected the pH, the Pco_2 or the Po_2. We can only ascertain the direction in which they are affected. Therefore, we know the true pH is lower than 7.26, the true Pco_2 is higher than 47 mmHg, and the true Po_2 is lower than 61 mmHg.

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In the umbilical arterial sample, the pH is low, the Pco_2 is normal, the Po_2 is high (normal 6 to 31 mmHg), and the base deficit of 18 mmol/L reflects a severe metabolic acidosis. The elevated Po_2 of 36 mmHg also suggests exposure to an air bubble in this sample. Therefore, we know the actual pH is lower than 7.04, the true Pco_2 is higher than 51 mmHg, and the true Po_2 is lower than 36 mmHg. The base deficit of 18 remains unaffected.

Umbilical cord occlusion with terminal fetal bradycardia should be suspected whenever differences between venous and arterial pH, Pco_2, and sometimes base deficit, are widened (see Case 11). Recognizing this is more difficult when one or both specimens have been exposed to an air bubble. However, since the base deficit is not affected by exposure to an air bubble, significantly differing base deficits suggest umbilical cord occlusion with terminal

fetal bradycardia as the etiology.

The final question to be answered is: What caused the umbilical cord occlusion? It is possible that when the needle became dislodged, a portion of the fetal transfusion went into the substance of the umbilical cord outside of the umbilical vein. This may have caused enough compression of the umbilical vein over time to result in occlusion of this vessel and subsequent fetal distress.

Additionally, an umbilical cord hematoma secondary to *in-utero* intravascular transfusion has been reported to cause umbilical arterial vasospasm. (3) Furthermore, a slowed umbilical venous blood flow before the total cessation of blood flow may have resulted in more time for downloading oxygen from the placenta to the fetus. (1) However, if the Po_2 of 61 mmHg represents improved downloading of oxygen rather than contamination with an air bubble, this would represent a personal new high by a considerable margin as previously, I have never seen a Po_2 as high as 50 mmHg secondary to improved downloading. When the placenta and attached cord were examined, an umbilical cord hematoma was not identified. Widened base deficits are also associated with fetal heart failure. However, following birth, there were no signs of heart failure, making this explanation of the etiology unlikely. The pathophysiology, in this case, remains unexplained.

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A final thought – one might have theorized that the blood gas initially labeled "venous" was an inadvertent sampling of blood given by transfusion via cordocentesis. However, the Po_2 of 61 mmHg in the correctly labeled venous sample is too high for that found in transfused blood; transfused blood is very dark. If the venous sample had been transfused blood that in turn was contaminated with an air bubble, this would have resulted in a falsely elevated Po_2 and would explain this particular "problem" away. However, other issues addressed above would remain unexplained.

Key Point

- Mislabeling of umbilical cord blood gas samples, contamination of both samples with an air bubble, and a possible unappreciated hematoma of the cord resulting in occlusion of the umbilical vein have tended to confound the interpretation of these blood gas results. Yet a deliberate, systematic approach permits deciphering many of these complex issues.

References:

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