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Interpreting Umbilical Cord Blood Gases
Section 7: Fetal Circulatory Failure, Part III

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Case 21: Fetal Septic Shock with Acute Fetal Heart Failure

The mother was a 26-year-old, gravida 2, para 0, aborta 1, with an intrauterine pregnancy at 40 2/7 weeks gestation. She was group B streptococcus (GBS) positive. Her membranes ruptured spontaneously an hour prior to admission. The fluid was clear and slightly pink. There was no vaginal bleeding. The mother reported good fetal movement up until the night before admission. She further reported fever and chills the night before admission, but at the time of admission, her temperature was 37.4° C (99.3° F). The FHR tracing revealed a fetus with tachycardia to 160-175 bpm with moderate variability and intermittent decelerations on admission. The mother reported an allergy to amoxicillin and was administered cefazolin every four hours (twice prior to delivery). There were no accelerations throughout the tracing.

Contractions were irregular. Shortly after admission, there was a prolonged deceleration lasting over four minutes associated with a uterine contraction. At times there was a sinusoidal pattern to the baseline. Over the next several hours, variable decelerations ensued with recovery to about 160 bpm. A deceleration to about 50 bpm occurred in association with a coupled contraction. The bradycardia remained below 90 for three minutes and below 100 for two minutes more. Terbutaline was administered to the mother, she was moved to the operating room for cesarean delivery, and amnioinfusion was begun. The FHR was 160-170 bpm with absent variability. This segued into a sinusoidal pattern. The mother was returned to the labor room. The baseline rate was about 130 bpm with absent variability immediately after decelerations. Shortly, the fetal baseline rose to 140 bpm. More severe variable decelerations appeared, prompting various maneuvers to deal with fetal distress. The cervix was 6/70%/-2. The fetal baseline heart rate trended down to 130 and then 120 bpm. A variable deceleration to 60 recovered to only 100 bpm. Terbutaline was given again. Acoustic stimulation produced a deceleration rather than an acceleration. The baseline was 120 bpm. Shortly thereafter, the baseline was 100 bpm, followed by deceleration to 60 bpm, which remained unchanged until the monitor was discontinued six minutes prior to emergent primary low-transverse cesarean delivery. All told, the FHR was 100 bpm or less for the final 22 minutes of monitoring prior to delivery. Thick, meconium-stained fluid was present. Apgar scores were 1, 1, 1, 4, and 4 at one, five, 10, 15, and 20 minutes, respectively.

Cord blood venous gases were as follows (an attempt at obtaining an umbilical arterial sample was unsuccessful):

	Umbilical Vein	Umbilical Artery
pH	7.04	Missing
Pco_2 (mmHg) ***(kPa)***	54 *7.20*	Missing
Po_2 (mmHg) ***(kPa)***	47 *6.27*	Missing
BD (mmol/L)	16	Missing

The birthweight was 3550 g. The infant was apneic and cyanotic. Resuscitation included repeated suctioning, but large amounts of fluid emanating from the trachea and esophagus prohibited adequate visualization of the glottis. Positive pressure ventilation by bag and mask did not improve the heart rate. The infant was successfully intubated at eight minutes of age. At that time, ETT suctioning yielded fluid with a curdled milk appearance. The ETT was removed under suction, and a second ETT was placed at age 10 minutes. Chest compressions began almost immediately after birth. Both epinephrine and sodium bicarbonate were administered IV twice, followed by a normal saline volume bolus. The heart rate became greater than 100 bpm at age 12 minutes.

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At age 35 minutes, the initial neonatal blood gas results were as follows:

	Infant's ABG
pH	6.59
Pco_2 (mmHg) ***(kPa)***	60 *8.00*
Po_2 (mmHg) ***(kPa)***	109 *14.53*
BD (mmol/L)	32

The base deficit did not normalize until age 30 hours. Low blood pressure was treated with dopamine. The corrected WBC count at

age 30 minutes was 14,640/mm^3 with an immature to total neutrophil (IT) ratio of 0.31; the follow-up corrected WBC count, and IT ratio were 9,820/mm^3 and 0.75, respectively. Initial and follow-up hematocrits were 53 and 54%, respectively. The blood culture was positive for GBS. Follow-up blood and spinal fluid cultures were negative. Initial treatment was with ampicillin, gentamicin, and cefotaxime. On day of life three, gentamicin and cefotaxime were discontinued; ampicillin was continued for a total of 21 days as the CRP remained positive for an extended period. The placenta demonstrated severe, acute chorioamnionitis and acute funisitis.

Blood lactate, liver function tests, serum creatinine, and clotting studies were all abnormal. An initial echocardiogram showed decreased right ventricular systolic function, a patent foramen ovale with bidirectional shunting, and a small patent ductus arteriosus with a left to right shunt.

The liver was palpated three cm below the right costal margin on initial physical examination. The infant was evaluated for total body cooling and qualified by: comatose/stupor, distal flexion/frog-legged, absent spontaneous activity and suck, incomplete Moro, and periodic breathing.

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Interpretation

The umbilical venous blood sample demonstrates mild respiratory acidosis, severe metabolic acidosis, and an elevated Po_2. An elevated umbilical venous Po_2 is associated with slowed venous blood flow, thus allowing improved downloading of oxygen across the placenta. (1) As obtaining an umbilical arterial sample is considerably more difficult than a venous sample (especially when there is little or no umbilical arterial blood flow), it is not unusual for this sample to be missing. The follow-up arterial blood gas taken from the infant shows mild respiratory acidosis and extremely severe metabolic acidosis. This is the second-worst metabolic acidosis I have ever seen in an infant who survived. It is worse than the great majority of infants who do not survive.

Now for the more difficult part, trying to reconstruct what the umbilical arterial blood gas would have been had it been successfully obtained. This is not as hard as it might seem. Categorically speaking, there are only two possibilities –similar to the umbilical venous gas or significantly worse. The hallmark of placental abruption is the presence of similar derangements in venous and arterial cord gases (see Case 8). In this infant, no abruption is reported, and although there was an occasional late deceleration present during the approximately seven hours of recorded FHR monitoring, it was hardly a prominent feature. Further, in placental abruption, the umbilical venous cord gas would be expected to demonstrate not only a significant base deficit, as was found in this infant, but additionally, a very elevated Pco_2 and a depressed Po_2, quite the opposite of this cord gas. These findings could result from significant exposure to an air bubble. This is a possibility. However, having the cord venous Po2 result fall in a physiologically plausible range would be a major coincidence.

The other categorical possibility is an arterial cord gas that is significantly worse than the venous cord gas. This finding is associated with terminal fetal bradycardia with either cord compression or fetal heart failure. In this case, variable decelerations were rather prominent in the recorded fetal monitoring strip, and some of them were quite deep and lasted more than 60 seconds. However, the terminal bradycardia was not precipitous but relatively gradual, suggesting the absence of cord occlusion as the terminal event. Additionally, cord gases associated with cord occlusion usually demonstrate a normal or near-normal umbilical venous blood gas, rather than the severe metabolic acidosis present in this infant. That leaves fetal heart failure as the one remaining established cause of an umbilical arterial cord gas that is significantly worse than its paired umbilical venous cord gas.

In this infant, profound anemia was not the cause of fetal heart failure as it was in Case 19. However, another known cause of fetal heart failure is present – septic shock. What supports this diagnosis? The mother was GBS positive and had a fever and chills the night before admission. The FHR tracing was tachycardic without an associated maternal fever and was never reactive or reassuring. There was severe umbilical venous metabolic acidosis and an umbilical venous Po_2 that was elevated, a finding consistent with slowed blood flow, as would be typical in fetal heart failure. Slowed fetal blood flow allows for an increased time for oxygen to download across the placenta to the fetus. Additionally, the newborn's liver was enlarged, further supporting the diagnosis of fetal heart failure. Neonatal sepsis was also supported by the WBC count and differential and the persistently elevated CRP. Of course, the sine qua non of neonatal sepsis is a positive blood culture — in this case, GBS. Further, the diagnosis of septic shock was supported by initial hypotension after birth, the requirement for pressor therapy, and the profound metabolic acidosis present at age 35 minutes which took over 30 hours to normalize. The base deficit in a "usual" case of hypoxic-ischemic encephalopathy normalizes within 4.5 to 7.9 hours. (2) This infant demonstrated the classic findings of septic shock.

"Fetal heart failure would be expected to generate widened venoarterial pH and Pco2 differences, as it does in both children and adults (although the differences are in the opposite direction). (3) A widened umbilical venoarterial base deficit difference would occur in the same manner as described in the previous infant (Case 20), i.e., right heart failure leading to elevated central venous pressure, which in turn can lead to slowed umbilical venous blood flow, as well as slowed systemic blood flow."

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cur in the same manner as described in the previous infant (Case 20), i.e., right heart failure leading to elevated central venous pressure, which in turn can lead to slowed umbilical venous blood flow, as well as slowed systemic blood flow. Terminally, there is complete cessation of umbilical venous blood flow. (2) Just as in the previous infant, complete cessation occurs prior to complete cessation of umbilical arterial flow for a substantial base deficit difference to exist. If the complete cessation of umbilical venous blood flow has not occurred, the base deficit on the arterial side will be transmitted to the venous side.

Why is it that this fetus with septic shock and heart failure, unlike the previous infants with heart failure, had severe umbilical venous metabolic acidosis? I am unsure as this is the only newborn with septic shock in whom I have seen umbilical cord blood gases (only a venous gas). Although we seldom obtain arterial and venous blood gases on newborns in the NICU, undoubtedly, if an arterial blood gas shows a metabolic acidosis, so will a venous gas. On the other hand, children or adults with heart failure alone do not develop metabolic acidosis until near death. What makes it clear is that the same thing that happens in the fetus is finding an absence of metabolic acidosis in the umbilical vein (see Case 19).

Perhaps the difference between a fetus with heart failure and a fetus with sepsis *and* heart failure (septic shock) is that heart failure without sepsis occurs slowly over time and better allows for compensation. Once further compensation is no longer possible, an additional insult, such as a further decrease in hemoglobin, results in a greater increase in central venous pressure that brings umbilical venous blood flow to a halt. Now that the only source of oxygen is entirely cut off, metabolic acidosis develops rapidly. The placenta continues to accept blood from the umbilical arteries for a short period, but because umbilical venous blood flow has stopped, metabolic acidosis cannot be transferred to the venous side. At this point, decompensation occurs very quickly. Unlike chronic fetal heart failure, fetal septic shock is a relatively acute event. At first, metabolic acidosis is transmitted to the umbilical vein, but terminally, umbilical venous blood flow also comes to a halt as it does in chronic fetal heart failure, allowing for very wide umbilical venoarterial blood gas differences.

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It is also worth noting that despite prompt and appropriate prophylactic antibiotic therapy administered to the mother, the infant had a positive blood culture. An interesting study (4) demonstrated that newborns with proven sepsis treated directly with IV antibiotics typically still had positive blood cultures 24 hours later. However, newborns may still be septic without positive blood culture (5) with or without prior maternal antibiotics.

Key Points

- A fetal sinusoidal heart rate pattern may be secondary to any cause of fetal heart failure, including severe fetal anemia. Further study on this point is warranted.
- Fetal septic shock appears to be associated with umbilical cord blood gases similar to those seen in other newborns who have heart failure at the time of birth, i.e., widened differences between venous and arterial cord pH, $Pco_{2,}$ and base deficit.
- Additionally, unlike a non-septic fetus in heart failure, a fetus with septic shock may show a significant umbilical venous base deficit rather than a normal or near-normal base deficit.

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