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

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*"Shoulder dystocia), and it took between five and 10 minutes with various maneuvers (including McRoberts maneuver - removing the legs from the stirrups and sharply flexing them up onto the abdomen) to deliver the entire infant."*

## Case 17: Shoulder Dystocia with Delayed Delivery

The mother was a 31-year-old, gravida 2, para 0, aborta 1, with an intrauterine pregnancy at 38 3/7 weeks gestation. She was a gestational diabetic taking NPH insulin twice daily. Her hemoglobin A1C was elevated at 7.3%. During her previous pregnancy, her gestational diabetes mellitus was diet controlled. Her group B streptococcus colonization status was negative, and the estimated fetal weight was 3200 g.

During labor, the mother received intravenous insulin at one unit/hour. Artificial rupture of membranes resulted in egress of fluid that was lightly stained with meconium. The FHR pattern was reassuring. Although the mother's maximum temperature was 99.4° F, she received two doses of ampicillin intravenously. About 75 minutes after complete cervical dilatation with the fetus in the OA position, a vacuum extractor was applied with rapid delivery of the head. There was considerable difficulty in extricating the shoulders (shoulder dystocia), and it took between five and 10 minutes with various maneuvers (including McRoberts maneuver - removing the legs from the stirrups and sharply flexing them up onto the abdomen) to deliver the entire infant. Apgar scores were 1 and 6 at one and five minutes, respectively. The infant was intubated, with no meconium found below the cords. Positive pressure bag-tube ventilation with 100% oxygen was applied for approximately two minutes. Birth weight was 4340 g.

Cord blood gas results were as follows:

|  | Umbilical Vein | Umbilical Artery |
|---|---|---|
| pH | 7.27 | 7.16 |
| Pco$_2$ (mmHg) *(kPa)* | 46/*6.13* | 61/*8.13* |
| Po$_2$ (mmHg) *(kPa)* | 27/*3.60* | 20/*2.67* |
| BD (mmol/L) | 6 | 7 |

A follow-up ABG at about 15 minutes of age was:

|  | Infant's ABG |
|---|---|
| pH | 7.27 |
| Pco$_2$ (mmHg) *(kPa)* | 30/*4.00* |
| Po$_2$ (mmHg) *(kPa)* | 96/*2.80* |
| BD (mmol/L) | 13 |

The infant was given 10 mEq of sodium bicarbonate intravenously. A follow-up capillary blood gas about one hour later was:

|  | CBG |
|---|---|
| pH | 7.42 |
| Pco$_2$ (mmHg) *(kPa)* | 43/*5.73* |
| Po$_2$ (mmHg) *(kPa)* | 47/*6.27* |
| BD (mmol/L) | -3 |

**Interpretation**

The umbilical venous blood gas is normal. The umbilical artery sample has a slightly low pH, but the other values are all normal. The relationships between the venous and arterial values are also normal. The difference between the umbilical venous and arterial pHs is 0.11, slightly widened and therefore suggestive of cord occlusion. The initial follow-up ABG on the infant showed mild metabolic acidosis and respiratory alkalosis. The second follow-up blood gas was a CBG. A CBG may not be as accurate as either an ABG or VBG. However, it tends to err in a poorly perfused baby by elevating the Pco$_2$ and increasing the base deficit. In this case, both the Pco$_2$ and the base deficit are entirely normal. Therefore, this result is probably accurate.

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Two mechanisms of cord occlusion seem tenable. When shoulder dystocia occurs, the cord is at increased risk of being compressed between a fetal shoulder and the lower uterine segment. Additionally, the umbilical cord suddenly becomes functionally shortened by the descent of the fetal head. This may be sufficient to put the umbilical cord on a significant stretch. Sudden stretching of the umbilical cord may be different from gradual stretching, as spasm of the vessels may ensue. With shoulder dystocia, sudden functional shortening of the cord or spasm of the cord vessels may be sufficient to occlude both umbilical vein and arteries.

The mechanism by which an infant becomes asphyxiated during shoulder dystocia is not established. The 25th edition of Williams Obstetrics offers only the notion that "shoulder dystocia may be associated with significant fetal morbidity and even mortality." It is likely that asphyxia, when it occurs, is secondary to cord occlusion and perhaps upper body compression during shoulder dystocia and consequent severe neonatal hypovolemia due to both mechanisms.

The effect on blood gases of increasing the time interval between delivery of the head and delivery of the body is contested. Allen et al. have reported that as the time required to deliver the shoulders increased, the 5-minute Apgar scores decreased. Stallings et al. failed to find any association between increasing intervals and either decreased Apgar scores at five minutes or with significant reductions in mean umbilical arterial blood gas parameters. When shoulder dystocia occurs, much force may be brought to bear both on the fetus and potentially on the umbilical cord as well. One would not expect occluded umbilical arteries to reflect the blood gas status of the infant after occlusion.

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Not every fetus in whom there is a considerable delay between delivery of the head and the body is adversely affected. This observation suggests that the mechanism of asphyxia is secondary to umbilical cord occlusion occurring in some fetuses and not in others. Following the FHR, if possible, during this critical time might allow one to separate those fetuses for whom time is of the essence from those for whom it is not.

Thus, one of several scenarios may accompany shoulder dystocia. Suppose the umbilical cord is compressed between the fetus and the lower uterine segment. In that case, all vessels may occlude, followed by reactive arterial hypertension, during which time the vein remains occluded but umbilical arterial blood flow is restored (if only briefly). Suppose the cord is placed on sufficient stretch or is suddenly stretched. In that case, the umbilical arteries and the umbilical vein may become permanently occluded, mimicking an umbilical cord that had been clamped. Then the umbilical cord blood gas values would reflect only the acid-base status of the infant at the time occlusion occurred.

Under stressful conditions, the length of time during which shoulder dystocia persists may be underestimated. In this case, the estimated time was five to 10 minutes. However, much can hap-

pen in this time interval, especially if the fetus is becoming hypovolemic. Mercer et al.(5) suggest that hypovolemia may become extreme. They hypothesize that fetal hypovolemia may result not just from cord occlusion with accompanying transfer of blood to the placenta but also with further transfer of blood to the placenta due to the "tight compressive squeeze of the body in the birth canal." Additionally, loss of the many stem cells present in cord blood may be essential for central nervous system healing and repair. Studies in rats have found that following induced brain damage, human umbilical stem cells can protect the rat's brain from developing a permanent injury.

In those infants who are significantly hypovolemic at birth, volume rather than red blood cells appears to be the critical item, as quite low fetal hemoglobin levels still appear to be sufficient to avoid brain injury. It is possible that simply infusing normal saline might be sufficient. The infant presented in this case was not depressed enough to warrant volume replacement of any sort, but other infants may be.

In the case above, the clinical appearance of the infant at the time of birth suggests that the umbilical cord blood gas values may not accurately reflect the condition of the baby. Obtaining a blood gas directly from the infant shortly after birth will frequently give a more accurate picture and provide insight into the meaning of the cord blood values. By 15 minutes of age, clinically much improved from birth, this infant was spontaneously hyperventilating ($Pco_2$ of 30 mmHg), compensating for the metabolic acidosis (base deficit of 13) with respiratory alkalosis One hour later, the infant was no longer hyperventilating, and the base deficit was three. The data of Shah et al. and Aschner and Poland ) suggest that the administration of sodium bicarbonate does not accelerate recovery from metabolic acidosis.

The infant did well and was discharged home on the third day of life.

### Key Points

- Shoulder dystocia appears to have a variable effect on occlusion of the umbilical cord.

- When the umbilical cord is occluded, the mechanism of occlusion may be cord occlusion at the site of shoulder dystocia or sudden stretch of the cord vessels as the head exits the cervix.

- Following the FHR, if possible, during shoulder dystocia, may enable one to separate those fetuses for whom time to delivery is of the essence from those for whom it is not.

- Widened umbilical venoarterial pH differences may occur in association with shoulder dystocia, just as they do in association with other causes of cord occlusion.

- Blood gas samples will only reflect fetal status prior to occlusion.

- Compression of the fetal body during shoulder dystocia may result in a greater transfer of blood to the placenta than in other situations.

Case 18: Breech Delivery with Trapped Head

The mother was a 19-year-old, gravida 1, para 0, aborta 0, with an intrauterine pregnancy at 30 6/7 weeks gestation in active labor. The cervix was five cm dilated and completely effaced with the fetus in breech presentation. Although the mother was advised that the infant should be delivered by cesarean section, she insisted on vaginal delivery. Labor ensued without problem until the aftercoming head became trapped for approximately 12 minutes. A female infant was ultimately delivered with Apgar scores of 0, 1, 5, 7, and 8 at one, five, 10, 15, and 20 minutes, respectively.

Cord blood gas results were as follows:

|  | Umbilical Vein | Umbilical Artery |
|---|---|---|
| pH | 7.31 | 7.20 |
| Pco$_2$ (mmHg) (kPa) | 42/5.60 | 57/7.60 |
| Po$_2$ (mmHg) (kPa) | 44/5.87 | 22/2.93 |
| BD (mmol/L) | 5 | 6 |

Resuscitation included intubation and ventilation, chest compressions, epinephrine via ETT, and placement of a UVC.

At six minutes of life, a blood gas sample was drawn from the UVC, just prior to giving five meq of sodium bicarbonate.

Results from the UVC sample were:

|  | UVC |
|---|---|
| pH | 7.02 |
| Pco$_2$ (mmHg) (kPa) | 48/6.40 |
| Po$_2$ (mmHg) (kPa) | 75/10.00 |
| BD (mmol/L) | 19 |

Birth weight was 1620 g. The infant had mild respiratory distress syndrome, received one dose of surfactant, and was extubated at approximately 24 hours of life. The infant was never tremulous, had good urine output and normal renal function tests. A head ultrasound examination was normal.

### Interpretation

The umbilical venous blood gas is normal, except for a slightly elevated Po$_2$, which is likely secondary to a brief period of slowed blood flow prior to complete occlusion.

The umbilical arterial blood sample results are entirely normal, as are the usual relationships between venous and arterial values. The difference between the venous and arterial pH is widened (>0.10), although only slightly, suggesting that after the fetal head became entrapped, the umbilical cord was occluded, followed by a brief period in which the umbilical artery blood flow was restored while the umbilical vein remained occluded.

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The umbilical arterial blood gas values do not represent the situation within the fetus at the time of birth. The umbilical venous blood gas represents the situation in the fetus at the time the fetal head became entrapped during delivery. If entrapment is brief, umbilical cord arterial blood gas values will closely reflect the infant's condition, but as the time of entrapment increases, this becomes progressively less so.

The UVC blood gas sample values obtained at six minutes of life are very different from those drawn from the umbilical cord at the time of birth. The base deficit comes much closer to what one would have expected, considering the clinical history and the infant's appearance at the time of birth. The Po$_2$ is too high for venous blood, even if the infant were receiving 100% oxygen. This suggests that the UVC extended into the heart and crossed through the foramen ovale into the left atrium, thus reflecting an "arterial" Po$_2$.

In a breech delivery with head entrapment, the umbilical cord extends from the umbilicus up through the cervical os, next to the infant's head, and then onto the placenta. As the fetal head becomes wedged in the pelvis, the umbilical cord is compressed between the infant's head and the lower uterine segment.

At the time of head entrapment, the umbilical cord becomes functionally shortened by the distance from the breech to the cervical os, likely placing the cord on a significant stretch.

Additionally, average umbilical cord length is significantly shorter in breech compared with cephalic presentations.[ ] However, strong external compression and occlusion of the umbilical cord may make these points moot.

### KEY POINTS

- Breech delivery with a trapped head will likely result in complete occlusion of the umbilical cord by compression between the fetal head and the lower uterine segment.

- Wider than normal differences between umbilical venous and arterial pH and Pco$_2$ suggest an interval when the umbilical vein was occluded, but the umbilical arteries were not.

- Blood in the umbilical vein will then reflect the infant's status prior to this event.

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