

The Use and Misuse of Cord Blood Gases in HIE Litigation

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“Umbilical cord blood gases are often used as evidence for or against the presence of intrapartum asphyxia in litigated cases of hypoxic-ischemic brain injury...However, there are many variables and questions regarding the cord blood gas values which are often not completely understood and may mislead the clinician in the assessment of the neonate for therapeutic hypothermia (TH) as well as obfuscate the facts in a hypoxic-ischemic encephalopathy (HIE) malpractice suit.”

Umbilical cord blood gases are often used as evidence for or against the presence of intrapartum asphyxia in litigated cases of hypoxic-ischemic brain injury. If blood is free-flowing in the umbilical cord, the measured values of the umbilical cord blood gas provide valuable objective evidence of the metabolic condition of the newborn at the time of birth. However, there are many variables and questions regarding the cord blood gas values which are often not completely understood and may mislead the clinician in the assessment of the neonate for therapeutic hypothermia (TH) as well as obfuscate the facts in a hypoxic-ischemic encephalopathy (HIE) malpractice suit. This article will deal with a number of these potentially confusing and contradictory issues. The following questions will be addressed:

1. Is the blood gas arterial or venous? How can you tell?
2. If the cord gas is venous, can you reliably predict what the arterial gas would have been?
3. Is there a change in the blood gas values if there is a delay in running the sample?
4. If the blood gas is collected in heparin, how will that affect blood gas values? How about an air bubble?
5. How does delayed cord clamping (DCC) affect the blood gas values?
6. What if the blood gas and the baby's condition are inconsistent (e.g., the baby has a 1-minute Apgar score of

0–2, and the cord gas looks relatively normal)?

7. Does a blood gas with a pH < 7.00 mean the baby has HIE? Why was this sharp line drawn in the criteria for HIE and cooling?

The reader is referred to two general references for a complete discussion of this topic: *Interpreting Umbilical Cord Blood Gases*, 2nd Edition by JJ Pomerance (1) and a recent comprehensive review by Per Olofsson. (2)

Is the blood gas arterial or venous? How can you tell?

In the fetus, the umbilical vein carries oxygenated blood from the placenta to the baby, and the blood passes through the ductus venosus in the liver and cardiac shunts into the left side of the heart and then into the aorta. The two umbilical arteries carry blood back to the placenta to be reoxygenated. When there is no obstruction to flow in the umbilical cord, the blood flows in equal volumes in both directions. The placenta functions as the fetal lung, and umbilical cord blood gases should reflect this function. The umbilical artery (UA) pH should be lower than the umbilical venous (UV) pH, the UA pCO₂ should be higher than the UV pCO₂, and the UA pO₂ should be lower than the UV pO₂. Normal values and ranges can be seen in Table 1. (1)

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The overall success rate of obtaining an arterial cord blood sample is <80%. Sometimes, only one umbilical cord blood gas sample is collected, and the clinician has to decide if it is appropriately labeled UA or UV. If two samples are obtained and the UV has a lower pH, higher pCO₂, or lower pO₂ than the UA, then the samples potentially are mislabeled, or other factors have affected

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Table 1: From Pomerance JJ (1)(with permission)

Normal Umbilical Cord Blood Gases		
	Venous Blood Normal Range (Mean $\pm$ 2SD)	Arterial Blood Normal Range (Mean $\pm$ 2SD)
pH	7.25 – 7.45	7.18 – 7.38
pCO ₂ (mmHg) (kPa)	26.8 – 49.2 3.57 – 6.56	32.2 – 65.8 4.29 – 8.77
pO ₂ (mmHg) (kPa)	17.2 – 40.8 2.29 – 5.44	5.6 – 30.8 0.75 – 4.11
HCO ₃ ⁻ (mmol/L)	15.8 – 24.2	17 – 27
BD (mmol/L)	0 to 8	0 to 8

the credibility of the values. One study revealed that at least 18% of paired samples were taken from the same vessel. (3) Moreover, samples are often mislabeled, labeling a UV sample as UA since the UV is much easier to access than the UA. A UA pO₂ cannot be higher than 32 mmHg; thus, a pO₂ greater than 32 in the sample would alert the clinician that a supposed UA sample is probably from the UV.

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If the cord gas is venous, can you reliably predict what the arterial gas would have been?

The UA and UV samples usually reflect a standard value difference when blood flows freely in the umbilical cord. If the fetus is becoming acidemic and there is no obstruction to blood flow, the returning UA pH can only be improved to a limited extent by the placenta's ability to restore acid-base balance. Thus, the standard difference between the UA and UV pH is 0.02–0.10 units. A pH of 7.25 in the UV will generally reflect a pH of no lower than 7.15 in the UA. Cantu et al. (4) looked at 11,455 paired umbilical cord blood gas samples and found a very high correlation between the UV and UA values. For example, if the UV pH was 7.23 or greater, the chance of the UA blood gas pH being < 7.0 was $\leq 1\%$. Similar findings were reported by Swanson et al. in paired samples collected from 36,325 births. (5) In a small number of cases, there may be obstruction to umbilical cord blood flow or a

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significant decrease in fetal cardiac output. In these cases, A-V differences may be as large as 0.4–0.5 units. Thus, a UV pH of 7.20 may be associated with a UA pH of 6.7–6.8. Often, these cases are associated with an obstetric sentinel event such as a cord prolapse, uterine rupture, shoulder dystocia, or maternal cardiac collapse.

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Is there a change in the blood gas values if there is a delay in running the sample?

Often, there is a delay in analyzing the umbilical cord blood gases after delivery. Optimally, the analysis should be done as soon as possible, but emergent care of the mother and newborn may delay this examination. The cord gas may represent the first gas in neonatal resuscitation, and many NICU units take an I-STAT device to the delivery room to facilitate the rapid processing of the sample, which may help guide neonatal care. In litigation, lawyers may claim that a delay of 30–60 minutes in analyzing a gas may invalidate the results. Studies have shown that no significant changes are seen in a cord gas if drawn from a segment of a doubly clamped cord, kept at room temperature, or collected into a plastic syringe and left at room temperature for 60 minutes after

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If the blood gas is collected in heparin, how will that affect blood gas values? How about an air bubble?

In most hospitals, cord blood gases are drawn in pre-heparinized syringes, and the effect of heparin contamination is moot. However, if heparin is used, there is the potential to lower the pH and $p\text{CO}_2$ levels in the sample and worsen the base deficit. Many blood gas analyzers will not process a sample with air bubbles, but even a small amount of air in a sample can cause significant errors. Room air has a $p_{\text{A}}\text{O}_2$ of about 150 mm Hg and a $p_{\text{A}}\text{CO}_2$ of nearly 0. Therefore, an air bubble in a cord blood gas sample will erroneously increase the $p\text{O}_2$ value, decrease the $p\text{CO}_2$ value, increase the base deficit, and have a negligible effect on the pH. (1)

What is the effect of delayed cord clamping on the blood gas values?

Delayed cord clamping (DCC) of 30–60 seconds is now recommended for both term and preterm newborns who do not require significant resuscitation. (8) There are conflicting data on whether this practice, which has been shown to have multiple benefits to the baby, will affect the umbilical cord blood gas. One study found no difference in umbilical cord arterial pH values and increased $p\text{O}_2$ levels after DCC. However, two other studies found that there was a small but statistically significant decrease in the UA pH (0.03 units) with DCC. (8) In clinical practice, babies who are non-vigorous at birth and likely to need resuscitation should not have DCC anyway, and the cord blood gas values will not be affected.

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What if the blood gas and the baby's condition are inconsistent (e.g., the baby has Apgar scores of 0–2, and the cord gas looks relatively normal)?

Occasionally, the cord blood gas values are inconsistent with those of the baby at birth. In an HIE litigation, the defense lawyer will argue that a baby born with an Apgar score of 0 or 1 at 1 minute was not asphyxiated in utero because the cord blood gas values were normal. In a non-dysmorphic newborn, this argument is not logical. There are many reasons why this phenomenon occurs, but usually, some event in utero has obstructed blood flow through the umbilical cord, or the fetal heart has lost the ability to perfuse the umbilical cord arteries. Clinically, there may be terminal bradycardia or a sentinel event such as cord prolapse or prolonged dystocia, but often, an adequate explanation may not be apparent.

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When the umbilical cord is obstructed, blood flow in both the arteries and the vein may stop, and the sample taken at birth will reflect the condition of the fetus prior to the obstruction. This may allow the defense attorney to argue that the fetal monitoring strip did not show signs of significant fetal compromise prior to the terminal obstruction and that the terminal event was unpredictable. In a clamped cord in an animal model, the cord arterial pH falls approximately 0.02–0.04 units per minute. (1) Thus, a fetus who suffers a complete cord obstruction while the mother is pushing at the end of labor may drop from an acceptable cord pH of 7.2 to a pH of 6.8 in 10 minutes. The baby is born severely depressed, but the cord gas reflects the condition of the fetus prior to the obstruction (i.e., pH 7.2). This may also affect the clinical care given to the baby since the provider may find that the baby does not meet the blood gas criteria for TH, and the baby does not receive the potential benefits of that therapy. (9)

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This condition may become even more problematic if the cord is partially obstructed, affecting only flow through the thin-walled umbilical vein and not affecting flow through the thicker-walled umbilical arteries. Now, the fetus is not only deprived of fresh oxygen from the placenta, but the arteries continue to flow blood to the placenta without the same amount returning to the fetus through the vein. The baby will then become hypovolemic as well as asphyxiated during this period of partial obstruction.

Following cord occlusion, blood gases often do not reflect the baby's condition. In these circumstances, believe the baby; and get an early neonatal blood gas and lactate, which should reflect the true condition of the baby at birth. Post hoc evaluation of this condition can be supported by a clinical history compatible with an obstruction (i.e., cord prolapse or shoulder dystocia) or physical findings (i.e., a true knot in the cord, decreased twists (coiling) per 10 cm in the cord, absent or decreased Wharton's jelly around the cord and vascular ectasia in the fetal vessels in placental evaluation). A tight nuchal cord has been associated with obstruction, but the evidence for this phenomenon is not compelling.

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Does a blood gas with a pH < 7.00 mean the baby has HIE? Why was this sharp line drawn in the criteria for HIE and cooling?

Since the first ACOG statement on criteria for intrapartum asphyxia (ACOG Technical Bulletin #163, 1992), the College has used the benchmarks of cord pH < 7.0 and base deficit of ≥ 12 mmol/L as criteria for the diagnosis of intrapartum asphyxia. These criteria were repeated in the 2003 and 2014 monographs on neonatal encephalopathy, although the 2014 Task Force no longer required these values to be “essential criteria” for diagnosing an acute intrapartum hypoxic event sufficient to cause brain injury. (10) However, when these values are critically examined, only a small fraction of babies born with these cord gas values end up with HIE. Goodwin et al. showed that when the UA pH was 6.90 to 6.99, only 12% of newborns developed HIE. In that study, it was not until the pH was < 6.8 that a majority of babies had neurologic and systemic

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features of intrapartum asphyxia. (11) Low and colleagues showed similar results with their review of UA base deficit. When the base deficit was 12–16 mmol/L, 72% of babies had no encephalopathy, and 19% had only minor symptoms such as irritability or jitteriness. (12) Thus, 91% of babies with a base deficit of 12–16 mmol/L had no or only minor symptoms of encephalopathy. It should be noted that asphyxia usually progresses from respiratory to mixed acidosis and then to predominant metabolic acidosis. Respiratory acidosis rarely causes brain injury since it causes vasodilation in the brain. (10) Every 10 mmHg rise in pCO₂ decreases the pH by 0.08 units. Thus, for example, a cord blood gas with a pH of 6.90, pCO₂ of 110 mmHg, and base deficit of 10 would generally indicate a much higher pH (possibly close to normal) based on the minimal metabolic contribution, a very recent asphyxial event and a very low chance of HIE or long term sequelae in the baby. (13)

Why, then, were these numbers chosen as indicia of intrapartum asphyxia? The incidence of cerebral palsy (CP) in the United States is approximately 1–4/1000 in term infants. The incidence of HIE in the acidemic patients described in the Goodwin study is 12%. In comparison, there is a 100-fold increase in CP in a term patient with a cord pH < 7.0 versus a baby whose cord pH is above 7.0. On the other hand, only approximately 1 in 10 newborns with a pH of 6.90 to 6.99 will have HIE, potentially leading to cerebral palsy. The cord blood gas values in and of themselves do not tell the whole story and must be viewed in the context of the rest of the maternal and neonatal history. Appropriately, in the last iteration of the neonatal encephalopathy monograph, published in 2014 and reaffirmed in 2019, the “essential criteria” were retired. Providers were advised to review the entire clinical history, fetal monitoring strips, labs, neuroimaging, and other factors before ascribing a neonatal encephalopathy to an acute intrapartum asphyxia event. (10)

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