Interpreting Umbilical Cord Blood Gases: Normal Cord Blood Gases

Jeffrey Pomerance, MD, MPH

Case 1: Normal Umbilical Cord Blood Gases

The mother was a 34-year-old, gravida 1, para 0, aborta 0, with an uncomplicated intrauterine pregnancy at 40 4/7 weeks. The fetal heart rate tracing showed mild, variable decelerations. After 11 hours of labor, the mother was taken to the delivery room and vaginally delivered a male infant with Apgar scores of 7 and 9 at one and five minutes, respectively.

Cord blood gas results were as follows:

	Umbilical Vein	Umbilical Artery
pH	7.35	7.28
Pco_2 (mm Hg)	38	49
(kPa)*	*5.07*	*6.53*
Po_2 (mm H)	29	18
(kPa)*	*3.87*	*2.40*
HCO_3^- (mmol/L)	20	22
BD (mmol/L)	4	4

* kPa, kilopascal, the unit of measure used in many countries outside the United States, 1kPa = 7.50 mmHg

Interpretation

These blood gas results represent the mean normal umbilical cord blood gas values as defined by a study published in 1985 of 146 uncomplicated term vaginal deliveries by Yeomans, Hauth, Gilstrap, and Strickland. The range of normal values from which these means were derived is found in Table 1 below.

Umbilical Cord Blood Gases Degree of Respiratory Acidosis		
	Venous Pco_2 (mmHg) *(kPa)*	**Arterial Pco_2 (mmHg) *(kPa)***
No Respiratory Acidosis (Normal Range)	27 – 49 *3.57 – 6.56*	32 – 66 *4.29 – 8.77*
Mild Respiratory Acidosis	50 – 65 *6.67 – 8.67*	67 – 80 *8.80 – 10.67*
Moderate Respiratory Acidosis	66 – 80 *8.80 – 10.67*	81 – 95 *10.80 – 12.67*
Severe Respiratory Acidosis	81 – 95 *10.80 – 12.67*	96 – 110 *12.80 – 14.67*
Very Severe Respiratory Acidosis	≥ 96 *≥ 12.80*	≥ 111 *≥ 14.80*

Table 1

Reprinted with permission from Elsevier, in part from Yeomans ER, Hauth JC, Gilstrap LC III, Strickland DM. Umbilical cord pH, PCO_2, and bicarbonate following uncomplicated term vaginal deliveries Am J Obstet Gynecol 1985;151:798-800.

Data are mean values ± 2 standard deviations (SD).
* Base deficit, estimated from data.
** 1 kPa = 7.50 mmHg; 1 mmHg = 0.133 kPa

Note: "Normal" is arbitrarily defined as the mean ± two times the standard deviation (approximately 95.4% of a normally distributed population).

These values should be used in interpreting the umbilical cord blood gas sets presented in the remainder of this text. While subsequent studies generally involving larger numbers also have defined normal umbilical venous and arterial blood gas values, or sometimes only umbilical artery blood gas values in both term , (6) and preterm infants, (4,5,7) the findings are very similar, differing only in the range of the standard deviations.

"Compared to the umbilical venous cord values in healthy term infants after vaginal delivery, (3) values in the umbilical vein obtained prior to delivery in unsedated, non-laboring patients at 35 weeks' gestation by cordocentesis, showed higher pH (7.41 vs. 7.35) and PO_2 (35 vs. 29 mmHg), and lower PCO_2 (36 vs. 38 mmHg) and base deficits (1 vs. 4 mmol/L). (11)"

White et al (8) found that despite the intention to obtain both an umbilical venous and an umbilical arterial sample, this was successful only 64% of the time. Both Armstrong and Stenson (9) and Westgate et al (6) emphasized that "Both artery and vein

cord samples must be taken and the results screened to ensure separate vessels have been sampled." The American Congress of Obstetricians and Gynecologists also recommends attempting to obtain both "venous and arterial blood cord samples … for blood gas analysis." (10)

Compared to the umbilical venous cord values in healthy term infants after vaginal delivery, (3) values in the umbilical vein obtained prior to delivery in unsedated, non-laboring patients at 35 weeks' gestation by cordocentesis, showed higher pH (7.41 vs. 7.35) and PO2 (35 vs. 29 mmHg), and lower PCO2 (36 vs. 38 mmHg) and base deficits (1 vs. 4 mmol/L).(11) Similarly, when umbilical artery samples obtained from healthy term infants born by cesarean section in the absence of labor were compared with infants born by vaginal delivery, pH was higher by 0.04 on average.(12) Increasing the duration of the second stage of labor also was significantly related to deteriorating umbilical artery blood gas values with the base deficit rising by about 1 mmol/L per hour. (13) Even uncomplicated labor and vaginal delivery exacts a toll on fetal acid-base values, although modest and apparently of little clinical importance.

The standard deviations provided by Yeomans, Hauth, Gilstrap, and Strickland (3) assume the distribution of values to be normal or bell-shaped. In reality, they are all skewed distributions with the long thin tail to the left. However, I do not believe that any of the cord blood gas interpretations are influenced by this deviation from the bell-shaped curve. The average PO_2 in umbilical venous and arterial blood gases are 29 and 18 mmHg, respectively. The standard deviations are approximately six mmHg in each. In the umbilical arterial blood gas, plus or minus two standard deviations includes values between six and 30 mmHg. Surprisingly, a PO_2 below the normal range is poorly associated with asphyxia. However, PO_2 is informative when it is above the normal range. This suggests contamination by an air bubble(s) or, if in a venous sample, a period of slow flow through the placenta with associated increased time for down-loading of oxygen from the mother to the fetus via the umbilical vein and uploading of carbon dioxide from the fetus to the mother.

There are no agreed-upon definitions of what constitutes mild, moderate, or severe respiratory or metabolic acidosis. However, to allow for easier discussion and consistency within this text, I have set arbitrary definitions (see Tables 2 and 3 below). One must appreciate that nothing is known to occur clinically as a value crosses from one range into another. By and large, clinical relationships operate on a continuum. The concept of fetal respiratory acidosis is peculiar as the presence or absence of fetal breathing has no relationship to fetal respiratory acid-base status. The placenta acts as the "lung" for the fetus. Nonetheless, within this text, the terms respiratory acidosis or alkalosis will be used conventionally, i.e., to define the contribution of PCO_2 to the acid-base status.

Umbilical Cord Blood Gases Degree of Respiratory Acidosis		
	Venous Pco_2 (mmHg) ***(kPa)***	**Arterial Pco_2 (mmHg)** ***(kPa)***
No Respiratory Acidosis (Normal Range)	27 – 49 *3.57 – 6.56*	32 – 66 *4.29 – 8.77*
Mild Respiratory Acidosis	50 – 65 *6.67 – 8.67*	67 – 80 *8.80 – 10.67*
Moderate Respiratory Acidosis	66 – 80 *8.80 – 10.67*	81 – 95 *10.80 – 12.67*
Severe Respiratory Acidosis	81 – 95 *10.80 – 12.67*	96 – 110 *12.80 – 14.67*
Very Severe Respiratory Acidosis	≥ 96 ≥ *12.80*	≥ 111 ≥ *14.80*

Table 2

Defining degree of respiratory acidosis in umbilical cord blood gases

Rounded from Table 1

Umbilical Cord Blood Gases Degree of Metabolic Acidosis		
	Venous BD (mmol/L)	**Arterial BD (mmol/L)**
No Metabolic Acidosis (Normal Range)	0 to 8	0 to 8
Mild Metabolic Acidosis	9 – 12	9 – 12
Moderate Metabolic Acidosis	13 – 16	13 – 16
Severe Metabolic Acidosis	17 – 20	17 – 20
Very Severe Metabolic Acidosis	≥ 21	≥ 21

Table 3

Defining the degree of metabolic acidosis in umbilical cord blood gases Rounded from Table 1

It is of note that while an elevated arterial PCO_2 is associated with increased cerebral blood flow in the term (14) and preterm human newborn, this response to arterial PCO_2 is attenuated in seriously asphyxiated term infants and in mechanically ventilated preterm infants prior to intracranial hemorrhage.(15) For further discussion of this topic, see Volpe JJ, Neurology of the Newborn, 5th edition. (16)

Elevated values of PCO_2 in cord blood (low pH but normal base deficit) have not been associated with poor neurological outcome. When combined with metabolic acidosis (low pH and elevated base deficit), the risk of neurological injury increases. (17) Respiratory acidosis may be associated with a better outcome in the absence of severe metabolic acidosis.(18,19)

Typically, arterial base deficit measured within the first hour of life is approximately 3 mmol/L higher than in arterial cord gases, including newborns who were not asphyxiated at birth. (20) Thereafter, the values return to normal. The arterial base deficit may increase by much more than this and persist longer in asphyxiated newborns whose umbilical arterial blood flow has slowed or stopped altogether, and no longer fully reflects fetal status. In nearly all severely asphyxiated newborns, perfusion at the time of birth is poor to nonexistent. Of course, poor perfusion includes

the umbilical circulation as well. The umbilical arteries will only reflect fetal tissue status up until the time flow in them stops. Lactic acid produced from hypoxia/anoxia at the tissue level will not be cleared to the central circulation and subsequently to the umbilical arteries. Therefore, in an asphyxiated newborn, an umbilical artery cord blood gas sample may seriously underestimate the acidosis in the fetus and newborn. As the infant is resuscitated, circulation improves, and tissue lactic acid is cleared into the central circulation (acid washout). Accordingly, a postnatal base deficit obtained from an asphyxiated newborn within the first hour after delivery is frequently found to be higher (worse) than in the umbilical arterial cord blood gas and is among the most accurate prognosticators of neurological outcome. (18)

Key Points

- Elevation of PCO_2 has not been associated with poor neurological outcome unless combined with metabolic acidosis.
- Low pH, combined with an elevated base deficit, has been associated with poor neurological outcome.
- Working definitions of respiratory and metabolic acidosis are presented. Nothing is known to happen clinically as a value crosses from one range of respiratory or metabolic acidosis into another.
- Typically, arterial base deficit increases by approximately 3 mmol/L in follow-up blood gases taken within the first hour of life, even in newborns who were not asphyxiated at birth.

References:

1. *Pomerance J: Interpreting Umbilical Cord Blood Gases, 2nd ed, Pasadena, 2012, pp 1-201 (www.cordgases.com)*
2. *Pomerance J. Umbilical cord blood gas casebook: Interpreting umbilical cord blood gases, Part I. J Perinat 1997;17:503-4.*
3. *Yeomans ER, Hauth JC, Gilstrap LC III, Strickland DM. Umbilical cord pH, PCO2, and bicarbonate following uncomplicated term vaginal deliveries. Am J Obstet Gynecol 1985;151:798-800.*
4. *Riley RJ, Johnson JWC. Collecting and analyzing cord blood gases. Clin Obstet Gynecol 1993;36:13-23.*
5. *Ramin SM, Gilstrap LC III, Leveno KJ, Burris JC, et al. Umbilical artery acid-base status in the preterm infant. Obstet Gynecol 1989;74:256-8.*
6. *Westgate J, Garibaldi JM, Greene KR. Umbilical cord blood gas analysis at delivery: A time for quality data. Br J Obstet Gynaecol 1994;101:1054-63.*
7. *Dickinson JE, Eriksen NL, Meyer BA, Parisi VM. The effect of preterm birth on umbilical cord blood gases. Obstet Gynecol 1992;79:575-8.*
8. *White CR, Doherty DA, Henderson JJ, Kohan R, et al. Benefits of introducing universal umbilical cord blood gas and lactate analysis into an obstetric unit. Aust N Z J Obstet and Gynaecol 2010;50:318-28.*
9. *Armstrong L, Stenson BJ. Use of umbilical cord blood gas analysis in the assessment of the newborn. Arch Dis Child Fetal Neonatal Ed. 2007;92:F430-4.*
10. *ACOG Committee on Obstetric Practice. ACOG Committee Opinion No 348: Umbilical cord blood gas and acid-base analysis. Obstet Gynecol, 2006;108:1319-22.*
11. *Weiner, CP. The relationship between the umbilical artery systolic/diastolic ratio and umbilical blood gas measurements in specimens obtained by cordocentesis. Am J Obstet Gynecol 1990;162:1198-1202.*
12. *Yoon BH, Kim SW. The effect of labor on the normal values of umbilical blood acid-base status. Acta Obstet Gynecol Scand. 1994;73:555-61.*
13. *Hagelin A, Leyon J. The effect of labor on the acid-base status of the newborn. Acta Obstet Gynecol Scand 1998;77:641-4.*
14. *Pryds O, Edwards, AD. Cerebral blood flow in the newborn infant. Arch Dis Child, 1996;74:F63-69.*
15. *Muller AM, Morales C, Briner J, Baenziger O, et al. Loss of CO2 reactivity of cerebral blood flow is associated with severe brain damage in mechanically ventilated very low birth weight infants. Eur J Paediatr Neurol, 1997;5:157-63.*
16. *Volpe JJ. Hypoxic-ischemic encephalopathy, Chapter 6, In: Neurology of the Newborn, 5th edition, Philadelphia, PA, Saunders Elsevier, 2008, p302-3.*
17. *Shankaran S, Laptook AR, Ehrenkranz RA, Tyson, JE, et al. Whole-body hypothermia for neonates with hypoxic-ischemic encephalo-pathy. N Engl J Med, 2005;353:1574-84.*
18. *Ambalavanan N, Carlo WA, Shankaran S, Bann CM, et al. Predicting outcomes of neonates diagnosed with hypoxemicischemic encephalopathy. Pediatrics 2006;118:2084-93.*
19. *Vannucci RC, Towflight J, Heitjan DF, Brucklacher RM. Carbon dioxide protects the perinatal brain from hypoxicischemic damage: An experimental study in the immature rat. Pediatrics 1995;95:868-74.*
20. *Weisbrot IM, James LS, Prince CE, Holaday DA, et al. Acidbase homeostasis of the newborn infant during the first 24 hours of life. J Pediatr 1958;52:395-403.*

Disclosure: The author has no disclosures.

NT

Corresponding Author

Jeffrey Pomerance, MD
Emeritus Professor of Pediatrics,
UCLA
Former Director of Neonatology,
Cedars-Sinai Medical Center, Los Angeles
Jeffrey Pomerance <jpomerance@msn.com>