Umbilical Cord Blood Gas and Acid-Base Analysis

ABSTRACT: Umbilical cord blood gas and acid-base assessment are the most objective determinations of the fetal metabolic condition at the moment of birth. Moderate and severe newborn encephalopathy, respiratory complications, and composite complication scores increase with an umbilical arterial base deficit of 12–16 mmol/L. Moderate or severe newborn complications occur in 10% of neonates who have this level of acidemia and the rate increases to 40% in neonates who have an umbilical arterial base deficit greater than 16 mmol/L at birth. Immediately after the delivery of the neonate, a segment of umbilical cord should be double-clamped, divided, and placed on the delivery table. Physicians should attempt to obtain venous and arterial cord blood samples in circumstances of cesarean delivery for fetal compromise, low 5-minute Apgar score, severe growth restriction, abnormal fetal heart rate tracing, maternal thyroid disease, intrapartum fever, or multifetal gestation.

Laboratory research demonstrates a complex relationship between fetal (antepartum and intrapartum) asphyxia, newborn asphyxia, and possible resulting brain damage. The degree, duration, and nature of the asphyxic insult are modulated by the quality of the cardiovascular compensatory response. A task force set up by the World Federation of Neurology Group defined asphyxia as a condition of impaired blood gas exchange, leading, if it persists, to progressive hypoxemia and hypercapnia (1). This is a precise definition of asphyxia as it may affect the fetus and neonate. In the American College of Obstetricians and Gynecologists’ Task Force on Neonatal Encephalopathy and Cerebral Palsy report, asphyxia is defined as:

... [a] clinical situation of damaging acidemia, hypoxia, and metabolic acidosis. This definition, although traditional, is not specific to cause. A more complete definition of birth asphyxia includes a requirement for a recognizable sentinel event capable of interrupting oxygen supply to the fetus or infant. This definition fails to include conditions that are not readily recognized clinically, such as occult abruptio, but is probably correct in a majority of cases. (2)

Asphyxia may occur in a transient fashion that, although of physiologic interest, has no pathologic sequelae. Significant fetal exposure to asphyxia.
leads to tissue oxygen debt, accumulation of fixed acids, and a metabolic acidosis. Thus, for intrapartum fetal asphyxia the following addition is proposed for this definition:

Fetal asphyxia is a condition of impaired blood gas exchange leading to progressive hypoxemia and hypercapnia with a significant metabolic acidosis. The diagnosis of intrapartum fetal asphyxia requires a blood gas and acid-base assessment. The important question for the clinician is what is the threshold of metabolic acidosis beyond which fetal morbidity or mortality may occur?

Low and associates have proposed a scoring system for predicting the likelihood of neonatal encephalopathy (3). They defined umbilical arterial base deficits at birth as mild at 4–8 mmol/L, moderate at 8–12 mmol/L, and severe at greater than 12 mmol/L. Newborn complications in the central nervous system, respiratory system, cardiovascular system, and kidney during the 5 days after delivery were documented. Assessment of the central nervous system included clinical evidence of newborn encephalopathy defined as minor with irritability or jitteriness, moderate with profound lethargy or abnormal tone, and severe with coma or abnormal tone and seizures. Cardiovascular complications were classified as minor with bradycardia (100 beats per minute or less) or tachycardia (170 beats per minute or more), moderate with hypotension or hypertension (defined by the 95% confidence limits for blood pressure in term neonates), and severe with continuous positive airway pressure or ventilation less than 24 hours, and severe if requiring mechanical ventilation more than 24 hours. Abnormalities of renal function were classified as minor if hematuria was observed, moderate with an elevation of serum creatinine level (greater than 100 mmol/L)*, and severe with anuria or oliguria (less than 1 mL/kg/h). A scoring system expressed the magnitude of the complications in each neonate. The score for each complication was “1” for minor, “2” for moderate, and “4” for severe. The maximum complication score was “16”. Moderate and severe newborn encephalopathy, respiratory complications, and composite complication scores were increased with an umbilical arterial base deficit of 12–16 mmol/L. Moderate or severe newborn complications occurred in 10% of neonates with this level of acidemia, increasing to 40% in neonates with an umbilical arterial base deficit greater than 16 mmol/L at birth. Low and associates concluded that the threshold of fetal metabolic acidosis at delivery associated with moderate or severe newborn complications was an umbilical arterial base deficit of 12 mmol/L and that increasing levels of metabolic acidosis were associated with a progression of the severity of newborn complications (3). At the mild base deficit range, there is no association with abnormal newborn outcome. A similar threshold for neonatal neurologic complications has been reported by other investigators (4, 5). Importantly, and in contrast to moderate or severe levels of acidemia, term neonates exposed to mild antepartum fetal asphyxia were not at an increased risk of minor motor or cognitive defects at the age of 4–8 years compared with controls with no evidence of asphyxia (6).

Term Infants

The prevalence of fetal asphyxia, ranging from mild to severe at delivery, in the term infant is reported at 25 per 1,000 live births; of these, 15% are either moderate or severe (3.75 per 1,000) (7). Even at these levels of acidemia, it must be appreciated that most fetuses will not be injured, yielding a final overall incidence of neonatal encephalopathy attributable to intrapartum hypoxia, in the absence of any other preconception or antepartum abnormalities, of approximately 1.6 per 10,000 (8, 9). Similar observations have been reported from Japan, where among a series of 10,030 infants there were nine cases of cerebral palsy at age 1 year or older diagnosed by pediatric neurologists. Analysis of these cases reveals that preexisting asphyxia existed before the initiation of fetal monitoring in six cases; two of the cases involved cytomegalovirus infections and one case involved a maternal amniotic fluid embolism (10). These investigators concluded that in low-risk pregnancies, cerebral palsy caused by intrapartum asphyxia was restricted to unavoidable intrapartum accidents.

Preterm Infants

Low and colleagues reported that the prevalence of asphyxia in preterm infants was 73 per 1,000 live births
births (7). Of these, 50% were at the moderate to severe level of asphyxia. The authors caution that it remains to be determined how often the asphyxia recognized at delivery may have been present before the onset of labor. This point is particularly germane in the preterm infant, inasmuch as medical or obstetric complications or both often are the preceding event necessitating the preterm delivery. Examples include significant degrees of intrauterine growth restriction, placental abruption, chorioamnionitis with funisitis, and severe preeclampsia, each of which has been shown to be a significant independent risk factor for moderate or severe neonatal encephalopathy (8, 9).

Acidemia and Cerebral Palsy
Both the International Cerebral Palsy Task Force and the American College of Obstetricians and Gynecologists’ Task Force on Neonatal Encephalopathy and Cerebral Palsy have published criteria to define an acute intrapartum event as sufficient to cause cerebral palsy (2, 11). Among the essential criteria cited by both task forces is evidence of metabolic acidosis in fetal umbilical cord arterial blood obtained at delivery (pH less than 7 and base deficit greater than or equal to 12 mmol/L) (see box). Additionally, the National Collaborating Center for Women’s and Children’s Health, commissioned by the National Institute for Clinical Excellence, has recommended that umbilical artery pH be performed after all cesarean deliveries for suspected fetal compromise, to allow review of fetal well-being and to guide ongoing care of the infant (12).

Technique for Obtaining Cord Blood Samples
Immediately after the delivery of the neonate, a segment of umbilical cord should be double-clamped, divided, and placed on the delivery table pending assignment of the 5-minute Apgar score. Values from the umbilical cord artery provide the most accurate information regarding fetal and newborn acid-base status. A clamped segment of cord is stable for pH and blood gas assessment for at least 60 minutes, and a cord blood sample in a syringe flushed with heparin is stable for up to 60 minutes (13, 14). If the 5-minute Apgar score is satisfactory and the infant appears stable and vigorous, the segment of umbilical cord can be discarded. If a serious abnormality that arose in the delivery process or a problem with the neonate’s condition or both persist at or beyond the first 5 minutes, blood can be drawn from the cord segment and sent to the laboratory for blood gas analysis. Analysis of paired arterial and venous specimens should prevent debate over whether a true arterial specimen was obtained. Therefore, the Committee on Obstetric Practice recommends obtaining an arterial umbilical cord blood sample, but, where possible, obtaining both venous and arterial samples (paired specimen). It is important to label the sample as either venous or arterial. Similarly, in known high-risk circumstances, such as severe growth restriction, an abnormal fetal heart rate tracing, maternal thyroid disease, intrapartum fever, or multifetal gestations, it is prudent to obtain blood gas and acid-base assessments (2). It should be noted that it occasionally may be difficult to obtain an adequate cord arterial blood sample. If the practitioner encounters difficulty in obtaining arterial blood from the umbilical cord (ie, in a very preterm infant), a sample obtained from an artery on
the chorionic surface of the placenta will provide accurate results (15). These arteries are relatively easy to identify because they cross over the veins.

**Conclusion**

Umbilical cord arterial blood acid-base and gas assessment remains the most objective determination of the fetal metabolic condition at the moment of birth. Thresholds have been established below which it is unlikely that an intrapartum asphyxial insult will have resulted in neurologic injury to the infant. Additionally, most infants born with umbilical arterial metabolic acidemia at a level consistent with causing a neurologic injury will, in fact, develop normally.

Physicians should attempt to obtain venous and arterial cord blood samples in the following situations:

- Cesarean delivery for fetal compromise
- Low 5-minute Apgar score
- Severe growth restriction
- Abnormal fetal heart rate tracing
- Maternal thyroid disease
- Intrapartum fever
- Multifetal gestations

**References**