REVIEW ARTICLE

Umbilical Cord Blood Gases: Sampling, Evaluation, and Application for Clinicians

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Abstract

Predicting the severity of birth asphyxia-related brain injury in newborn infants is a difficult task. Cord blood gases can be useful indices in the assessment of the impact of peripartum events. Cord blood gas parameters are particularly important because, despite all the progress in fetal monitoring, the time gap between the onset of fetal heart rate (FHR) abnormalities and birth asphyxia-related brain injury has remained difficult to predict. In this paper, we have focused on cord blood gas values in understanding the degree of compromise. These data can help determine the timing of fetal compromise prior to labor, and whether these precipitating events were acute or prolonged. When combined with some adverse clinical markers, the accuracy of low-cord pH in predicting neonatal mortality and morbidity can be even higher. Low-cord pH or eucapnic neonatal pH can also help in the surveillance of at-risk infants and in timely institution of neuroprotective therapies. We present a detailed review on sampling, evaluation, and application of cord blood gas values for clinicians.

Keywords: Arterio-venous difference, '20, 30, 40, 50 rule', Maternal hypoxemia, Base deficit, Birth asphyxia, Brain injury, Carbonic acid, Cerebral palsy, Cord blood gas, Eucapnic pH, pH qu 40, Hypercapnia, Hypoxic–ischemic encephalopathy, Maternal positioning, Neonatal encephalopathy, Nuchal cord, Organic acids, Oxygen-carrying capacity, Peripartum events, Placenta, Rectal temperature, Regional anesthesia, Respiratory acidosis, Stillbirth, Surveillance, Umbilical arteries, Umbilical venous blood, Vascular zone, Universal cord blood gas analysis.

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Key Points

- Cord blood gas values can be useful indices in the assessment of the impact of peripartum events.
- In combination with adverse clinical markers, cord pH can be a useful predictor of neonatal mortality; serial measurements of cord pH or eucapnic neonatal pH can help in the surveillance of at-risk infants.
- An uneventful first stage of labor can show a base deficit of 3 mM/L, which further drops at a rate of 1 mM/L per hour during the second stage of labor. In contrast, prolonged uterine contractions may be associated with fetal heart decelerations, and the base deficit may drop by 1 mM/L every 30 minutes.
- Low umbilical arterial pH <7 has been associated with a 10-fold higher risk of 1-min Apgar score of <4, 5-min Apgar score of <7, and 7.6-times higher risk of acute NE.
- Increasing information suggests that universal cord blood gas analysis can provide useful predictors of adverse neurocognitive outcomes.

Introduction

The umbilical cord is the critical conduit in the womb between the mother and her growing fetus; any insult or injury to this supply line can disrupt fetal oxygenation. In clinical units, the materno-fetal unit is routinely monitored for changes in maternal heart rate and blood pressure, fetal heart rate (FHR), FHR variability, and fetal movements during pregnancy and labor. Unfortunately, manual methods to monitor these are cumbersome and may not be cost-effective. To circumvent these difficulties, external and internal electronic fetal heart monitoring came into vogue and these are now the standard of care during labor.[1]

Recent years have brought increasing recognition that despite all the advances made in fetal monitoring, we still face a time lag between the detection of FHR abnormalities and the delivery of the infant. This sometimes jeopardizes fetal circulation and results in birth asphyxia. Measurement of cord blood gas values can help determine the degree of compromise and whether the precipitating event was acute or prolonged, and its timing prior to the onset of labor. Umbilical cord blood gas, a marker of neonatal vitality, is now increasingly accepted as the standard of care in all high-risk deliveries.[2] Hence, it is important for those who manage newborn babies to be familiar with the practice of obtaining and interpreting cord blood gas values.[3]

Physiology of Placental Circulation

The placenta is a vascular zone where gas/nutrient exchange between maternal and fetal circulation occurs. There is one large umbilical vein that carries oxygenated blood and nutrients to the fetus. Following tissue extraction of gases and nutrients, fetal blood returns to the placenta in two small umbilical arteries. Hence, umbilical artery blood primarily reflects fetal metabolism, while umbilical venous blood represents placental functions.[4] Normal fetal metabolism produces volatile (carbonic acid) and nonvolatile

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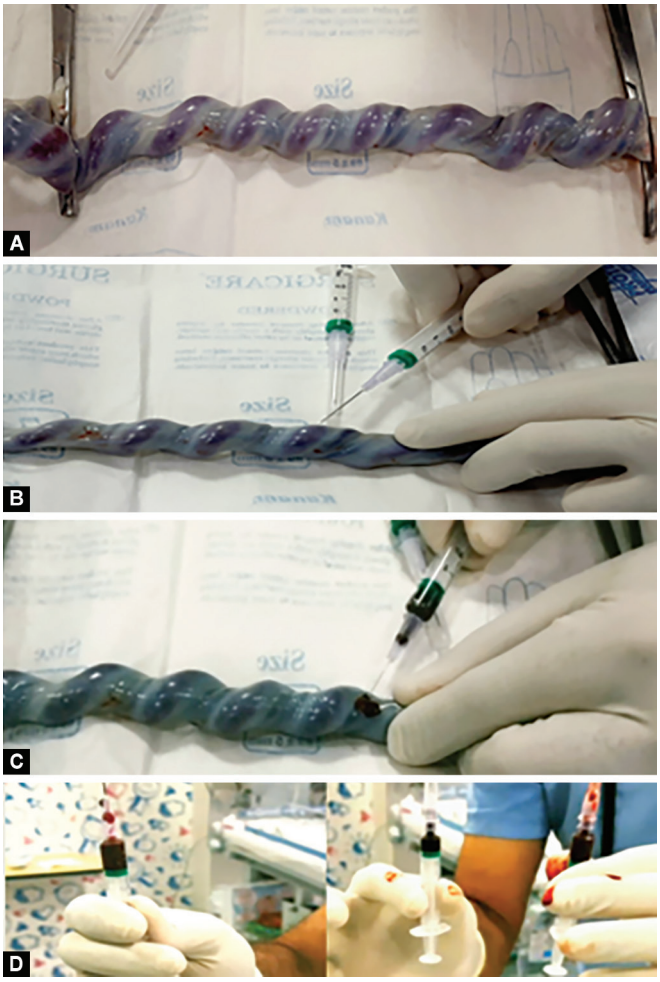

Figs 1A to D: Cord blood gas sampling procedure essentials. (A) Double clamp, a 20-cm long segment of cord. (B) Differentiate umbilical artery from vein Take 2 heparinized syringes for paired samples. (C) Sample umbilical artery first at 45°. (D) Do not contaminate the sample with air remove air bubbles, label both samples correctly transport and analyze within 30 minutes

A brief description of the umbilical cord blood gas sampling procedure can be viewed in the video available on its journal website at https://www.newbornjournal.org/

acids (lactate and organic acids). These are neutralized by buffer bases (bicarbonate and hemoglobin) to maintain the fetal pH within a narrow range.[5] However, unlike newborn infants, the fetus is unable to compensate for acidemia by respiratory and renal responses. The placenta can help maintain the bicarbonate pool, but biocarbonate depletion eventually manifests as base deficit.[6]

Cord Blood Gas Sampling Procedure

The accuracy of cord blood gas (CBG) analysis is improved by double clamping of the cord on either side to isolate a 20 cm-long portion, ideally before the baby's first breath. Clamping of the cord arrests the umbilical circulation; analysis of a sample drawn soon thereafter gives an estimate of the acid–base status of the infant at the time of birth. However, if the cord is not clamped to isolate its blood circulation from that of the placenta, the ongoing placental metabolism may alter the acid–base status in cord blood.[7] There may be a progressive decline in pH and base excess, increase in pCO_2 and lactate; these changes have been described as "hidden acidosis", which might just reflect a transient effect of initiation of neonatal breathing and give a false impression of significant acidosis at birth.[8] In other cases, cord blood gas values may remain unchanged for up to an hour after isolating the cord from maternal/neonatal circulation.

In most infants, umbilical arterial blood gas values may represent fetal metabolism, and if abnormal, may predict neonatal morbidity. In many cases, blood drawn from a relatively superficial umbilical vein can show less-accurate values. Hence, both arterial and venous blood should be sampled. Blood can be drawn into preheparinized syringes by needle aspiration, taking precautions to exclude air bubbles before capping the syringe, and should be analyzed within 30 minutes of sampling (Fig. 1). Contamination of the syringe with air has no effect on pH, pCO_2, or bicarbonate but can significantly elevate pO_2 values when there is more than 37.5% residual air in the syringe.[9–11] The validation of paired (arterial and venous) sample is based on minimum arterio-venous (A-V) difference for pH and pCO_2. The pH difference should be >0.02 units, and the PCO_2 difference should be >3.75 mm Hg. If the minimum A-V difference is documented, it can be safely assumed that the two samples are from different vessels, and the one with lower pH and higher pCO_2 is from the artery.[12] With the practice of placental transfusion gaining universal acceptance, concerns about

its effects on cord blood gas seem unfounded even after delayed cord clamping of up to 2 minutes.[13]

Interpretation of Cord Gas Values in Practice

In clinical practice, the following key questions need to be considered about CBG values:

(A) Normal Values

In CBG analysis, pH, PCO_2, and PO_2 are measured, whereas base deficit (BD) is calculated. The normal ranges for umbilical pH and blood gases which are broad and overlapping (shown in Table 1). It is often difficult to assign it to the umbilical artery or vein.[14,15] Ross and Gala.[16] recommend using the "20, 30, 40, 50 rule" as a simple tool for remembering normal CBG values (Table 2). Umbilical arterial PO_2 is typically lower than umbilical venous PO_2, at 20- and 30-mm Hg, respectively. PCO_2 value is higher in the umbilical artery than the umbilical vein, thus, 50 mm Hg denotes umbilical artery value, and 40 mm Hg indicates the umbilical vein value.

A normal uneventful first stage of labor leads to a base deficit of 3 mM/L, which further drops at a rate of 1 mM/L per hour during the second stage of labor. In contrast, prolonged uterine contractions may be associated with fetal heart decelerations, and the base deficit may drop by 1 mM/L every 30 minutes. The most severe fetal compromise, as seen in sudden cord compression or uterine rupture, may increase the base deficit by 1 mM/L every 2–3 minutes. In these situations, the obstetric team may have only 10–15 minutes to deliver the baby.[17]

pH_{UA} is typically lower than the umbilical vein pH, reflecting its higher PCO_2 and lactate levels. As the base deficit increases during labor, the pH falls. Brief rise in CO_2 levels (respiratory acidosis) may cause some fluctuations in pH, but this does not usually cause a significant insult and normalizes within a few minutes.[18]

Table 1: Normal cord blood gas values

	Umbilical artery	*Umbilical vein*
pH	7.18–7.38	7.25–7.45
PO_2	5.6–30.4 mm Hg	17.4–41.0 mm Hg
pCO_2	32.4–66.0 mm Hg	27.0–49.4 mm Hg
BD mean (SD)	4.79 (3.46) mmol/L	4.0 (3.5) mmol/L

Table 2: The "20, 30, 40, 50 rule" cord blood gases rule

Value mm Hg	*Cord gas value depicted*
20	Umbilical artery PO_2
30	Umbilical vein PO_2
40	Umbilical vein PCO_2
50	Umbilical artery PCO_2

(B) Factors Affecting Fetal Acid–Base Metabolism

Several factors have been associated with impaired fetal oxygenation and cord blood metabolic acidosis (Table 3). Maternal hypoxemia (respiratory diseases, seizures) and reduced oxygen-carrying capacity (anemia) may result in low maternal, and consequently, low fetal PO_2. Decreased uterine blood flow due to hypotension (shock, sepsis, regional anesthesia, and maternal positioning) and impaired perfusion on the maternal side of the placenta (abruptio) can be seen as narrowed arterio-venous differences in CBGs.[19] However, partial or complete restriction of umbilical blood flow (cord prolapse or entanglement, nuchal cord) may be seen as progressive widening of the umbilical artery and venous blood gas values. An arterio-venous pH difference of more than 0.15 units can differentiate reliably between cord prolapse and placental abruption. By virtue of being thin walled, during cord compression, the umbilical vein is significantly more compressed as compared with the umbilical arteries, reducing the supply of blood from the placenta to the fetus. A hypoxic fetus extracts most of the oxygen and generates more CO_2, making the arterial pH more acidotic, while umbilical venous pH is maintained by a normally functioning placenta.[20] Apgar scores and umbilical venous samples in such infants alone may be misleading, and it is crucial to confirm the diagnosis by documenting acidosis in the umbilical arterial blood.[21]

Rarely, despite severe intrapartum asphyxia in cord accidents (prolapse, true knot, and tight nuchal cord), the cord blood gas

Table 3: Factors affecting fetal acid base metabolism

Maternal factors	*Utero-placental factors*	*Fetal factors*
Maternal hypoxia	Uterine hyperstimulation	Umbilical cord factors
• Respiratory diseases	• Oxytocic drugs	• Oligohydramnios
• Respiratory depression	• Prolonged labor	• Cord prolapse/compression
• Trauma, seizures	• Instrumental delivery	• Nuchal cord
• Smoking	• Placental abruption	• Knots/entanglement
Maternal reduced oxygen-carrying capacity	Utero-placental dysfunction	Decreased fetal oxygen-carrying capacity
• Anemia	• Abruptio placenta	• Anemia: isoimmunization
• Carboxy hemoglobin	• Infarction	Vasa previa
	• Dysfunction: reversal or reduction of flow	• Carboxy hemoglobinemia
	• Premature rupture of membranes	• Twin-to-twin transfusion
Reduced perfusion of placenta	Uterine infection	
• Hypotension (shock/sepsis)	• Chorioamnionitis	• Shoulder dystocia
• Regional anesthesia		• Congenital malformations
• Maternal positioning		• Genetic abnormalities
• Preeclampsia		• Errors of metabolism
• Diabetes		• Cardiac abnormalities
• Heart disease		• Multiple births

values may be absolutely normal. Isolated cord segment on account of complete obstruction of the umbilical vessels reflects blood gas values unaffected by anaerobic fetal metabolism. On restoration of circulation, blood gas taken after 30 minutes reveals true disturbed fetal metabolism. In cases with intrapartum fetal death (fresh stillbirth), normal cord gases suggest sudden fetal cardiac arrest.[22] Infants born by elective cesarean section and those born to multiparous mothers have CBG values closer to normal adult values.[23] In twin deliveries, the second twin exhibits a time-bound precipitous fall in cord pH.[24] Abnormal umbilical cord morphology, length, vascular coils per centimeter, and true knots seem to have a very weak correlation with low-cord pH.[25,26] Although placental infection like chorioamnionitis with or without funisitis is associated with cerebral palsy, the mechanism appears independent of a hypoxic–ischemic insult.[27] Helwig et al.,[28,29] in their study of 15,000 vigorous newborn infants, showed that the cord arterial pH dropped more in term/post-term than in preterm infants. This trend was attributed to a shorter duration of labor in those born prior to term and increased placental oxygen consumption with advancing maturity.

(C) Evaluation of Fetal Hypoxia and Acidosis

Cord blood gas provides the most accurate and objective evidence of asphyxia at the time of birth. It complements routine clinical assessment of the baby at birth using the APGAR scoring system.[16,30] Cord blood gas is an indicator of fetal oxygenation – a measure of fetal well-being and perhaps an indicator of the quality of obstetric care. Deranged cord gases suggest possible birth asphyxia, a term that summarizes a lack of pulse, oxygen supply, and carbon dioxide washout. One can question about the mechanisms by which the fetus can thrive in the relatively hypoxic state *in utero*, but the predominance of fetal hemoglobin, Bohr's effect, and the still state of the fetus are plausible protective factors. Low umbilical artery pH (pH_{UA}) is an established marker of hypoxia and can help in early identification of asphyxia. The umbilical artery base deficit (BD_{UA}) may be a more linear measure of the accumulation of metabolic acids. Cord blood metabolic acidosis differs from cord blood respiratory acidosis, which results in only minimal reduction in pH with no base deficit.[31] Isolated respiratory acidosis at birth suggests an impaired gas exchange of short duration, resulting in minimal hypoxia and moderate hypercapnia. It is a relatively transient state of little significance that resolves soon after the newborn starts to breathe. If hypoxia is prolonged, the ensuing anaerobic metabolism produces lactic acidosis, which neutralizes the buffer bases, ultimately resulting in metabolic acidosis and base deficit.[32–34]

Hypoxic–Ischemic Encephalopathy as a Cause of Neonatal Encephalopathy

Cord arterial pH <7.0 and base deficit >12 mM/L is considered as reflecting significant neonatal metabolic acidemia and is seen in around 1% of all childbirths. This degree of fetal acid–base imbalance is associated with risk of hypoxic brain cell injury and consequent hypoxic–ischemic encephalopathy (HIE) and its long-term complications, such as cerebral palsy and neurodevelopmental abnormalities.[35]

Newborn infants who present with poor feeding, respiratory difficulties, seizures, tone abnormalities, and altered levels of consciousness are described as having neonatal encephalopathy (NE).[36] Neonatal encephalopathy is just a clinical condition that can result from several antenatal and/or perinatal factors. The most common causes of NE are HIE, perinatal infections, placental abnormalities, metabolic disorders, coagulopathies, and neonatal vascular stroke. However, in more than 50% of cases of NE, the cause remains unidentified.[37] The interchangeable use of the terms, HIE and NE, is controversial, as we often do not know when hypoxia–ischemia is indeed the cause of NE. It is proposed that in symptomatic newborns with no identifiable sentinel event, the term NE may be used, as we do not need etiologic labels for disease entities. All current parameters like pH, base deficit, and seizures are non-specific. The pattern of brain injuries produced by hypoxia–ischemia also does not prove that all NE is caused by HIE. Hence, for medico-legal clarity, attributing a cause (hypoxia–ischemia) to the disorder (encephalopathy) without documenting reduced cerebral blood flow should be avoided.[38,39] The incidence of NE is 1/10th (1.5/1,000 live birth) of significant cord blood acidosis (1/100 live births). Also, NE is not an inevitable consequence of significant fetal acidosis; nearly 75% of these acidotic babies do not show any neurological signs of NE. However, a diagnosis of HIE as the cause of NE in part depends upon demonstrating significant cord blood acidosis. A normal cord arterial pH and base deficit usually eliminate asphyxia as a cause of neurological manifestations in a newborn. In fact, the incidence of asphyxia contributing to cerebral palsy is relatively small at 10% of all causes.[40]

In 1990, a multidisciplinary International Cerebral Palsy Task Force set out criteria for retrospective labeling of an intrapartum hypoxic event as sufficient to cause cerebral palsy. These criteria were then updated in 2003 by the Task Force on Neonatal Encephalopathy and Cerebral Palsy, a joint effort of the American College of Obstetricians and Gynecologists and the American Academy of Pediatrics, and were further modified in the year 2014 (Table 4).[41,42] Based on these criteria, the Joint Task Force Committee suggested that the pathway from intrapartum hypoxic–ischemic injury to subsequent cerebral palsy must progress through NE. Any neurological damage, including cerebral palsy, cannot be attributed to birth asphyxia in the absence of NE. Past assumption of HIE causing all NE was misplaced and was the reason behind this changed approach to search for other genetic, metabolic, and developmental causes contributing to neurological damage. This also has medico-legal significance in the court of law in resolving disputes related to brain damage sustained during childbirth.[43] This approach was released as a safety document meant to identify some of the above factors operating even before the onset of labor, where NE can be anticipated and minimized by offering therapeutic hypothermia.[42]

Therapeutic hypothermia (controlled cooling of infants >36 weeks to a rectal temperature of 34°C for 48–72 hours) is the only effective neuroprotective intervention in asphyxiated babies, and its efficacy in a baby with HIE depends upon initiating it within 6 hours of birth. A cord blood pH <7 and a base deficit >16 mM/L is one of the inclusion criteria for the application of this therapy.[44]

Pathological Acidosis and Neurological Outcome

Acidosis is generally well-tolerated by the fetus without adverse neurological sequelae until it reaches a pathological threshold. A practical pH threshold for significant neonatal academia is an umbilical artery pH <7, a value below which adverse clinical events begin to be seen frequently.[45] Evidence in support of this threshold value was derived from a 2010 meta-analysis of 51 cohort and case-controlled studies of over 480,000 infants, indicating a

Table 4: International consensus criteria to determine a severe acute hypoxic event as a potential cause of cerebral palsy

(A) Essential criteria (all mandatory)

- Metabolic acidosis CBGUA (pH<7 and BD 12 mmol/L or more).
- Onset of moderate-to-severe neonatal encephalopathy within 72 hours of birth in a baby 34 weeks gestation or more.
- Cerebral palsy of spastic quadriplegic or dyskinetic type.
- Exclusion of other known causes of cerebral palsy, e.g., genetic disorders, infections, intrapartum fever, antepartum hemorrhage, prematurity, growth-restricted babies, tight cord around the neck and as a result of complication of multiple pregnancy.

(B) Nonspecific criteria that collectively suggest a significant intrapartum acute or chronic insult

- A sentinel event severe enough to cause sudden hypoxia in a healthy fetus occurring immediately before or during labor, e.g., cord prolapse, antepartum hemorrhage, and ruptured uterus.
- A sudden and sustained fetal bradycardia from that sentinel event.
- Apgar scores of less than 4 beyond 5 minutes.
- Onset of multisystem organ involvement within 72 hours of birth.
- Early imaging study (within 5 days) showing evidence of edema and intracranial hemorrhage.

strong association with neonatal mortality at pH <7 and neonatal morbidity at pH <7.1.[16,46,47] In a cohort study of 8,700 term infants, pH_{UA} <7 was a strong predictor of all adverse outcomes, including NE or death. These outcomes were seen in 2.3% of all acidemic babies and 8.5% of infants with severe acidemia.[48]

Most infants with a cord pH <7 who appear well at birth and are free of any cardiovascular compromise do not require admission in the neonatal unit or extensive investigation simply based on low-cord pH. These babies did not develop neurological problems after birth, even when followed for 6.5 years.[49] Universally, a cord pH_{UA} <7 is considered significant neonatal metabolic academia and if associated with other short-term markers, is a reliable indicator of acute peripartum events and a good predictor of adverse neurocognitive outcomes.[46,50–52] Cord pH is widely used as an outcome measure in obstetric clinical trials and is a measure of the quality of obstetric care.[53] However, based on the cord pH value without clinical signs and symptoms of NE, there is substantial uncertainty in the management and neurocognitive prognosis of neonates.[53]

Neonates with pH_{UA} <7 have shown 10-fold higher risk of 1-min Apgar score of <4, 5-min Apgar score of <7, and 7.6-times higher risk of acute NE. Also, multiparous women had higher risks of low Apgar score and NE as compared with nulliparas.[54] Shoulder dystocia and women with urological problems had a statistically higher risk of delivering a baby with a low 5-min Apgar score.[34] In their series of 3506 infants, Goldaber et al.[55] observed neonatal death to be more likely at pH <7, and seizures as more likely at pH <7.05. Worsening acidosis predisposed to increased morbidity due to HIE with 12% of infant being symptomatic at cord pH <7 to 80% at cord pH <6.8%. No infants were born alive at pH <6.6.[34,56] Using a scoring system for renal, central nervous system, and respiratory and cardiovascular morbidity, Low et al.[57] showed that significant acidosis (base deficit >12 mM/L) was a good predictor of multi-organ involvement.

As an isolated finding, neonatal metabolic acidemia is a poor predictor of the risk of HIE. However, when combined with other clinical parameters such as non-reassuring fetal heart tracings, need for continued resuscitation/intubation beyond 5 minutes, low 5-min Apgar score, early occurrence of seizures, and development of moderate HIE, the risk of neuro-cognitive impairment is strong.[58,59] Cord blood lactate levels are an additional parameter included in numerous new-generation blood gas analyzers. Lactate, a product of anaerobic metabolism, barely diffuses across the placenta and is the main metabolite responsible for fall in cord pH, and base buffers.[60] Considering its fetal origin, lactate should be a much more reliable predictor of poor neonatal outcomes than other tests such as the cord arterial pH and base deficit.[61,62] The presence of high lactate levels >4.1 mM/L and lactate/pyruvate ratios <22 is predictive of neonatal encephalopathy with a 100% sensitivity and 95% specificity.[63]

In a study of 4910 term infants, high mean lactate levels (6.49 mmol/L compared with 3.26 mM/L, $p < 0.001$) correlated well with composite neonatal morbidity (1.1%) as compared with pH (7.19 compared with 7.29, $p < 0.001$).[64] High umbilical cord lactate is thus a reliable method to quantify acidosis and is more prognostic of neonatal morbidity than pH.[65,66] In 2014, the American College of Obstetricians and Gynecologists' Task Force on Neonatal Encephalopathy[42] stated: "To elucidate the different causal pathways leading to HIE and CP, it is important that a reliable and readily available assessment of fetal status is possible and that more specific markers of an intrapartum insult be developed." The existing association between cord arterial pH and/or BD and short-/long-term neurocognitive outcomes reveals that traditionally accepted cutoff values to define significant neonatal acidemia still need more investigations.[67,68]

Sailing and Schmidt[69] and Sigaard-Andersen[70] noted very high pCO_2 levels, much higher than those seen in adults, in neonatal acidemia. These respiratory abnormalities usually corrected with appropriate ventilation. Hence, it is crucial to identify respiratory acidosis in the neonatal period from NMA to select newborns with mixed or predominantly metabolic acidosis to be admitted in the NICU for close monitoring and treatment. Scientists have now proposed calculated neonatal eucapnic pH (pH euc-n), which includes nonrespiratory pH, reduced pH, standard pH, or pH qu 40 (base required to bring the pH to 7.40 and pCO_2 at 40 mm Hg), as reflecting the metabolic component of pH due to nonrespiratory acid–base balance as a specific marker for neonatal metabolic acidosis (NMA) rather than currently used biomarkers (pH or base deficit).[71] A pH euc-n > 7.11 derived after appropriate correction of hypercapnia reconciled to the fetal *milieu interior* predicts that the infant will likely remain asymptomatic and have an uneventful course. p-euc-n values < 7.11 are more often associated with NE. pH euc-n is easy to calculate at the bedside (Blickstein method) by adding 0.08 to ua pH value for every 10 mm Hg rise of pCO_2 above the threshold of 50 mm Hg from the routine blood gas reports and provides valuable objective defense for obstetricians in allegations of professional liability.[72] It also informs neonatologists about the need for therapeutic hypothermia.[69]

Cord Blood Gas Analysis: Selective or Universal

Universal cord blood gas analysis (UCBGA) was first advocated 60 years back to assess the metabolic condition of a newborn infant at birth. The costs of UCBGA are justified as these can reduce the need for neonatal ICU admissions. Considering the temporal relationship between cord blood acidemia and adverse neurocognitive outcomes, many international associations have recommended UCBGA. This transition from selective to UCBGA has reduced the frequency of cord arterial acidemia and improved perinatal outcomes. These changes in obstetric practice have improved fetal/neonatal outcomes. The financial costs and emotional burden arising from perinatal hypoxic–ischemic injuries (cerebral palsy) provide a much more compelling societal perspective than the added cost of UCBGA.[73] Thorp and Rushing.[74] have summarized their views on the pros and cons of selective vs UCBGA.

Advantages of UCBGA

A cord blood value will be available for all babies who become symptomatic sometime after birth (crucial for management of a baby and future medico-legal disputes).

- The team becomes adept at sampling and processing the CBG sample.
- Omission to perform a CBG in an emergency is avoided.
- Provides insight into the interpretation of electronic fetal heart monitoring for safe and effective intervention strategies.

Disadvantages of UCBGA

- Added costs to obstetric care;
- Need for additional staff that might not be available at all units;
- A low-cord pH in a vigorous newborn baby might pose a potential medicolegal concern because it falsely suggests birth asphyxia.

Universal cord blood gas analysis can be used to audit the quality of fetal monitoring and intervention strategies used by the obstetric team to prevent significant fetal acidemia and its associated neonatal morbidity and mortality.[53]

Conclusion

Cord blood gas analysis is frequently used at birth to assess the effects of peripartum events in a newborn. Neonatal and childhood neuro-cognitive morbidities, including cerebral palsy, are often attributed to fetal acidemia. Existing associations between cord pH and adverse outcomes are conflicting. Low-cord pH along with some adverse clinical markers reliably predicts neonatal mortality and morbidity. Hence, it is justified to enhance surveillance of infants born with a low-cord pH or, more specifically, neonatal eucapnic pH so that those who fulfill the required criteria be offered various neuroprotective therapies. The obstetric and neonatal teams should be well-versed with the CBG sampling procedure and its evaluation. Future studies should assess the use of CBG across neonatal populations and explore the cost-effectiveness of universal screening. Meanwhile, the search for an absolute reliable marker of NMA should be the focus for future research.

Author's Contributions

- Substantial contributions to the conception or design of the work; or the acquisition, analysis, or interpretation of data for the work: MH, IK, and SG.
- Drafting the work or revising it critically for important intellectual content: MH and IK.
- Final approval of the version to be published: MH.
- Agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved: MH.

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