OBSTETRICS

The significance of base deficit in acidemic term neonates

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OBJECTIVE: Much emphasis is placed on the metabolic component of umbilical cord acidemia at birth, with an importance attached to an arterial level of <7.00 accompanied by a base deficit of 12 mmol/L. We hypothesized that in acidemic neonates, the level of arterial base deficit provides no prognostic information beyond that provided by the level of arterial pH.

STUDY DESIGN: This is a cohort study using a database of deliveries from a major teaching hospital, with additional information from neonatal records. A total of 8797 term, singleton, nonanomalous neonates were identified who had paired and validated cord blood gas analysis. Of these, 520 were acidemic (pH <7.1) and 84 were severely acidemic (pH <7.0). Outcomes examined were encephalopathy grade 2/3 and/or death, Apgar <7 at 5 minutes, neonatal unit admission, and composite outcomes of neurological and systemic involvement. Hierarchical logistic regressions were done using IBM SPSS Statistics 20.0 (Armonk, NY) to assess the predictive value of arterial pH and arterial base deficit.

RESULTS: For each outcome the median pH and base deficit of those neonates affected by the adverse outcome was significantly lower than for those who were unaffected. Hierarchical logistic regressions showed that pH is a significant predictor of all adverse outcomes studied ($P < .001$ for all outcomes). When base deficit, and then the cross-product, are added to the model, neither add predictive value.

CONCLUSION: In acidemic neonates, the metabolic component does not predict those at risk of adverse outcomes once pH is taken into account. The apparently worse outcomes with greater base deficit simply reflect a greater degree of acidemia. The prognostic significance attached to the base deficit among acidemic neonates is questionable.

Key words: adverse neonatal outcome, base deficit, metabolic acidosis, pH, umbilical cord

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Intrapartum hypoxia is an important and high-profile precursor of cerebral palsy and neonatal death. Assessment of its severity at birth is currently performed by analysis of umbilical vessel gas parameters, particularly pH and base deficit in the umbilical artery.

An accepted "threshold" of arterial pH (ApH) is commonly quoted as <7.00, although a recent analysis of >50,000 consecutive cord samples suggested that it is at 7.1 that the risk of encephalopathy, an accepted precursor to cerebral palsy of intrapartum origin, begins to rise.[1] The arterial base deficit (ABD) threshold has been quoted as 12 mmol/L and Low et al[2] found increasing base deficit values to be associated with higher complication rates.

It is widely believed that the metabolic component is crucial. Fetal asphyxia is defined as "a condition of impaired gas exchange leading, if it persists, to progressive hypoxemia and hypercapnia with significant metabolic acidosis."[3] In 1999, the International Cerebral Palsy Task Force stated that cord blood metabolic acidosis (ApH <7.00, base deficit ≥12) is an essential requirement before cerebral palsy can be attributed to intrapartum events[4]; the American Congress of Obstetricians and Gynecologists alone requires the ABD to be at least 12 mmol/L.[5] Further, the base deficit has been used as an outcome measure in trials examining intrapartum interventions[6,7] and as a criterion for cooling in the Total Body Hypothermia for Neonatal Encephalopathy trial.[8]

The aim of this study is to examine the relationship among ApH, base deficit, and outcome in acidemic neonates. Our hypothesis is that, in acidemic neonates, base deficit does not add to the predictive value of ApH.

Materials and Methods

Dataset

This was an observational cohort study of nonanomalous term singleton deliveries where complete and validated cord blood gas analyses were recorded.

Details of all deliveries at a major teaching hospital, including cord gas analysis if performed, were prospectively recorded in a database. Cord gases are taken at approximately half of all deliveries. These are manually entered after birth: so to avoid potential transposition errors, we merged outcome data, using hospital number and date, with the output from the unit blood gas analyzer.

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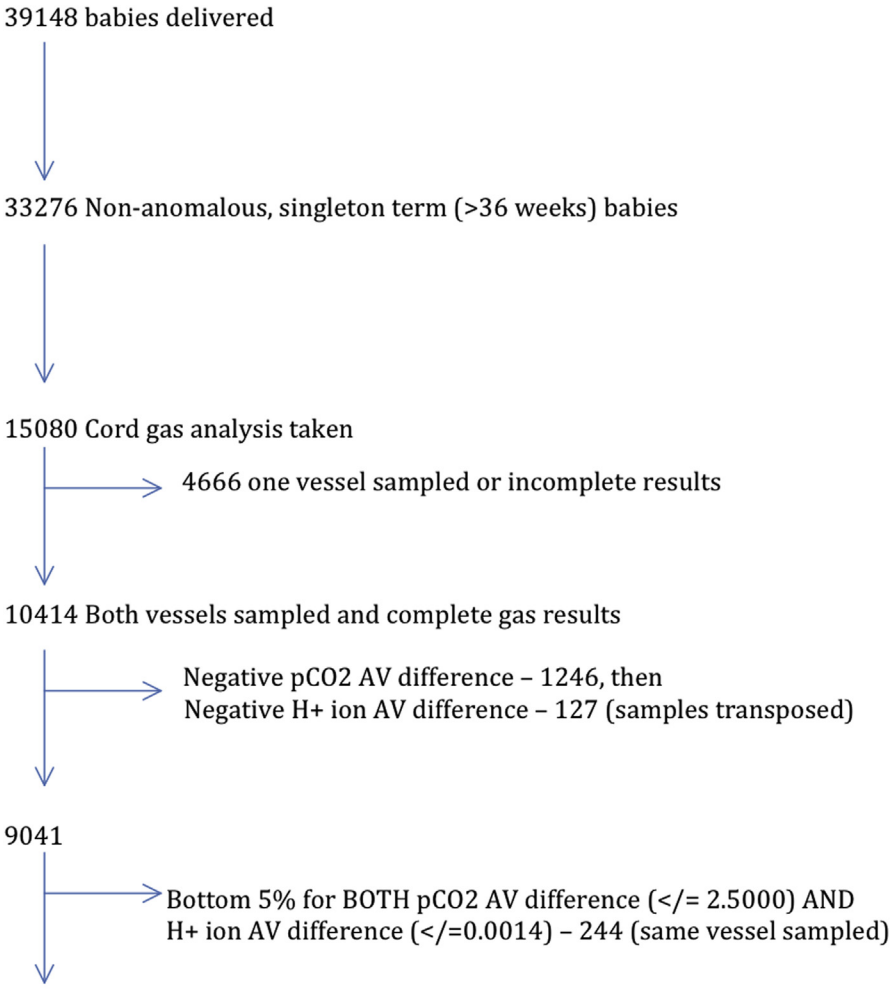
FIGURE
Summary of exclusions



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Of appropriate deliveries from June 23, 2005, through Dec. 31, 2009, where cord samples were taken, we excluded all where only 1 vessel was sampled or any values, eg, bases deficit, pH, or partial pressure of carbon dioxide (pCO2), were missing. We then excluded unphysiological values, ie, where the arteriovenous (AV) pCO2 or hydrogen ion (H+) difference was negative. We also excluded unreliable results where samples might be contaminated, or the same vessel sampled twice, with an adaptation of Westgate and Greene.[9] We excluded cases in the lowest 5% for both pCO2 AV difference ($\leq$2.5000) and H+ ion AV difference ($\leq$0.0014). We used hydrogen ion difference, since excluding by pH difference preferentially excludes those results with a low pH and a narrow AV difference because pH is a logarithmic number. Finally we limited our analysis to moderately acidemic (ApH $<$7.1 and $\geq$7.0) and severely acidemic (ApH $<$7.00) cohorts. The Figure shows a summary of exclusions.

As the object of this study was simply to examine the relationship between ApH and ABD as risk factors for adverse outcomes, we did not confine analysis to babies delivered after labor, or examine other potential risk factors for adverse outcomes, such as birthweight.

Cord gas analysis

Unit policy is that paired cord gases should be taken if there has been electronic fetal monitoring or meconium, or at midwife or doctor discretion, eg, low Apgar scores. The sampling rate for the period was 45%. The cord is double-clamped immediately after delivery at a minimum length of 10 cm with the placenta in situ. Arterial and venous samples are taken in preheparinized labeled syringes and processed within 15 minutes in a Radiometer ABL800 blood gas analyzer (Radiometer Medical ApS, Brønshøj, Denmark). Radiometer reports base excess (BE) in 2 compartments: blood and extracellular. As the more physiological value, arterial extracellular fluid BE (ABE[Ecf]) was the measure used. Unfortunately the values of ABE(Ecf) were not calculated by the analyzer for the year 2005. For consistency of language the sign was changed and base deficit values used. For the entire cohort the values of base deficit (Ecf) were calculated from pH and pCO2. Firstly bicarbonate (HCO_3) was calculated using the Henderson-Hasselbalch equations ($HCO_3 = 0.03 \times pCO_2 \times 10^{[pH-6.1]}$). These values were then entered into the algorithm used by Westgate and Greene[9] to calculate base deficit (Ecf).

Outcome measures

Data on neonatal events were initially assessed using *International Statistical Classification of Diseases* codes of all babies. Neonatal records, discharge summaries, and imaging reports of babies admitted to the neonatal unit prior to maternal discharge were inspected.

We selected outcome measures in conjunction with a neonatologist to reflect clinical condition at birth, neurological involvement, and/or multisystem involvement. We used encephalopathy (grade 2 and 3) and/or death as well as more frequent outcomes of neonatal unit admission and Apgar score $<$7 at 5 minutes as the main outcome measures. We also developed composite outcomes to identify babies with multisystem involvement. The composite neurological outcome included those with $\geq$1 of: