

# The case for the routine use of Umbilical Cord pH in all deliveries

Dr Ismail Borat<sup>1</sup>, Dr Lou Pistorius<sup>2</sup>, Prof Priya Soma-Pillay<sup>3</sup>, Prof Izelle Smuts<sup>4</sup>

<sup>1</sup>Subdepartment of Fetal Medicine, Department of Obstetrics and Gynaecology, University of Kwa-Zulu Natal, South Africa

<sup>2</sup>Department of Obstetrics and Gynaecology, University of Stellenbosch and University Medical Centre Utrecht, Netherlands

<sup>3</sup>Department of Obstetrics and Gynaecology, University of Pretoria, Pretoria, South Africa

<sup>4</sup>Department of Paediatrics, Paediatric Neurology Unit, University of Pretoria, Pretoria, South Africa

Cerebral palsy (CP) cases are some of the primary reasons for litigation of high quantum amounts against obstetricians both in the public and private sector, on the basis of acute intrapartum hypoxia resulting in hypoxic ischaemic foetal brain damage and by extension invoking “negligent intrapartum care”. This has resulted in a steep rise of insurance premiums<sup>1</sup> placing service delivery in both the public and private sector under a serious threat. This is a worldwide phenomenon and MacLennan already expressed his concerns in 2005 by asking: ‘Who will deliver our grandchildren?’.<sup>2</sup> It has widely been believed that CP is the direct result of an adverse event at birth and that it could have been prevented, but <10% of CP is caused by “birth asphyxia”.<sup>3</sup> There are multiple risk factors and causes now identified to be associated with CP.<sup>4,5</sup>

Furthermore the terminology ‘birth asphyxia’ and ‘hypoxic ischaemic encephalopathy’ are both outdated and should be replaced by neonatal encephalopathy (NE).<sup>5</sup> The objectives of umbilical cord blood assessment is to assess the infant’s stress during labour and to add additional information to the Apgar scores regarding the status of the newborn.<sup>6</sup> Umbilical cord acid-base assessments have also been used as a measure of quality of care, to guide hypothermia treatment decisions and provide information for medico-legal purposes.<sup>7</sup> To determine the likelihood that NE has been caused by an acute intrapartum hypoxic-ischaemic incident, well-defined international consensus criteria have been published and reviewed.<sup>5,8</sup>

At present the obstetrician is vulnerable to these types of claims as the only defence the obstetrician has at his or her disposal is the cardiotocogram (CTG),

clinical notes and the Apgar score. As the CTG is a subjective assessment, different opinions in interpretation are often obtained with different experts and biased expert witnesses may often manipulate these subjective interpretations. Furthermore it has a very high false positive rate, having been introduced in the sixties without prior testing with the hope that it would prevent cerebral palsy.<sup>1,5</sup> The rate of caesarean sections has increased six-fold over the past 50 years without a decrease in the CP prevalence.<sup>5</sup> Clinical notes are often problematic as they are often inadequate, non-exhaustive, “lost” or “misplaced” which in effect removes any defence for the obstetrician. The Apgar score is a semi-quantitative measurement that has some subjectivity with variable intra-observer reliability.<sup>9,10</sup> Although a low Apgar score is often used as a proxy for acidosis, it was not primarily designed for this aim.<sup>11</sup>

The question is whether biochemical parameters could be assessed in the immediate postpartum period that could adequately reflect the intrapartum process. Parameters that could be used in this regard include arterial and venous cord blood pH, base excess, lactate, pO<sub>2</sub> and pCO<sub>2</sub>. Umbilical cord-blood acid-base analysis provides an objective assessment of newborn metabolic status. Umbilical cord arterial pH falls and base excess decreases (deficit increases) when hydrogen ions from anaerobic metabolism overwhelm the buffer capacity of the fetus.<sup>12</sup> Lactate is a direct end product of anaerobic metabolism. Under hypoxic conditions glucose is broken down to pyruvate that is converted to lactate and hydrogen ions. The foetus is the key contributor to lactate concentrations in labour with minimal contribution by maternal and uteroplacental production.<sup>13</sup> A survey of the scientific literature was undertaken to determine the accuracy of umbilical artery biochemical parameters in predicting poor neonatal outcome.

## Correspondence

Dr Ismail Borat

email: borat@worldonline.co.za

A normal pH in effect “excludes a causal relationship between an acute intrapartum hypoxic event and subsequent neurological disability”.<sup>14</sup> Thorp et al<sup>15</sup> in their study showed that the incidence of newborn depression (1- or 5-minute Apgar score less than 7) was 14.1%; of these depressed newborns, the incidence of normal umbilical cord arterial pH values (greater than or equal to -2 SD) was 77.8%. Of the vigorous newborns, there was a 2.1% prevalence of umbilical cord arterial blood acidaemia. Umbilical cord arterial blood acidaemia in vigorous newborns was not highly predictive of specific morbidity in the immediate newborn period. The following conclusions were reached in their study: (1) that obtaining cord arterial pH values in vigorous newborns should be considered since the values will provide objective documentation of normal fetal acid base balance in 98% of infants. (2) An umbilical cord blood pH value is extremely useful in ruling out the diagnosis of birth asphyxia in the depressed newborn. Furthermore a systematic review of 12 studies has found that umbilical cord lactate is a clinically applicable, inexpensive and effective way to measure acidosis and is a tool that may be used in the assessment of neonatal outcome.<sup>16</sup> Foetal acid-base status is the end point that all antepartum and intrapartum surveillance tools are designed to assess, either directly or indirectly.<sup>7</sup>

Another important clinical question is whether delayed cord clamping has an effect on cord pH? The Committee on Obstetric Practice Opinion of the American College of Obstetrics and Gynaecology states that no difference in umbilical cord pH levels were found with delayed cord clamping and in fact found an increase in umbilical cord pO<sub>2</sub> levels with the technique in normal pregnancies.<sup>17</sup>

It needs to be understood that there will be false positives and false negatives in this assessment. This is inherent in all predictive parameters in clinical medicine but the benefit in terms of the high predictive value in ruling out an acute intrapartum hypoxic event outweighs the possible drawbacks. As with most predictive parameters in clinical medicine, this would be an additional parameter, albeit a very important one, to be interpreted in the context of the existing available parameters ie the CTG, clinical data (intrapartum and neonatal) and Apgar scores. We believe the addition of this quantitative parameter will significantly bolster the defense armamentarium of the obstetrician in instances where claims of CP and NE relating to intrapartum hypoxia are vexatious.

The one question in doing routine cord pH that needs to be addressed is the incidental finding of a low pH in a vigorous infant ie a most likely false positive result, which may be used against the obstetrician. Firstly we know that only 2% of vigorous infants have a low pH<sup>15</sup> which is probably

a false positive so we are dealing with a very small group, putting this into perspective, and that secondly according to the Thorp study<sup>15</sup> the low pH in vigorous infants was not indicative of any specific morbidity; more importantly cord pH in vigorous infants should be interpreted in the context of other available standard parameters like CTG, Apgars and the postnatal course. It needs to be remembered that cord pH is an additional parameter in the overall neonatal assessment, and that false positives will exist as with any screening test (as long as it is low which it is at 2%) and that if the Apgars and CTG's are normal, the finding of an abnormal pH in a vigorous infant should and will be disregarded. The crux for the use of cord pH is protecting the obstetrician from the 98% of normal infants who have a normal cord pH but who develop CP from some other cause ( due to 90 %)<sup>7,8</sup> but for which almost all litigation (for CP) is based on “negligent” intrapartum care which accounts for less than 10% of cases.<sup>3-5</sup> In depressed infants it is protective in 80% of cases, excluding birth asphyxia as a cause.<sup>15</sup>

Another benefit of universal cord blood assessment is the identification of at-risk neonates who require more intensive observation and targeted intervention.<sup>18</sup> Universal assessment of cord blood parameters also reduces the risk of missing the neonate where it is most valuable and keeps the skills intact. Sampling both arterial and venous blood is the only way to confirm that the umbilical artery has, in fact, been sampled.

After review of the scientific literature we would like to make the following recommendations:

Cord blood ph should be done:

- Essential:
  - Universal cord blood assessment
  - Arterial & venous assessment
  - Assessment of pH & base excess (pH < 7.0 and base deficit of > 12mmol/l would be indicative of acute intrapartum hypoxia)
- Recommended but not essential:
  - Assessment of lactate, pO<sub>2</sub> and pCO<sub>2</sub>
  - Placental histology (Also in the light that many babies are ill due to a long-standing problem and this predisposed them to a secondary hypoxic incident during birth. The pH may be low, but there has been other pathology)<sup>3,12</sup>

We strongly believe that a cogent argument for the routine use of cord pH has been presented which will go a long way to protecting the obstetrician from vexatious litigation in cases of cerebral palsy, reduce litigation and in the long term put a hold on the steep rise in insurance premiums which is fast leading to extinction of the discipline of Obstetrics in the private sector.

References

1. Sartwelle TP, Johnston JC. Cerebral palsy litigation: Change course or abandon ship. *J Child Neurol* 2015;30(7):828-41.
2. MacLennan A, Nelson KB, Hankins G, Speer M. Who will deliver our grandchildren? Implications of cerebral palsy litigation. *JAMA* 2005;294(13):1688-90.
3. Jacobsson B, Hagberg G. Antenatal risk factors for cerebral palsy. *Best Pract Res Clin ObstetGynaecol* 2004;18(3):425-36.
4. McIntyre S, Taitz D, Keogh J, Goldsmith S, Badawi N, Blair E. A systematic review of risk factors for cerebral palsy in children born at term in developed countries. *Dev Med Child Neurol* 2013;55(6):499-508.
5. MacLennan AH, Thompson SC, Gecz J. Cerebral palsy: Causes, pathways, and the role of genetic variants. *Am J Obstet Gynecol* 2015;213(6):779-88.
6. Armstrong L, Stenson BJ. Use of umbilical cord blood gas analysis in the assessment of the newborn. *Arch Dis Child Neonatal Ed* 2007; 92:F430-F434.
7. Thorp JA, Dildy GA, Yeomans ER, Meyer BA, Parisi VM. Umbilical cord blood gas analysis at delivery. *Am J ObstetGynecol* 1996; 175: 517-522.
8. MacLennan A, Int Cerebral Palsy Task F. A template for defining a causal relation between acute intrapartum events and cerebral palsy: International consensus statement. *Br. Med. J.* 1999;319(7216):1054-9.
9. Bharti B, Bharti S. A review of the Apgar score indicated that contextualization was required within the contemporary perinatal and neonatal care framework in different settings. *J clinEpidemiol* 2005;58: 121-9.
10. O Donnell CP, Kamlin CO, Davis PG, Carlin JB, Morley CJ. Interobserver variability of the 5-minute Apgar score. *J Pediat* 2006; 149: 486-9.
11. Buchmann EJ, Velaphi SC. Confidential enquiries into hypoxic ischemic encephalopathy. *Best PracResClinObstetGynaecol* 2009; 23: 357-68.
12. Uzan S et al. Acid base balance in the fetus during labour: pathophysiology and exploration methods. *J Gynecol Obstet Biol Reprod* 2003.
13. Nordstrom L et al. Fetal and maternal lactate increase during active second stage of labour. *BJOG* 2001; 108: 263-268.
14. Wong L, Maclellan AH. Gathering the evidence; cord gases and placental histology for births with low Apgar scores. *Australian and New Zealand Journal of Obstetrics and Gynaecology* 2011;5 (1);17-21.
15. Thorp JA(1), Sampson JE, Parisi VM, Creasy RK. Routine umbilical cord blood gas determinations? *Am J Obstet Gynecol.* 1989 Sep;161(3):600-5.
16. Allanson ER, Waqar T, White CRH, Tunçalp O, Dickson JE. Umbilical lactate as a measure of acidosis and predictor of neonatal risk: a systematic review. *BJOG* 2017; 124:584-594.
17. De Paco C, Florido J, Garrido MC et al. Umbilical cord blood acid base and gas analysis after early vs delayed cord clamping in neonates at term. *Arch GynaecolObstet* 2011;283:1011-1.
18. Allanson ER, Pattinson RC, Nathan EA, Dickinson JE. The introduction of umbilical cord lactate measurement and associated neonatal outcomes in a South African tertiary hospital labor ward. *The Journal of Maternal-Fetal & Neonatal Medicine* doi.org/10.1080/14767058.2017.1315094.