International Journal of Gynecology and Obstetrics 131 (2015) 5–8

Contents lists available at ScienceDirect

International Journal of Gynecology and Obstetrics

journal homepage: www.elsevier.com/locate/ijgo

www.figo.org

FIGO GUIDELINES

FIGO consensus guidelines on intrapartum fetal monitoring: Physiology of fetal oxygenation and the main goals of intrapartum fetal monitoring☆,★

Diogo Ayres-de-Campos [a], Sabaratnam Arulkumaran [b], for the FIGO Intrapartum Fetal Monitoring Expert Consensus Panel [1]

[a] Medical School, Institute of Biomedical Engineering, S. Joao Hospital, University of Porto, Portugal
[b] St George's, University of London, London, UK

1. Introduction

This article focuses on the major aspects of the physiology of oxygen supply to the fetus and the main goals of intrapartum fetal monitoring: (1) timely identification of fetuses that are being inadequately oxygenated, to enable appropriate action before the occurrence of injury; and (2) reassurance on adequate fetal oxygenation to avoid unnecessary obstetric interventions. It should be emphasized that to avoid adverse outcome, fetal surveillance requires a timely clinical response and the ready availability of both adequate equipment and trained staff in intrapartum care.

2. The importance of oxygen supply to the fetus

All human cells require oxygen and glucose to maintain aerobic metabolism, their main source of energy production. Glucose can usually be stored and mobilized when needed, but total lack of oxygen supply for just a few minutes is enough to place the cells at risk. During fetal life, oxygen supply is entirely dependent on maternal respiration and circulation, placental perfusion, gas exchange across the placenta, and umbilical and fetal circulations. Complications occurring at any of these levels may result in decreased oxygen concentration in fetal arterial blood (hypoxemia) and ultimately in the tissues (hypoxia). Some degree of hypoxemia occurs in almost all fetuses during labor, but it is the intensity, duration, and repetitive nature of the event, together with the individual variation in the capacity of each fetus to cope with the situation, that will determine the severity of the resulting hypoxia.

Difficulties in carbon dioxide (CO_2) elimination across the placenta will result in elevated CO_2 concentrations, and this gas will combine with water to increase carbonic acid (H_2CO_3) concentration, a phenomenon called respiratory acidemia. The process is quickly reversible with re-establishment of placental gas exchange, as CO_2 diffuses rapidly across the placenta. There is no evidence of injury from isolated respiratory acidemia.

When hypoxia occurs, cellular energy production can still be maintained for a limited time by anaerobic metabolism, but this process produces 19 times less energy and results in the accumulation of lactic acid inside the cell, and its dispersion to the extracellular fluid and fetal circulation. The increased concentration of hydrogen ions of intracellular origin in the fetal circulation is called metabolic acidemia, but it closely parallels hydrogen ion concentration in the tissues, so the term metabolic acidosis is frequently used as a synonym. The hydrogen ions of lactic acid are transferred very slowly across the placenta, but they are buffered by circulating bases, comprised mainly of bicarbonate, hemoglobin, and plasma proteins. The depletion of these buffering agents (increasing base deficit, or base excess in negative numbers) indicates the growing inability to neutralize hydrogen ions, and their continued production will ultimately lead to the disruption of cellular enzyme systems and to tissue injury.

3. Documentation of fetal hypoxia

As oxygen concentration in the tissues cannot in practice be quantified, the occurrence of fetal hypoxia can only be assessed by the documentation of metabolic acidosis. Metabolic acidosis can be evaluated by sampling arterial and venous blood from the umbilical cord immediately after birth (see Appendix 1 for a detailed description of the method), measuring pH and partial pressure of carbon dioxide (pCO_2), and the derived bicarbonate (HCO3-) and base deficit (BD) values. Base deficit in the extracellular fluid (BD_{ecf}), as calculated from umbilical cord

☆ Developed by the FIGO Safe Motherhood and Newborn Health Committee; coordinated by Diogo Ayres-de-Campos.

★ The views expressed in this document reflect the opinion of the individuals and not necessarily those of the institutions that they represent.

[1] Consensus panel: Daniel Surbek (Switzerland*), Gabriela Caracostea (Romania*), Yves Jacquemyn (Belgium*), Susana Santo (Portugal*), Lennart Nordström (Sweden*), Tullia Todros (Italy*), Branka Yli (Norway*), George Farmakidis (Greece*), Sandor Valent (Hungary*), Bruno Carbonne (France*), Kati Ojala (Finland*), José Luis Bartha (Spain*), Joscha Reinhard (Germany*), Anneke Kwee (Netherlands*), Ehigha Enabudoso (Nigeria*), Fadi Mirza (Lebanon*), Tak Yeung Leung (Hong Kong*), Ramon Reyles (Philippines*), Park in Yang (South Korea*), Henry Murray (Australia and New Zealand*), Yuen Tannirandorn (Thailand*), Krishna Kumar (Malaysia*), Taghreed Alhaidari (Iraq*), Tomoaki Ikeda (Japan*), Ferdousi Begum (Bangladesh*), Jorge Carvajal (Chile*), José Teppa (Venezuela*), Renato Sá (Brazil*), Lawrence Devoe (USA**), Gerard Visser (Netherlands**), Richard Paul (USA**), Barry Schifrin (USA**), Julian Parer (USA**), Philip Steer (UK**), Vincenzo Berghella (USA**), Isis Amer-Wahlin (Sweden**), Susanna Timonen (Finland**), Austin Ugwumadu (UK**), João Bernardes (Portugal**), Justo Alonso (Uruguay**), Catherine Spong (USA**), Edwin Chandraharan (UK**).

* Nominated by FIGO associated national society; ** Invited by FIGO based on literature search.

http://dx.doi.org/10.1016/j.ijgo.2015.06.018

blood parameters using the Siggaard-Andersen formula [1,2], is believed by some experts to be the best representative of hydrogen ion concentration of metabolic origin in the different fetal compartments, but the slightly higher BD_{blood}, as calculated by blood gas analyzers can also be used. It should however be noted that different blood gas analyzers may use different algorithms to calculate BD_{blood} [3]. Metabolic acidosis is defined as the measurement in umbilical artery blood of a pH value below 7.00 and a BD in excess of 12 mmol/L [4–6]. However, there is already an association with adverse short-term newborn outcome when pH values are below 7.05 and BD_{ecf} values are above 10 mmol/L [7]. Alternatively, umbilical artery blood lactate concentration may be used to quantify metabolic acidosis, and values exceeding 10 mmol/L have been strongly associated with adverse short-term newborn outcome [8]. However, analyzing devices are often calibrated differently or measure lactate concentrations in different blood compartments, therefore reference values may vary according to the device [9].

Blood gas and lactate analysis in the umbilical cord or in the newborn circulation during the first minutes of life is currently the only way of quantifying objectively the occurrence of hypoxia/acidosis just prior to birth. Umbilical blood sampling is innocuous to the newborn and relatively inexpensive. The resulting information provides useful and immediate feedback to the labor ward staff and can enhance the team's experience with intrapartum monitoring. Umbilical cord blood analysis is also frequently considered important evidence in medico-legal claims. Local guidelines should determine the clinical situations in which umbilical blood analysis will be performed, but if the technology and resources are available, it is recommended in all cases of suspected fetal hypoxia/acidosis and/or low Apgar scores. It should be noted that the presence of metabolic acidosis does not exclude other contributory factors in the causation of neonatal depression and/or subsequent handicap (e.g. prematurity, birth trauma, infection, meconium aspiration, certain congenital anomalies, pre-existing lesions, neonatal hypoxia). Similarly, the absence of metabolic acidosis at birth does not exclude the occurrence of hypoxia/acidosis during pregnancy or earlier in labor.

The Apgar score reflects the pulmonary, cardiovascular, and neurological functions of the newborn, and is depressed when hypoxia is sufficiently intense and prolonged to affect these systems. The 1-minute Apgar score is a crucial parameter to decide the start of newborn resuscitation [10], but has a relatively low association with intrapartum hypoxia/acidosis. Low Apgar scores at both 1 and 5 minutes are expected when severe intrapartum hypoxia/acidosis occurs, but the 5-minute Apgar has a stronger association with short- and long-term neurological outcome and neonatal death [11–13]. However, it is important to remember that Apgar scores are not affected by minor degrees of fetal hypoxia, score assignment is subject to some interobserver disagreement [14], and values can be low due to nonhypoxic causes, such as prematurity, birth trauma, infection, meconium aspiration, certain congenital anomalies, pre-existing lesions, medication administered to the mother, and early neonatal interventions such as vigorous endotracheal aspiration [15].

4. What are we trying to avoid with intrapartum fetal monitoring?

Low intracellular pH and inadequate energy production caused by hypoxia/acidosis have the potential to compromise cell function and to cause cell death. However, the vast majority of fetuses born with metabolic acidosis, with or without decreased Apgar scores, recover quickly and will not incur any short- or long-term complications [4,13,16,17]. In only a few cases will fetal hypoxia/acidosis be of sufficient intensity and duration to cause malfunction of important organs and systems, and thereby put the newborn at risk of death or long-term morbidity.

Short-term neurological dysfunction caused by intrapartum hypoxia/acidosis is called hypoxic-ischemic encephalopathy (HIE), and this diagnosis requires the confirmation of metabolic acidosis, low Apgar scores, early imaging evidence of cerebral edema, and the appearance of changes in muscular tone, sucking movements, seizures, or coma in the first 48 hours of life [18,19]. The Sarnat classification [18] divides HIE into three grades, which in a simplified version can be described as: Grade 1: no seizures present—the vast majority of newborns do not develop major long-term neurological sequelae; Grade 2: seizures—associated with a 20%—30% risk of death or major neurological sequelae; Grade 3: coma—the majority of newborns die or develop long-term neurological sequelae [19,20]. Importantly, there are other nonhypoxic causes for neonatal encephalopathy, and the hypoxic-ischemic nature of this entity needs to be confirmed by the documentation of metabolic acidosis in the umbilical artery or in the newborn circulation during the first minutes of life [21]. HIE may also be accompanied by dysfunction of the cardiovascular, gastrointestinal, hematological, pulmonary, or renal systems.

Cerebral palsy of the spastic quadriplegic or dyskinetic type is the long-term neurological complication that is more commonly associated with intrapartum hypoxia/acidosis at term, but in high-resource countries only 10%—20% of cerebral palsy cases are caused by birth asphyxia [22,23]. Infection, congenital diseases, metabolic diseases, coagulation disorders, antepartum and postnatal hypoxia, and the complications associated with birth trauma and prematurity constitute the majority of causal situations. It may also be linked to a combination of antepartum and intrapartum events. To implicate intrapartum hypoxia/acidosis as the cause of cerebral palsy in term infants there is a need to document the joint occurrence of metabolic acidosis, low 1- and 5-minute Apgar scores, early onset grade 2 or 3 HIE, early imaging studies showing evidence of an acute and nonfocal cerebral anomaly, the development of spastic quadriplegic or dyskinetic types of cerebral palsy, and to exclude other identifiable etiologies (birth trauma, coagulation disorders, infection, and genetic disorders) [6,24].

While avoiding adverse fetal outcome related to hypoxia/acidosis is the main objective of intrapartum fetal monitoring, it is equally important that it does not result in unnecessary obstetric intervention, as some of these procedures, such as instrumental vaginal delivery and cesarean delivery, are associated with increased maternal and fetal risks [25–29].

5. Intrapartum events leading to fetal hypoxia

Contractions compress the maternal blood vessels running inside the myometrium, decreasing placental perfusion [30], and this can result in a temporary reduction of maternal–fetal gas exchange. If during contractions the umbilical cord is compressed between fetal parts, or between fetal parts and the uterine wall, this will result in interference with blood circulation. The frequency, duration, and intensity of uterine contractions are key determinants of the magnitude and effects of these disturbances. The interval between contractions is of particular importance for re-establishment of fetal oxygenation. There are data to suggest that in spontaneous labor it takes up to 90 seconds after a contraction for fetal oxygenation to be restored [31], while in oxytocin-augmented labors this recovery period averages 138 seconds [32]. Excessive uterine activity (see [33] for a definition) is often responsible for decreased fetal oxygenation, and where possible, should be avoided irrespective of fetal heart rate changes [34]. Whether spontaneous or iatrogenic in nature, excessive uterine activity can usually be reversed by reducing or stopping oxytocin infusion and/or starting acute tocolysis with beta-adrenergic agonists (salbutamol, terbutaline, ritodrine) [35], atosiban [36], or nitroglycerine [37].

Other less frequent intrapartum complications can also affect fetal oxygenation. Some of these are of maternal origin, such as the occurrence of acute respiratory distress, cardiorespiratory arrest following amniotic fluid embolism or pulmonary thromboembolism, or sudden maternal hypotension that may occur after epidural or spinal analgesia [38]. Major placental abruption and uterine rupture will also severely

impact fetal oxygenation, the latter due to acute maternal blood loss and/or to the disruption of placental blood supply. Several mechanical complications of delivery may cause compression of the umbilical cord and/or parts of the fetal circulation, such as umbilical cord prolapse, shoulder dystocia, and retention of the after-coming head in a breech delivery. It is also important to note that maternal supine position can lead to aortocaval compression by the pregnant uterus, resulting in reduced placental gas exchange and temporary hypoxemia. Finally, the rare occurrence of fetal hemorrhage, associated with ruptured vasa previa or fetal–maternal hemorrhage, will reduce the oxygen-carrying capacity of the fetal circulation.

All of these complications require specific interventions for their resolution, to tackle the underlying cause and to determine the timing of delivery, with the objective of avoiding prolonged fetal hypoxia/acidosis, as well as unnecessary obstetric intervention. While the specific management of each of these situations is beyond the scope of this document, the general principles involved in the clinical reaction to the fetal heart rate patterns associated with these events are included in the cardiotocography chapter that forms part of FIGO's guidelines on intrapartum fetal monitoring [33].

Conflict of interest

The authors have no conflicts of interest.

References

[1] Siggaard-Andersen O. An acid-base chart for arterial blood with normal and pathophysiological reference areas. Scand J Clin Lab Invest 1971;27(3):239–45.
[2] Wiberg N, Källén K, Olofsson P. Base deficit estimation in umbilical cord blood is influenced by gestational age, choice of fetal fluid compartment, and algorithm for calculation. Am J Obstet Gynecol 2006;195(6):1651–6.
[3] Mokarami P, Wiberg N, Olofsson P. An overlooked aspect on metabolic acidosis at birth: blood gas analyzers calculate base deficit differently. Acta Obstet Gynecol Scand 2012;91(5):574–9.
[4] Low J, Lindsay BG, Derrick EJ. Threshold of metabolic acidosis associated with newborn complications. Am J Obstet Gynecol 1997;177(6):1391–4.
[5] ACOG Committee on Obstetric Practice. ACOG Committee Opinion No. 348, November 2006: Umbilical cord blood gas and acid-base analysis. Obstet Gynecol 2006; 108(5):1319–22.
[6] MacLennan A. A template for defining a causal relation between acute intrapartum events and cerebral palsy: international consensus statement. BMJ 1999;319(7216): 1054–9.
[7] Wayenberg JL. Threshold of metabolic acidosis associated with neonatal encephalopathy in the term newborn. J Matern Fetal Neonatal Med 2005;18(6):381–5.
[8] Wiberg N, Kallen K, Herbst A, Olofsson P. Relation between umbilical cord pH, base deficit, lactate, 5-minute Apgar score and development of hypoxic-ischemic encephalopathy. Acta Obstet Gynecol Scand 2010;89(10):1263–9.
[9] Nordstrom L. Fetal scalp and cord blood lactate. Best Pract Res Clin Obstet Gynaecol 2004;18(3):467–76.
[10] Wall SN, Lee AC, Niermeyer S, English M, Keenan WJ, Carlo W, et al. Neonatal resuscitation in low-resource settings: what, who, and how to overcome challenges to scale up? Int J Gynecol Obstet 2009;107(Suppl. 1):S47–62 S63–4.
[11] Nelson KB, Ellenberg JH. Apgar scores as predictors of chronic neurologic disability. Pediatrics 1981;68(1):36–44.
[12] Casey BM, McIntire DD, Leveno KJ. The continuing value of the Apgar score for the assessment of newborn infants. N Engl J Med 2001;344(7):467–71.
[13] Ruth VJ, Raivio KO. Perinatal brain damage: predictive value of metabolic acidosis and the Apgar score. BMJ 1988;297(6640):24–7.
[14] O'Donnell CP, Kamlin CO, Davis PG, Carlin JB, Morley CJ. Interobserver variability of the 5-minute Apgar score. J Pediatr 2006;149(4):486–9.
[15] Lissauer TJ, Steer PJ. The relation between the need for intubation at birth, abnormal cardiotocograms in labour and cord artery blood gas and pH values. Br J Obstet Gynaecol 1986;93(10):1060–6.
[16] Andres RL, Saade G, Gilstrap LC, Wilkins I, Witlin A, Zlatnik F, et al. Association between umbilical blood gas parameters and neonatal morbidity and death in neonates with pathologic fetal acidemia. Am J Obstet Gynecol 1999;181(4):867–71.
[17] van de Riet JE, Vandenbussche FP, Le Cessie S, Keirse MJ. Newborn assessment and long-term adverse outcome: a systematic review. Am J Obstet Gynecol 1999; 180(4):1024–9.
[18] Sarnat HB, Sarnat MS. Neonatal encephalopathy following fetal distress. A clinical and electroencephalographic study. Arch Neurol 1976;33(10):696–705.
[19] Levene MI, Sands C, Grindulis H, Moore JR. Comparison of two methods of predicting outcome in perinatal asphyxia. Lancet 1986;1(8472):67–9.
[20] Dennis J, Chalmers I. Very early neonatal seizure rate: a possible epidemiological indicator of the quality of perinatal care. Br J Obstet Gynaecol 1982;89(6):418–26.
[21] The American College of Obstetricians and Gynecologists' Task Force on Neonatal Encephalopathy and Cerebral Palsy, the American College of Obstetricians and Gynecologists, the American Academy of Pediatrics. Neonatal encephalopathy and cerebral palsy: defining the pathogenesis and pathophysiology. Washington, DC: ACOG; 2003 1–85.
[22] Nelson KB, Ellenberg JH. Antecedents of cerebral palsy. Multivariate analysis of risk. N Engl J Med 1986;315(2):81–6.
[23] Blair E, Stanley FJ. Intrapartum asphyxia: a rare cause of cerebral palsy. J Pediatr 1988;112(4):515–9.
[24] American College of Obstetricians and Gynecologists (ACOG). Neonatal encephalopathy and cerebral palsy: executive summary. Obstet Gynecol 2004;103(4):780–1.
[25] Villar J, Carroli G, Zavaleta N, Donner A, Wojdyla D, Faundes A, et al. Maternal and neonatal individual risks and benefits associated with cesarean delivery: multicentre prospective study. BMJ 2007;335(7628):1025.
[26] Silver RM. Implication of the first cesarean: perinatal and future reproductive health and subsequent cesareans, placentation issues, uterine rupture risk, morbidity, and mortality. Semin Perinatol 2012;36(35):315–23.
[27] Signore C, Klebanoff M. Neonatal morbidity and mortality after elective cesarean delivery. Clin Perinatol 2008;35(2):361–71.
[28] Wilmink FA, Hukkelhoven CW, Lunshof S, Mol BW, van der Post JA, Papatsonis DN. Neonatal outcome following elective cesarean section beyond 37 weeks of gestation: a 7-year retrospective analysis of a national registry. Am J Obstet Gynecol 2010;202(3):250.e1–8.
[29] O'Mahony F, Hofmeyr GJ, Menon V. Choice of instruments for assisted vaginal delivery. Cochrane Database Syst Rev 2010;10(11):CD005455.
[30] Reynolds SR, Freese UE, Bieniarz J, Caldeyro-Barcia R, Mendez-Bauer C, Escarcena L. Multiple simultaneous intervillous space pressures recorded in several regions of the hemochorial placenta in relation to functional anatomy of the fetal cotyledon. Am J Obstet Gynecol 1968;102(8):1128–34.
[31] McNamara H, Johnson N. The effect of uterine contractions on fetal oxygen saturation. Br J Obstet Gynaecol 1995;102(8):644–7.
[32] Peebles DM, Spencer JA, Edwards AD, Wyatt JS, Reynolds EO, Cope M, et al. Relation between frequency of uterine contractions and human fetal cerebral oxygen saturation studied during labour by near infrared spectroscopy. Br J Obstet Gynaecol 1994; 101(1):44–8.
[33] Ayres-de-Campos D, Spong CY, Chandraharan E, for the FIGO Intrapartum Fetal Monitoring Expert Consensus Panel. FIGO consensus guidelines on intrapartum fetal monitoring: Cardiotocography. Int J Gynecol Obstet 2015;131:13–24.
[34] Heuser CC, Knight S, Esplin S, Eller AG, Holmgren CM, Richards D, et al. Tachysystole in term labor: incidence, risk factors, outcomes, and effect on fetal heart tracings. Am J Obstet Gynecol 2013;209(1):32.e1–6.
[35] de Heus R, Mulder EJ, Derks JB, Visser GH. Acute tocolysis for uterine activity reduction in term labor: a review. Obstet Gynecol Surv 2008;63(6):383–8.
[36] Heus R, Mulder EJ, Derks JB, Kurver PH, van Wolfswinkel L, Visser GH. A prospective randomized trial of acute tocolysis in term labour with atosiban or ritodrine. Eur J Obstet Gynecol Reprod Biol 2008;139(2):139–45.
[37] Pullen KM, Riley ET, Waller SA, Taylor L, Caughey AB, Druzin ML, et al. Randomized comparison of intravenous terbutaline vs nitroglycerin for acute intrapartum fetal resuscitation. Am J Obstet Gynecol 2007;197(4):414.e1–6.
[38] Reynolds F, Sharma SK, Seed PT. Analgesia in labour and fetal acid-base balance: a meta-analysis comparing epidural with systemic opioid analgesia. BJOG 2002; 109(12):1344–53.

Appendix 1. Umbilical blood sampling technique, interpretation, and pitfalls

Sampling of umbilical arterial and venous blood shortly after delivery is needed to document objectively the occurrence of fetal hypoxia/acidosis. Clamping of the cord is not necessary before vessels are sampled, but umbilical blood gas concentrations change quickly after birth, so this needs to be performed as soon as possible [1,2]. Even if the cord is doubly clamped, sampling of vessels should be performed as soon as possible and preferably within 15 minutes, as blood gas and lactate values change significantly over time [3,4]. Blood should be drawn, introducing as little air as possible, into two different 1- or 2-mL pre-heparinized syringes (if pre-heparinized syringes are not available, a small quantity of heparin can be drawn into normal syringes, and the excess heparin expelled before blood sampling). After blood is drawn, existing air bubbles should be removed from the syringes, these should be capped, rolled between the fingers to mix blood with heparin, and blood gas analysis should be performed in a calibrated apparatus within the next 30 minutes [3].

Umbilical arterial blood reflects the fetal acid-base status better than venous blood. However, it is important to obtain blood from both artery and vein to assure that a valid arterial sample is present. Sampling of the wrong vessel is not uncommon, particularly when the needle crosses the artery to pierce the vein, and this can also result in mixed sampling. Arterial pH is lower than that of the vein, and when the difference in pH

between the two blood samples is less than 0.02 and the difference in pCO_2 is less than 5 mm Hg or 0.7 kPa (kilopascal), then the samples are most likely mixed or were obtained from the same vessel [4]. In addition, a pCO_2 less than 22 mm Hg or 2.9 kPa is almost impossible to achieve in the umbilical artery, so such a value indicates likely contamination from the umbilical vein or from air [5].

Median umbilical artery pH in deliveries after 36 weeks of gestation is 7.25 (5th percentile 7.06; 95th percentile 7.37), median arterial BD_{ecf} 2.8 mmol/L (5th percentile −1.8; 95th percentile 10.0) [4]. Mean arterial BD_{blood} in a similar population was 5.6 mmol/L (5th percentile 0.28; 95th percentile 11.48) [6]. When placental gas exchange is preserved, there is slow transfer of hydrogen ions in both directions, so maternal hyperventilation may result in an increase in fetal pH and likewise maternal acidemia will slowly result in fetal acidemia.

When gas exchange across the placenta is compromised or when there is significant umbilical cord occlusion, both increased carbon dioxide and decreased oxygen concentrations may occur in the fetus, and thus an acidemia of mixed respiratory and metabolic origin is documented. However, the metabolic component, reflected in the base deficit is the one with the greatest potential for harm, as it indicates decreased cellular oxygen concentration and reduced energy production.

References

[1] Armstrong L, Stenson B. Effect of delayed sampling on umbilical cord arterial and venous lactate and blood gases in clamped and unclamped vessels. Arch Dis Child Fetal Neonatal Ed 2006;91(5):F342–5.
[2] Wiberg N, Kallen K, Olofsson P. Delayed umbilical cord clamping at birth has effects on arterial and venous blood gases and lactate concentrations. BJOG 2008;115(6):697–703.
[3] ACOG Committee on Obstetric Practice. ACOG Committee Opinion No. 348, November 2006: Umbilical cord blood gas and acid-base analysis. Obstet Gynecol 2006;108(5):1319–22.
[4] White CR, Mok, T, Doherty DA, Henderson JJ, Newnham JP, Pennell CE. The effect of time, temperature and storage device on umbilical cord blood gas and lactate measurement: a randomized controlled trial. J Mater Fetal Neonat Med 2012;25(6):587–94.
[5] Kro GA, Yli B, Rasmussen S, Noren H, Amer-Wahlin I, Saugstad OD, et al. A new tool for the validation of umbilical cord acid-base data. BJOG 2010;117(12):1544–52.
[6] Victory R, Penava D, da Silva O, Natale R, Richardson B. Umbilical cord pH and base excess values in relation to adverse outcome events for infants delivering at term. Am J Obstet Gynecol 2004;191(6):2021–8.