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Original Article

EUROPEAN ASSOCIATION OF PERINATAL MEDICINE (EAPM) Position statement: Use of appropriate terminology for situations related to inadequate fetal oxygenation in labor

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ABSTRACT

In high-resource countries, adverse perinatal outcomes are currently rare in term, non-malformed fetuses, undergoing labor, but they remain a leading cause of medico-legal dispute. Precise terminology is important to describe situations related to inadequate fetal oxygenation in labor, to ensure appropriate communication between healthcare professionals and adequate transmission of information to parents. This position statement provides consensus definitions from European perinatologists and midwives regarding the most appropriate terminology to describe situations related to inadequate fetal oxygenation in labor: suspected fetal hypoxia, severe newborn acidemia, newborn metabolic acidosis, and hypoxic-ischemic encephalopathy. It also identifies terms that are imprecise or nonspecific to this situation, and should therefore be avoided by healthcare professionals: fetal well-being, fetal stress, fetal distress, non-reassuring fetal state, and birth asphyxia.

Introduction

Adequate fetal tissue oxygenation is essential for the birth of a healthy newborn. A reduction in the oxygen content of fetal arterial blood (hypoxemia), when severe and prolonged, will lead to reduced oxygen supply to fetal tissues (hypoxia), triggering the onset of anaerobic metabolism in fetal cells. This results in the intracellular production of lactic acid, which when of sufficient magnitude, will lead to lowered blood pH and a depletion of buffering agents - increased base deficit (BD). If the situation persists, the newborn may display low Apgar scores, and require resuscitation. It may also develop adverse short- and long-term outcomes, such as hypoxic-ischemic encephalopathy, perinatal death, and a wide spectrum of neurodevelopmental and neurocognitive impairments, including cerebral palsy [1,2].

It is important to use precise and consistent language in situations of inadequate fetal oxygenation during labor, in order to ensure optimal communication, both with parents and between healthcare professionals. Moreover, adverse outcomes may result in medico-legal dispute, but they are not always caused by inadequate fetal oxygenation in labor, so a clear terminology is necessary to describe all of these potential causes of litigation.

The European Association of Perinatal Medicine (EAPM) has a special interest group on "Intrapartum Care", constituted by Obstetricians, Midwives and Neonatologists with expertise and a special interest in this area of knowledge. Its main aim is to contribute to improvement in the quality of care provided to laboring women in Europe, and it organizes

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the biennial European Congress of Intrapartum Care. The present position statement was developed by this special interest group, using regular online discussions and shared-document iterations to achieve a consensus. The document was subsequently approved by the EAPM Executive Board.

Suspected fetal hypoxia

This term refers to the likely occurrence of ongoing reduced oxygen supply to fetal tissues. There is no clinically available method of objectively documenting or quantifying the occurrence of fetal hypoxia during labor, but the situation can be suspected from analysis of cardiotocographic tracings, electrocardiography, or fetal blood sampling. For interactions with non-healthcare professionals a more adequate term to describe this situation may be "suspected insufficient oxygenation of the baby".

Periods of fetal hypoxia are common during labor, and most of the time they are mild and self-limited. Uterine contractions can cause transient falls in oxygen delivery to the fetus, but in healthy women with adequately grown fetuses, hypoxia is usually transient and poses little risk of permanent damage. On the other hand, if fetal hypoxia is severe, prolonged or recurring in nature, it may cause permanent damage to fetal organs or even perinatal death.

Severe newborn acidemia

This term refers to the documentation of a pH value lower than 7.10 in umbilical arterial blood taken shortly after delivery, when the other criteria for newborn metabolic acidosis (please see below) are not met. The clinical and scientific value of "severe newborn acidemia" is that it identifies situations where the fetus was subjected to (a) relevant episode(s) of inadequate oxygenation during labor, but still within tolerable limits of risk. It is likely that a degree of fetal hypoxia occurred in these situations, but it is not expected to result in permanent organ damage or newborn death.

It is difficult to select arbitrary cut-off values to define "severe" fetal acidemia. The average pH in a newborn infant after vaginal delivery is lower (7.22–7.27) than fetal pH prior to the onset of labor (7.35–7.45). In a cohort study from the United Kingdom, including more than 50,000 singleton term newborns, the median arterial pH was 7.22, and the 5th percentile was 7.05 [3]. In a European study including a low-risk population (spontaneous vaginal deliveries, 5-minute Apgar score $\geq$ 7), the median arterial pH was 7.27, and the 2.5th percentile was 7.09 [4].

Increasing umbilical artery acidemia is associated with a continuum of growing risk for newborn morbidity and mortality [5,6]. In the previously mentioned cohort from the United Kingdom [3], the risk of adverse neurological outcome was only slightly increased when umbilical artery pH was lower than 7.10. A population study evaluating more than one million live-born singletons in Finland also found that cord artery pH between 7.00 and 7.10 was associated with only a marginally increased risk of neurological morbidity (cerebral palsy, epilepsy and sensorineural defects), when compared with higher pH values [7].

While the vast majority of neonates with severe acidemia have no short- or long-term damage to neurological or other systems, when this finding coincides with maternal intrapartum hyperthermia, a synergistic damaging effect on the fetal brain has been demonstrated [8].

Newborn metabolic acidosis

This term refers to the documentation of a pH value lower than 7.00 and a base deficit of 12 mmol/L or more (in blood or extracellular fluid), in umbilical blood taken shortly after delivery. Alternatively, it can be defined as lactate values in umbilical blood exceeding 10 mmol/L. When umbilical blood is unavailable, similar cut-off values apply to newborn blood obtained during the first minutes of life. The documentation of metabolic acidosis is considered the most objective evidence of the occurrence of severe fetal hypoxia in the final stages of labor.

The international consensus of the Cerebral Palsy Task Force in 1999 established umbilical blood pH less than 7.00 and base deficit exceeding 12 mmol/L as the levels of fetal hypoxia that are considered sufficient to cause permanent organ damage leading to cerebral palsy [9]. These values were upheld by the International Federation of Gynecology and Obstetrics in their 2015 consensus guidelines on intrapartum fetal monitoring [10].

When umbilical artery blood gas values are not indicative of metabolic acidosis, permanent organ damage and perinatal death are unlikely to have been caused by inadequate fetal oxygenation in labor. However, a normal umbilical arterial pH does not exclude the occurrence of severe fetal hypoxia and organ damage before labor, or in the initial stages of labor, as sufficient time may have elapsed for fetal acidemia to have corrected. Moreover, in the rare occurrence of a sudden and near-complete cord obstruction, umbilical cord blood taken from the placental side of the obstruction may not reflect the severity of fetal hypoxia [11]. The same holds true for neurological hypoxic damage that can arise from the continuous compression of fetal neck vessels, as occurs in shoulder dystocia. In the latter situation, umbilical blood gas values may not reflect the degree of hypoxia and acidemia occurring in fetal head vessels.

Hypoxic-ischemic encephalopathy (HIE)

This term refers to a syndrome occurring in the first 48 h of life, which includes changes in newborn muscular tone, movement and alertness, as well as the occurrence of seizures or coma, in association with the documentation of metabolic acidosis (please see above). The appearance of the aforesaid neurological changes during the first 48 h after delivery is termed "neonatal encephalopathy", but several causes for this exist, of which hypoxia–ischemia is one. The hypoxic-ischemic nature of this entity, and its occurrence during the final stages of labor, require the documentation of metabolic acidosis, low Apgar scores, and the presence of early developing encephalopathy. HIE may also be accompanied by dysfunction of the cardiovascular, gastrointestinal, hematological, respiratory, and urinary systems. HIE can vary from mild, moderate to severe, and different scores can be used to make this classification more objective. The most severe forms of HIE can lead to neonatal death or to a wide spectrum of long-term neurodevelopmental and neurocognitive impairments, including cerebral palsy. However, it is important to note that most neonates developing long-term neurological impairments do not have evidence of inadequate fetal oxygenation during labor [1].

Terms that should be avoided

Several terms have been used in the past to describe situations related to inadequate fetal oxygenation during labor, and some of them are imprecise and nonspecific of this situation. The terms "fetal well-being", "fetal stress", "fetal distress" and "non-reassuring fetal state" do not have precise or widely accepted definitions, and include concepts that are not specific to fetal oxygenation, so they should not be used by healthcare professionals [1,12]. The term "perinatal asphyxia" also has many poorly specified and non-consensual definitions, so it should be avoided by healthcare professionals [1,12].

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

[1] Ayres-de-Campos D, Arulkumaran S. for the FIGO Intrapartum Fetal Monitoring Expert Consensus Panel. FIGO consensus guidelines on intrapartum fetal monitoring: Physiology of fetal oxygenation and the main goals of intrapartum fetal monitoring. Int J Gynaecol Obstet 2015;131:5–8.

[2] Moshiro R, Mdoe P, Perlman JM. A global view of neonatal asphyxia and resuscitation. Front Pediatr 2019;7:489.

[3] Yeh P, Emary K, Impey L. The relationship between umbilical cord arterial pH and serious adverse neonatal outcome: analysis of 51,519 consecutive validated samples. BJOG 2012;119:824–31.

[4] Kro GAB, Yli BM, Rasmussen S, Norèn H, Amer-Wåhlin I, Saugstad OD, et al. A new tool for the validation of umbilical cord acid-base data. BJOG 2010;117:1544–52.

[5] Kelly R, Ramaiah SM, Sheridan H, Cruickshank H, Rudnicka M, Kissack C, et al. Dose-dependent relationship between acidosis at birth and likelihood of death or cerebral palsy. Arch Dis Child Fetal Neonatal Ed 2018;103:F567–72.

[6] Malin GL, Morris RK, Khan KS. Strength of association between umbilical cord pH and perinatal and long term outcomes: systematic review and meta-analysis. BMJ 2010;13(340):c1471.

[7] Seikku L, Gissler M, Andersson S, Rahkonen P, Stefanovic V, Tikkanen M, et al. Asphyxia, neurologic morbidity, and perinatal mortality in early-term and postterm birth. Pediatrics 2016;137:e20153334.

[8] Impey LWM, Greenwood CEL, Black RS, Yeh PSY, Sheil O, Doyle P. The relationship between intrapartum maternal fever and neonatal acidosis as risk factors for neonatal encephalopathy. Am J Obstet Gynecol 2008;198(49):e1–6.

[9] MacLennan AH, Thompson SC, Gecz J. Cerebral palsy: causes, pathways, and the role of genetic variants. Am J Obstet Gynecol 2015;213:779–88.

[10] 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 Gynaecol Obstet 2015;131:13–24.

[11] Pomerance J. Umbilical cord blood gases casebook. Interpreting umbilical cord blood gases, VII. J Perinatol 2000;20(5):338–9.

[12] American College of Obstetricians and Gynecologists. Committee Opinion N° 197: Inappropriate use of the terms fetal distress and birth asphyxia. Obstet Gynecol 2005;106:1469–7070.