FIGO CONSENSUS GUIDELINES ON INTRAPARTUM FETAL MONITORING

Safe Motherhood and Newborn Health Committee
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PHYSIOLOGY OF FETAL OXYGENATION AND THE MAIN GOALS OF INTRAPARTUM FETAL MONITORING

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Introduction

This chapter 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; (2) reassurance on adequate fetal oxygenation to avoid unnecessary obstetric interventions. It should be emphasized that in order 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.

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 mobilised 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, 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 labour, 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, haemoglobin and plasma proteins. The depletion of these buffering agents (increasing base deficit, or base excess in negative numbers) indicates the growing inability to neutralise hydrogen ions, and their continued production will ultimately lead to the disruption of cellular enzyme systems and to tissue injury.

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 Annex 1 for a detailed description of the method), measuring pH and partial pressure of carbon dioxide (pCO_2), and the derived bicarbonate (HCO_3^-) and base deficit (BD) values. Base deficit in the extracellular fluid (BD_{ecf}), as calculated from umbilical cord 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 analysers can also be used. It should however be noted that different blood gas analysers 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 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, analysing

devices are often calibrated differently or measure lactate concentrations in different blood compartments, so 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 labour 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 should 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 labour.

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/acidemia. Low Apgar scores at both 1 and 5 minutes are expected when severe intrapartum hypoxia/acidemia 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 inter-observer disagreement [14], and values can be low due to non-hypoxic 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].

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 [13,16-18]. 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 [19,20]. In a simplified way, it can be divided into three grades (Sarnat & Sarnat classification [19]): 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 [20,21]. Importantly, there are other non-hypoxic 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 [22]. HIE may also be accompanied by dysfunction of the cardiovascular, gastrointestinal, haematological, 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 developed countries only 10-20% of cerebral palsy cases are caused by birth asphyxia [23,24]. Infection, congenital diseases, metabolic diseases, coagulation disorders, antepartum and post-natal 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 hypoxic-ischemic encephalopathy, early imaging studies showing evidence of an acute and non-focal 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) [6,25].

While avoiding adverse fetal outcome related to hypoxia/acidemia is the main objective of intrapartum fetal monitoring, it is equally important that it does not result in unnecessary obstetrical intervention, as some of these procedures, such as instrumental vaginal delivery and caesarean section, are associated with increased maternal and fetal risks [26-30].

Intrapartum events leading to fetal hypoxia

Contractions compress the maternal blood vessels running inside the myometrium, decreasing placental perfusion [31], 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 labour it takes up to 90 seconds after a contraction for fetal oxygenation to be restored [32], while in oxytocin-augmented labours this recovery period averages 138 seconds [33]. Excessive uterine activity (please see Chapter 3 for a definition) is often responsible for decreased fetal oxygenation, and where possible, should be avoided irrespective of FHR

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-adrenegic 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 complications, a cardio-respiratory 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 aorto-caval 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 FHR patterns associated with these events are included in the following chapters.

Annex 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 [39,40]. 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 [41,42]. Blood should be drawn, introducing as little air as possible, into two different 1 or 2 ml pre-heparinised syringes (if pre-heparinised 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 [41].

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 in order 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 [42]. In addition, a pCO_2 < 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 [43].

Median umbilical artery pH in deliveries after 36 weeks of gestation is 7.25 (5^{th} percentile 7.06; 95^{th} percentile 7.37), median arterial BD_{ecf} 2.8 mmol/l (5^{th} percentile -1.8; 95^{th} percentile 10.0) [42]. Mean arterial BD_{blood} in a similar population was 5.6 (5^{th} percentile 0.28; 95^{th} percentile 11.48 mmol) [44]. 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 CO_2 and decreased O_2 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 BD is the one with the greatest potential for harm, as it indicates decreased cellular oxygen concentration and reduced energy production.

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