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Introduction: Why is intrapartum foetal monitoring necessary – Impact on outcomes and interventions

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Maintaining maternal oxygen supply is essential for foetal life, and labour constitutes an increased challenge to this. Good clinical judgement is required to evaluate the signs of reduced foetal oxygenation, to diagnose the underlying cause, to judge the reversibility of the condition and to determine the best timing for delivery. The main aim of intrapartum foetal monitoring is to identify foetuses that are being inadequately oxygenated, enabling appropriate action before the occurrence of injury. It is also to provide reassurance in cases of adequate foetal oxygenation, and thus to avoid unnecessary obstetric intervention. Poor foetal oxygenation is diagnosed by documenting metabolic acidosis in the umbilical cord immediately after birth or in the newborn circulation during the first minutes of life. However, most newborns recover quickly, and they do not develop relevant short- or long-term complications. Hypoxic–ischaemic encephalopathy is the short-term neurological dysfunction caused by inadequate intrapartum foetal oxygenation, and cerebral palsy of the spastic quadriplegic or dyskinetic types is the long-term neurological complication most commonly associated with it. Although there is insufficient evidence from randomised controlled trials to demonstrate that any form of intrapartum foetal monitoring reduces the incidence of adverse outcomes, reports from the clinical setting have documented a decrease in metabolic acidosis, hypoxic–ischaemic encephalopathy and intrapartum death over the last decades. It may be difficult to demonstrate the benefit of

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diagnostic techniques in complex environments such as the labour ward, but a reduction in the incidence of adverse clinical outcomes constitutes important evidence that intrapartum foetal monitoring makes a difference.

The importance of oxygen supply to the foetus

The foetus requires oxygen and glucose to maintain cellular aerobic metabolism, its main source of energy production. Although glucose can be stored and later mobilised, oxygen needs to be supplied continuously, as an interruption of only a few minutes is enough to place the foetus at risk. Oxygen is obtained via the maternal respiration and circulation, placental perfusion, placental gas exchange, umbilical cord and foetal circulation. Complications occurring at any of these levels may result in decreased oxygen supply, with a subsequent reduction in foetal arterial oxygen concentration (hypoxaemia), and ultimately oxygen supply to the foetal tissues (hypoxia).

In the absence of oxygen, the energy production in the foetal cells can still be maintained for a limited period of time, using the anaerobic metabolism pathway, but this yields 19 times less energy and results in the production of lactic acid. The consequent increase in hydrogen ion concentration inside the cell, in the extracellular fluid and in the foetal circulation is called metabolic acidosis. Reduced energy production and increased hydrogen ion concentration will ultimately lead to cell death and to tissue injury.

Why does labour increase the risk of foetal hypoxia/acidosis?

Uterine contractions compress the blood vessels running inside the myometrium, and this may temporarily decrease the perfusion of the placental bed. Sometimes, the umbilical cord is compressed between foetal parts, or between the foetus and the uterine wall, and umbilical blood circulation may be reduced during contractions. The frequency, duration and intensity of uterine contractions determine the magnitude of these disturbances, and the interval between contractions is crucial for re-establishment of foetal oxygenation. Excessive uterine activity is the most common cause of foetal hypoxia/acidosis, and should be avoided irrespective of the occurrence of foetal heart rate (FHR) changes [1]. This can usually be accomplished by reducing oxytocin infusion, removing administered prostaglandins and/or starting acute tocolysis with beta-adrenergic agonists (salbutamol, terbutaline and ritodrine), atosiban or nitroglycerin. During the second stage of labour, maternal pushing may aggravate the effect of uterine contractions, so the mother should also be asked to stop pushing until the situation is reversed. Transient cord compression can sometimes be resolved by changing the maternal position or performing amnioinfusion [2].

Another frequent cause of reduced foetal oxygenation is the maternal supine position, which may cause aorto-caval compression by the pregnant uterus and decreased placental perfusion. Turning the mother on her side or asking her to stand up is usually followed by the normalisation of foetal oxygenation.

Other maternal respiratory or circulatory complications may affect foetal oxygenation, such as acute respiratory distress, sudden hypotension or cardiac dysfunction. Sudden maternal hypotension occurs more frequently after epidural or spinal analgesia, and it can usually be reversed by rapid fluid administration and/or an intravenous ephedrine bolus. Other maternal complications require specific management, and the normalisation of foetal oxygenation depends on their reversible nature and on the expected speed of recovery.

Major placental abruption and uterine rupture usually have a severe impact on foetal oxygenation due to maternal blood loss and to the disruption of placental gas exchange. Both situations cause an irreversible foetal hypoxia, and they require expedite delivery to avoid an adverse outcome.

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