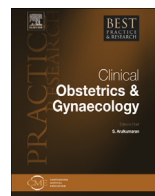




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Foetal scalp blood sampling during labour for pH
and lactates

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Second-line methods of foetal monitoring have been developed in an attempt to reduce unnecessary interventions due to continuous cardiotocography (CTG), and to better identify foetuses that are at risk of intrapartum asphyxia.

Very few studies directly compared CTG with foetal scalp blood (FBS) and CTG only. Only one randomised controlled trial (RCT) was published in the 1970s and had limited power to assess neonatal outcome. Direct and indirect comparisons conclude that FBS could reduce the number of caesarean deliveries associated with the use of continuous CTG.

The main drawbacks of FBS are its invasive and discontinuous nature and the need for a sufficient volume of foetal blood for analysis, especially for pH measurement, resulting in failure rates reaching 10%. FBS for lactate measurement became popular with the design of test-strip devices, requiring <0.5 mL of foetal blood. RCTs showed similar outcomes with the use of FBS for lactates compared with pH in terms of obstetrical interventions and neonatal outcomes.

In conclusion, there is some evidence that FBS reduces the need for operative deliveries. However, the evidence is limited with regard to actual standards, and large RCTs, directly comparing CTG only with CTG with FBS, are still needed.

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Introduction

Q4 The main objective of foetal monitoring during labour is to identify and prevent perinatal asphyxia and its most serious consequences: perinatal death, hypoxic ischaemic encephalopathy and cerebral palsy. One of the major criteria to establish the causal link between asphyxia and cerebral palsy is metabolic acidosis on arterial cord blood or on very early neonatal samples: pH < 7.00 and base deficit (BD) > 12 mmol/L [1,2]. Deep neonatal acidosis is also one of the major criteria of birth asphyxia. Logically, biochemical parameters of acidosis, such as pH, BD and lactate measurement, seem to be natural candidates for foetal monitoring.

Foetal heart rate (FHR) monitoring has long been considered the key method for diagnosis of intrapartum asphyxia. However, doubts arose with the lack of reduction in the incidence of cerebral palsy since the widespread use of FHR monitoring [3]. In addition, a significant increase in caesarean section rates during labour was also recorded during the same period. The role of the FHR monitoring in reducing perinatal mortality since the 1970s cannot be formally established, but it is still considered the best screening method because of its high sensitivity, meaning that a normal FHR almost ascertains foetal well-being.

However, interventions for abnormal FHR are very frequently labelled ‘for foetal distress’, because, in most cases, there are no signs of asphyxia at birth. Some interventions would probably have been avoided by better assessment of the foetus, resulting in the development of adjunctive or ‘second-line’ techniques. Foetal scalp blood sampling (FBS) for pH or lactate measurements is one of the oldest methods. This study was conducted on MEDLINE database with the following keywords: foetal asphyxia, FBS, intrapartum foetal monitoring, scalp blood pH, scalp blood lactates, umbilical artery blood gases and cerebral palsy.

Acid–base balance of the foetus and neonate

Before labour, the normal arterial pH of the foetus is close to 7.35 [4]. The main difference with someone breathing in air is the low oxygenation of foetal blood. The normal foetal PaO₂ is between 20 and 30 mmHg, which corresponds to an average oxygen saturation of foetal haemoglobin at 40–50%. The arterial cord blood flows from the foetus to the placenta and reflects foetal acid–base balance, while venous blood comes from the placenta and has higher oxygen content. During labour, there is a physiological decrease in pH. The average pH of the umbilical artery blood at birth is 7.25 and the 10th centile is around 7.15 [5]. Moderate neonatal acidosis may thus be defined as a pH below 7.15 in the umbilical artery, which does not mean that this represents a risk for immediate or long-term complications. The risk for the foetus and neonate depends on the severity and the type of acidosis. The following are the two types of acidosis.

- *Respiratory acidosis* is due to the accumulation of CO₂, responsible for a movement to the right of the equation: $\text{CO}_2 + \text{H}_2\text{O} \leftrightarrow \text{H}^+ + \text{HCO}_3^-$, and production of H⁺ ions. This can occur very rapidly (within minutes) and is also quickly solved after birth, when the newborn eliminates the accumulated CO₂ while breathing in the air. Pure respiratory acidosis has no long-term consequences to the newborn in terms of neurological outcome.
- *Metabolic acidosis* is related to a shift to anaerobic pathways during prolonged hypoxia. Anaerobic glycolysis converts glucose into pyruvate, and then into lactate and H⁺ ions, resulting in a decrease in pH. This phenomenon is also longer to disappear, and metabolic acidosis may remain for several hours after the correction of hypoxia. Above all, deep metabolic acidosis may be responsible for irreversible organ damage.

The diagnosis and the type of acidosis are mainly based on the following parameters, available by gas analysis of umbilical artery blood [2,5–7] (Table 1):

- pH: decreased in all types of acidosis (severe when pH < 7.00).
- pCO₂: increased in case of respiratory acidosis (≥75 mmHg).

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