Lactate assessment during first and second stage

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Lactate analysis with a modern electrochemical microvolume test strip method (Lactate Pro™) is simple, quick, reliable and reproducable (1). Possible sources of errors in sampling and analysis (insufficient sample, contamination of amniotic fluid, caput formation) are minor problems. In a rct lactate determination in FBS has been tested in comparison with pH analysis in the management of fetal distress, and shown to be feasable in a clinical setting. A large, retrospective study has suggested a new lactate concentration as cut off level for intervention, 4.8 mmol/l. With this cut off level lactate performed better than pH in the identification of severe neonatal outcome, i.e Apgar score <4 at 5´and moderate/severe HIE (2). In clinical practice we have experienced no falce negatives. However, occationally lactate is measured high without concomitant acidaemia. In the group with severe hypoxic damage, a number of cases have shown to be hyperlactaemic but not acidaemic. Meantime we therefore regard high lactate as indication for intervention irrespective of pH value. A speculation in explaining the patophysiology could be; increased lactate in subcutaneous tissue as an early marker for hypoxia. Alternatively, a transient hypoxic insult might have corrected acidaemia at the time of FBS while lactate is still measured high.

During active second stage fetal lactate increases with about 1 mmol/l for every 30 min of pushing (3). The lactate increase is mainly fetally derived. We believe this fact makes clinical interpretation of lactate measurements during the second stage more difficult.
A rct comparing pH and lactate, using the new suggested cut off level, is discussed.

References

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