Physiological insights based on fetal tcPo₂ monitoring

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With the help of the tcPo₂ technique, it is possible to monitor continuously fetal Po₂ which was previously only possible with intermittent sampling according to the technique of Saling. Results from continuous recordings of biochemical blood gas parameters in subjects after birth have shown how quickly levels can change and that even very frequent blood sampling cannot ensure that a short term hypoxia is always detected. It is therefore an old dream of obstetricians to get continuous information about the status of fetal oxygen supply.

The value of the tcPo₂ method compared to other methods to assess fetal well being will greatly depend on the value of fetal Po₂ in arterialised scalp blood as an indicator of a generalised disturbance of gas exchange. As it is true for all new, and to some extent sophisticated methods, the actual limitation of the method first depends on the experience of the obstetrician. Furthermore it should be pointed out clearly that new methods potentially bear two advantages, not known before, which one will be dominating. The question always persists, if the major contribution of a method is it's general application in clinical medicine or if the major contribution is it's clinical insight in physiology and pathophysiology resulting in an improved clinical management.

In the following, we will give few examples of new insights:

Fig. 1

The typical influence of contractions on fetal tcPo₂. From the onset of the contraction marked C to the beginning of the fall in fetal tcPo₂ 50-60 sec elapse. Fenoterol (Partusisten) was given 85 min before delivery and resulted in suppression of the contraction (from (1))

Figure 1 describes the typical Po₂ behaviour in relation to uterine contractions which can only be observed with a continuous technique. At the end of a pause between two contractions fetal transcutaneous Po₂ has its highest value. Additional evidence that the observed fluctuations in fetal tcPo₂ are the result of the contractions is shown, when labour is stopped by infusion of betamimetics (2).

Fig. 2
Correlation between intrauterine pressure and the fall in tcPo₂ in 8 cases. The line of the least square is shown for each case (from (1,2))

An analysis was made of 8 registrations where contractions were measured internally and where the extent of the contractions varied significantly during the recording time (1,2). Figure 2 shows that the fall in fetal tcPo₂ after contractions is dependent on the contractions' intensity. The fall in fetal tcPo₂ is correlated to the intrauterine pressure.

The practice of administration of supplementary oxygen to the mother during labour and delivery is still under discussion. The consistent increase of fetal transcutaneous Po₂ after supplementary oxygen administration to the mother (3,4) will hopefully be a sufficiently convincing argument for supplementary oxygen administration to the mother when fetal oxygenation is in jeopardy.

Figure 3 shows an individual example of our polygraphic recordings. The arrows indicate a 9-minute interval of supplemental oxygen administration to

Fig. 3

Recordings of fetal tcPo₂ and "flow", fetal heart rate (FHR), uterine contractions (U.C.), maternal tcPo₂ and "flow", maternal heart rate (MHR) and thoracic impedance. During the period between the arrows on the maternal and fetal tcPo₂ recordings the mother breathes 100 % oxygen (from (4)).

the mother. Her tcPo₂ rose from 96 to 380 mm Hg. Fetal tcPo₂ increased to 23 mm Hg from an initial level of 18 mm Hg. An increase of fetal Po₂ of 5 mm Hg during maternal O₂ breathing may be considered to be small, particularly if compared to the large increase in maternal Po₂. This has frequently been cited as a proof that the uterine vessels contract. This misinterpretation is due to the fact that only Po₂ is considered. If, instead, the oxygen saturation or the total oxygen content of the blood is considered, a totally different, and much more accurate result emerges.

Fig. 4
Standard fetal O₂ dissociation curve (Po₂ = 22 mm Hg) to show schematically the effect on saturation when fetal Po₂ increases from 16.5 to 21.5 mm Hg (from (4)).

Figure 4 shows schematically the large effect on fetal saturation when fetal Po₂ increases from 16.5 to 21.5 mm Hg (based on mean tcPo₂ values from (4)). Fetal saturation increases from 30 to about 50 %. Expressed in ml O₂ this would be an increase of 4 ml O₂, producing an increase in oxygen availability to the fetus of as much as 58 %.

Numerous further examples of insights gained by continuous Po₂ monitoring have proven the potentials of the tcPo₂ tool to extend our knowledge in respiratory and circulatory physiology and clinical management of the human fetus during labour.

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