Quantitative T2* measurements of fetal brain oxygenation during hypoxia with MRI at 3T: Correlation with blood gas analyses

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Introduction:

Intrauterine growth restriction (IUGR) is associated with an increased risk of perinatal mortality, morbidity, and impaired neurodevelopment (1-3). Thus methods to assess fetal tissue oxygenation would be desirable to detect the fetus at risk with the option to initiate precautionary or therapeutic measures (e.g. induction of preterm delivery). It has been shown recently that Blood oxygen level dependent (BOLD) MRI is capable of demonstrating changes in fetal tissue oxygenation (4). Thus BOLD might be an important tool to detect and monitor the fetus at risk, which is still a major problem in obstetrics.

MRI delivers only arbitrary units of the signal intensities. To compare signal intensities of different experiments, relative changes of BOLD SI have to be calculated requiring measurements during different phases of oxygenation (e. g. normoxia and hyoxia). The necessity of different oxygenation plateaus is a substantial drawback of this method in the practical application on the human fetus. Quantitative multiecho sequences allow the determination of T2 and T2* relaxation times.

The purpose of this study is the determination of oxygen saturation in the fetal brain during hypoxia by T2* measurements and comparison with fetal blood gas analysis.

Methods: 11 sheep fetuses were examined during a control and hypoxic period on a 3T MRI scanner (Intera, Philips, Netherlands). Multiecho gradient-echo (39 echoes, spacing of 3.9 ms) and turbo-spin-echo-sequences (30 echoes, spacing 5 ms) were performed on the fetal brain. Quantitative T2* and T2 maps were calculated (Figure 1). MR oxygen saturation ($MRSO_{2calc}$) in the fetal brain was calculated using the following formula (5):

$$Y = 100\% \cdot \left[1 - \frac{1}{T2'_{ox} \cdot \lambda \cdot \frac{4}{3}\pi \cdot \Delta\chi_0 \cdot Hct \cdot \gamma \cdot B_0} \right], \text{ where } \frac{1}{T2`_{ox}} = \frac{1}{T2} - \frac{1}{T2*} - \frac{1}{T2`_{FE}}$$

(λ=0.045 blood volume fraction; $T2`_{FE}$=contributes from brain iron and other factors; $\Delta\chi_0$= susceptibility difference fully oxy/deoxygenated blood; γ=gyromagnetic ratio; B_0=field strength). Fetal blood samples were taken from a carotid catheter at control and hypoxic periods to determine blood oxygen saturation. Linear regression analysis was used to compare the calculated $MRSO_{2calc}$ with oxygenation measured from blood samples.

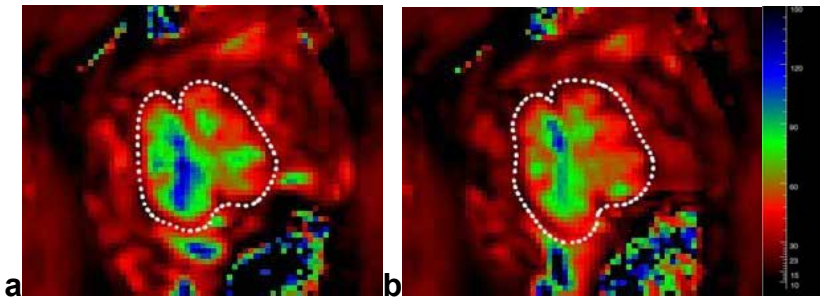

Figure 1 a, b: T2* maps during normoxia (a) and hypoxia (b) with region of interest in the fetal cerebrum. There is a slight decrease of T2* during hypoxia illustrated by a decrease of blue and increase of red and green.

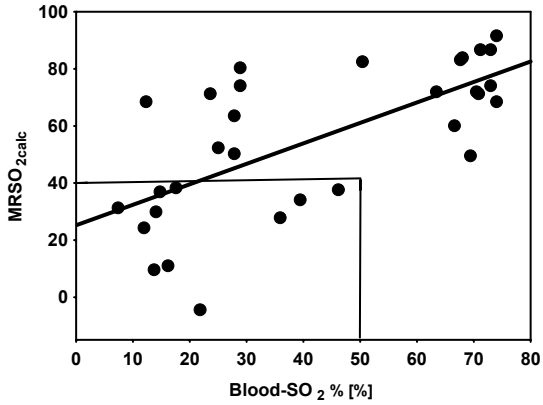

Figure 2: Linear regression of $MRSO_{2calc}$ for cerebrum with blood samples oxygenation.

Results: During control (n=17 measurements) mean values were 71% [95%confidence interval: 62-79] for $MRSO_{2calc}$ and 60% SO_2 [50-69] for blood oxygenation. During hypoxia $MRSO_{2calc}$ decreased to 37% [23-50] and blood oxygenation to 21% [15-27]. $MRSO_{2calc}$ and blood oxygenation correlated significantly (r=0.67; p=0.00003). If an oxygen saturation of less than 40% SO_2 was calculated by MRI, then blood oxygen saturation determined by blood samples was less than 50% SO_2% in all experiments (Figure 2).

Discussion and Conclusions: It is feasible to perform quantitative T2* measurements in the fetal brain.

Although there are some differences between SO_2 measured from MRI and determined by blood samples, there is a reasonable correlation between these two methods. MRI thus has potential to assess oxygen saturation non-invasively and therefore fetal risk. The fact that there is scatter of the calculated SO_2 might result from the various assumptions that have been made for calculation, e. g. brain iron concentration and blood volume fraction in the brain. The estimated SO_2 values ($MRSO_{2calc}$) could be improved with blood volume quantification.

Acknowledgement: We thank the “Deutsche Forschungsgemeinschaft” (DFG) for funding.

References:

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