In-vivo MRI measurement of fetal blood oxygen saturation in cardiac ventricles of fetal sheep: a feasibility study

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Introduction

The basic idea of this study was the application of MRI to non-invasively assess fetal blood oxygen saturation. This is of clinical importance in the management of high risk pregnancies like intrauterine growth restriction (IUGR). The lack of possibilities to measure fetal oxygenation directly, contributes to the fact, that IUGR is still one of the major problems in obstetrics.

Recent studies have suggested that oxygen saturation can be measured accurately and non-invasively using the MR oximetry technique [1-3]. The MR oximetry technique is based on the direct relationship between the T2 relaxation time of whole blood and the blood`s oxygen saturation [4]. This technique has been successfully applied to children with congenital heart disease to determine non-invasively the blood oxygen saturation [5, 6]. In contrast to children and in-vitro measurements the fetal model is more challenging. The main problem is the lack of cardiac triggering. In addition to that, there is respiratory motion of the mother as well as voluntary motion of the fetus. Therefore MRI sequences had to be developed that take these difficulties of the fetal model into account.

The purpose of this study was to assess the feasibility to determine fetal blood oxygen saturation with T2-weighted MR sequences using a fetal sheep model. To perform T2 measurements (1) in-vitro of fetal blood samples, (2) in-vivo in the fetal right and left ventricles.

Material and Methods:

T2-measurements were performed on a 1.5 T scanner using T2 preparation pulse in combination with a 3D balanced steady state free precession sequence repeated at different echo times (TE 30, 60, 90, 120, 150 msec). For in-vitro measurements blood samples from the fetal carotid artery with different oxygenation levels were measured. For in-vivo measurements eight sheep fetuses were examined during a control, hypoxic and recovery phase to perform T2-weighted scans of the fetal blood in the heart. Signal intensities in the left (LV) and right ventricle (RV) were measured to calculate the MR blood oxygen saturation (MR-sO2).

According to the description of the exchange model by Luz and Meiboom [7], the MR-signal S of blood can be written as:

\[ S = A \cdot \exp[-T_2^e R_{180} + \text{hct}(1-\text{hct}) \cdot \alpha \cdot \tau_s \cdot (1-100)(1/T_2 + 1/T_2^e)] \]

where \( T_2^e \) is the echo time, \( R_{180} \) is the relaxation rate (1/T2) of fully oxygenated blood, \( \text{hct} \) is the hematocrit value, \( \alpha \) is a constant, \( \tau_s \) is a time constant related to the exchange rate, and \( Y \) is the oxygen saturation. The spacing between the echo pulses is denoted as t180. The proportionality constant A depends on the receive chain of the MR-system.

During each phase fetal carotid artery sO2 was directly measured and correlated with MR-sO2. A Bland-Altman plot was performed.

Results:

For the in-vitro measurements the T2W signal intensities of each tube were measured with a ROI (size: 80 pixel) for each echo time. The signal values and the curves fitted to them are shown in figure 1. Fetal carotid artery-sO2 was 69 sO2% during control, 16 sO2% during hypoxemia and 67 sO2% during recovery. Mean values of the MR-sO2 were 49 sO2% and 40 sO2% for control, 6 sO2% and 3 sO2% for hypoxemia, 51 sO2% and 43 sO2% for recovery in LV and RV, respectively. Mean values of fetal carotid artery-sO2 and MR-sO2 were highly correlated (LV: r=0.87, p=0.000002 and RV: r=0.89, p=0.000001) (figure 2). According to the bland-altman plot MR-sO2 was lower compared to fetal carotid artery-sO2 (15% in LV and 20% in RV).

Discussion and Conclusion:

Based on our preliminary results it seems to be possible to assess fetal oxygen saturation with MR oximetry. However, for practical application measurements have to be more accurate.

References: