COMPREHENSIVE HUMAN FETAL HEMODYNAMIC ASSESSMENT BY PHASE CONTRAST MRI AND MR OXIMETRY

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Introduction: Fetal blood flow assessment with phase contrast (PC) MRI with metric optimized gating (MOG) is feasible in late gestation fetuses with normal hearts and those with congenital heart disease (CHD) [1, 2, 3]. We have shown good agreement between T2 based MR oximetry measurements and conventional blood gases in the vessels of children with CHD [4]. The feasibility of fetal T2 oximetry has been shown in lambs [5], and human fetal MR oximetry has been performed with susceptibility weighted imaging [6]. We aimed to achieve a comprehensive hemodynamic assessment of the human fetal circulation using fetal MR oximetry with T2 mapping combined with PC MRI.

Methods: Seven normal fetuses and nine fetuses with CHD were studied with MRI on a 1.5T system (Siemens Avanto, Erlanghen, Germany) at a mean gestational age of 37 weeks (SD 1.2 weeks). PC and T2 measurements were made in the major fetal vessels. The PC parameters are described in our previous publications [1, 2]. The T2 mapping sequence involved a T2 preparation sequence followed by a rapid SSFP data acquisition with motion correction [7,8]. T2 mapping parameters were as follows: TEs 0, 50, 100, 150ms, slice thickness 6mm, matrix size 224 × 181, FOV 350mm, NSA 1, parallel imaging factor 2 (GRAPPA), 6/8 phase partial Fourier. 8300 ms of magnetization recovery time was provided between T2 preparation pulses.

Assuming a hemoglobin concentration of [Hb] = 15g/dL in the late gestation fetus [9], we calculated oxygen delivery (DO_2) = umbilical vein (UV) flow (Q_{UV}) × UV oxygen content, where oxygen content = oxygen saturation of hemoglobin [HbO_2] × 1.34 × [Hb] [10]. We calculated fetal oxygen consumption (VO_2) from the product of the UV flow and the arterio-venous difference in oxygen content ($AV\Delta O_2$) between the UV and umbilical artery (UA), using descending aortic (DAo) oxygen content for UA: $VO_2 = Q_{UV} \times AV\Delta O_2$ (UV-DAo) [10]. Normal and CHD fetal VO_2 and DO_2 were compared using a student *t*-test and the correlation between fetal VO_2 and DO_2 was examined with Pearson's correlation.

Results: Figure 1 shows a PC measurement and flow curve from the DAo and T2 maps in the umbilical cord and 3-vessel view in a normal human fetus at 37 weeks gestation. Figure 2 shows that fetal VO_2 correlates with fetal DO_2 ($R^2 = 0.57$). The fetus with the lowest VO_2 (✖) had coarctation of the aorta and clinical evidence of fetal distress at birth with a cord pH of 7.0 and Apgar scores of 3 at 1 minute and 5 at 5 minutes. The placental histology revealed chorangiosis and thrombotic vasculopathy and periventricular leukomalacia was demonstrated on neonatal brain cranial ultrasound and MRI. Table 1 shows fetal DO_2 ($p < 0.01$) and VO_2 ($p < 0.01$) were reduced in the CHD group compared with normals.

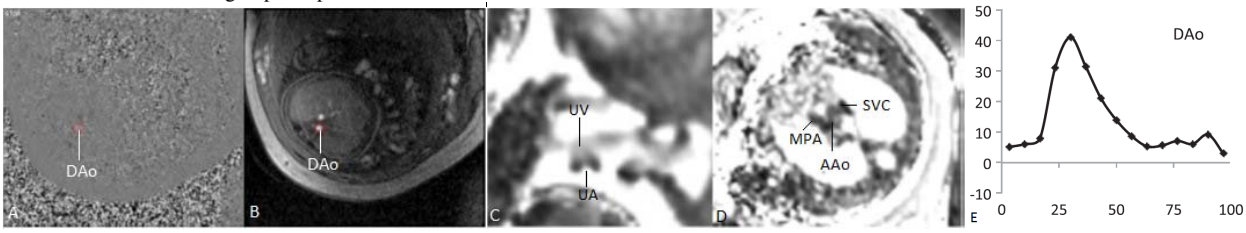

Figure 1- A, B, E show an example PC measurement and flow curve from the DAo made using MOG. T_2 maps showing the umbilical cord (C) and mediastinal 3 vessel view (D) made in a normal human fetus at 37 weeks gestation. The vessel lumen signal intensity indicates the relative oxygen content of the blood in each vessel, with the highest oxygen content in the UV and lowest in the SVC.

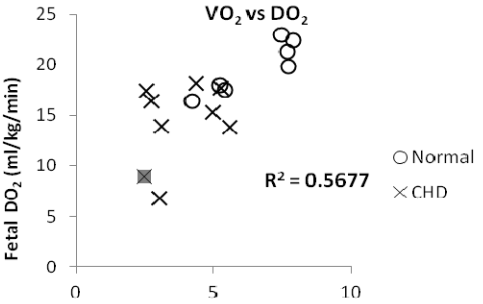

Figure 2- The relationship between fetal DO_2 and fetal VO_2.

Table 1- Oxygen consumption in normal fetuses and fetuses with CHD

	VO_2 (ml/kg/min)		DO_2 (ml/kg/min)	
	Normal	CHD	Normal	CHD
Mean	6.51	3.78	19.78	14.28
SD	1.52	1.25	2.58	4.40
P	0.0014		0.007	

Conclusions: This approach represents the first attempt to non-invasively characterize oxygen delivery and consumption in the human fetal circulation, made possible by combining MR oximetry and PC MRI. The results are in keeping with previous animal experiments showing that fetal DO_2 is matched by VO_2 [10]. We demonstrated evidence of reduced DO_2 and VO_2 due to placental insufficiency in a fetus with aortic coarctation and clinical evidence of fetal distress with associated hypoxic brain injury that was undetected by routine clinical monitoring. An unexpected finding was the lower placental oxygen delivery in fetuses with CHD, which could be due to abnormal placental development and function in the setting of CHD.

References

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