Assessment of late-onset fetal growth restriction by phase contrast MR

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Introduction During the last few weeks of pregnancy, there is rapid growth in the fetal cerebral cortex [1] and this places an increased demand for nutrition on a placenta that is nearing the end of its life. Late-onset fetal growth restriction (FGR) affects up to 10% of all pregnancies and is increasingly being recognized as an important cause of developmental delay in children [2]. The diagnosis is usually based on the demonstration of a reduced pulsatility index (PI) in the middle cerebral artery (MCA) by Doppler ultrasound, with increased umbilical artery (UA) PI also indicative of FGR. However, diagnosis remains challenging and characteristic Doppler changes may be relatively late manifestations of FGR [3]. Recently, metric optimized gating [4] has enabled fetal flow measurement by phase contrast MR. This study investigates MR assessment of late-onset FGR in a small group of patients.

Methods As part of an IRB approved prospective cohort study, five small for gestational age fetuses were studied with Doppler and MR at a mean gestational age of 37 weeks and subsequently confirmed as having late-onset FGR based on a combination of placental pathology, asymmetric growth restriction, and a requirement for urgent delivery. MR data were acquired on a 1.5-T Avanto MR system (Siemens, Germany) according to methods previously reported [5]. Doppler measurements of MCA and UA PI and MR measurements of flow in the major fetal vessels were compared with normal control values using a Mann-Whitney *U* test [5,6].

Results Table 1 shows that compared with normal controls, superior vena caval (SVC) flow was increased in all cases. Doppler showed reduced MCA PI or increased UA PI in patients 1-4, but failed to identify any hemodynamic abnormality in patient 5. MR showed a significant increase in SVC (p<0.001) and arterial ductal (AD) flows (p<0.006) and reduced pulmonary artery flow (PBF) (p=0.06) in fetuses with late-onset FGR.

Discussion The fetal circulation, shown in Figure 1, adapts to reduced oxygen and nutrient supply by redistributing blood flow to different organs [7] through modification of the resistance of various vascular beds and changes in shunting patterns [8]. The MR results presented here are in keeping with the concept of "brain sparing physiology" whereby the fetus responds to placental insufficiency with an increase in cerebral perfusion demonstrated with a rise in SVC flow. The increase in cerebral blood flow may be achieved by an increase in left ventricular output resulting in increased AAo flow, as in patient 5, or through increased right ventricular output with reduced PBF and increased arterial ductal flow supplying retrograde flow to the aortic arch [9], as in patients 1-4.

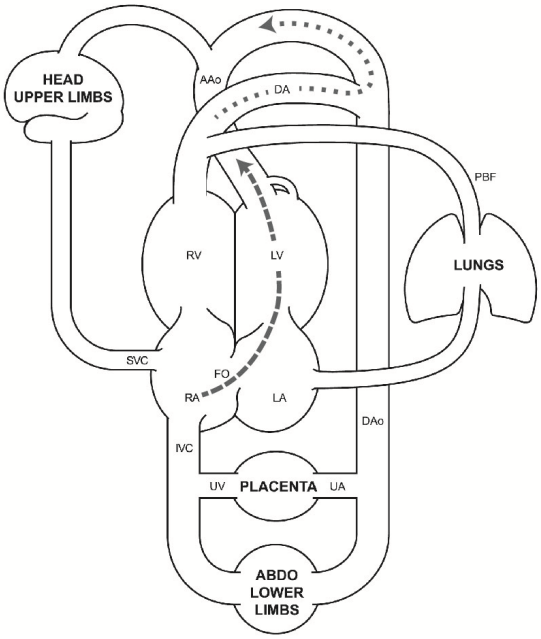

Figure 1 – Fetal circulation showing two possible redistributions supplying increased cerebral blood flow in late-onset FGR. LA-left ventricle; RA-right ventricle; MPA-main pulmonary artery; AAo-ascending aorta; SVC-superior vena cava; AD-arterial duct; PBF-pulmonary blood flow; DAo-descending aorta; UV-umbilical vein; UA-umbilical artery; FO-foramen ovale; LA-left atrium; RA-right atrium.

Conclusions Measurement of the distribution of the fetal circulation by PC MR is feasible in patients with late-onset FGR and may be more sensitive than Doppler at identifying early hemodynamic changes associated with placental insufficiency. By contrast with early-onset FGR, where the complications of prematurity generally lead to conservative management until imminent fetal demise, early detection and delivery in late-onset FGR is associated with a lower risk of complications of prematurity and may reduce the risk of adverse neurodevelopmental outcomes resulting from fetal starvation and hypoxia.

Table 1 – Doppler and MR *in utero* flow assessment of late-onset FGR patients. Gestational age (GA) at the time of the studies is listed. Values below the 5th and above the 95th percentiles are indicated with arrows. Reference normal control MR flows are given as mean ± standard deviation.

		Doppler PI		MR Flow (% of combined ventricular output)						
Patient	**GA**	**MCA**	**UA**	**MPA**	**AAo**	**SVC**	**AD**	**DAo**	**PBF**	**UV**
1	36	1.3	1.1 ↑	65	32	55 ↑	50	23 ↓	17	22
2	41	0.8 ↓	0.9	70 ↑	27 ↓	72 ↑	57 ↑	51	13	22
3	35	0.7 ↓	1.2	64	33	57 ↑	61 ↑	47	3	15
4	35	0.9 ↓	1.9 ↑	68 ↑	29 ↓	41 ↑	60 ↑	52	12	
5	37	1.4	0.7	49 ↓	48 ↑	49 ↑	48	47	5	40
Normals	36 ± 2			60 ± 4	37 ± 4	27 ± 7	41 ± 8	52 ± 12	19 ± 10	30 ± 10

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