

ORAL PRESENTATION

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# MRI reveals increased superior vena caval blood flow in human fetuses with congenital heart disease, abnormal placental pathology and neonatal brain white matter changes

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## Background

Delayed brain development in newborns with congenital heart disease (CHD) results in increased vulnerability to white matter injury before and after cardiac surgery [1]. Doppler showing reductions in pulsatility index (PI) in the middle cerebral arteries (MCA) of fetuses with CHD is interpreted as evidence of “brain sparing physiology”. It supports the hypothesis that *in utero* brain dysmaturation in CHD is due to reduced cerebral oxygen delivery [2]. We were interested in the relationship between cerebral blood flow, placental pathology and white matter changes in CHD fetuses using ultrasound and MRI.

## Methods

Late gestation CHD fetuses and normal controls were studied using Doppler and a 1.5T MRI system (Siemens Avanto). We measured the MCA PI with ultrasound, superior vena cava (SVC) flow and fetal brain weight (EBW) with MRI using our previously published technique [3]. Placental histology was studied. Head ultrasound (HUS) performed after delivery was used to classify the newborn brains as: normal, increased white matter echogenicity (WME), or periventricular leukomalacia (PVL). The fetal parameters were compared in babies with and without brain abnormalities using an unpaired Student *t*-test with  $p < 0.05$  taken as statistically significant. Odds ratios for abnormal brain imaging, placental abnormalities and SVC flow were calculated.

## Results

Forty four fetuses with CHD [mean gestational age (GA): 36 weeks, SD 1.4 weeks] and forty normal fetuses (mean GA: 37 weeks, SD 1.3 weeks) were studied. There was no difference in MCA PI between normal and CHD fetuses with or without white matter changes at birth (Fig. 1a, Table 1). 34% of neonates with CHD had increased WME on HUS and 7% had PVL. Fetuses with changes on brain imaging at birth had higher SVC flow than normals ( $p = 0.03$ ) (Fig. 1b) and smaller brains than those with normal HUS ( $p = 0.05$ ) (Fig. 1c). Elevated SVC flow was associated with a markedly increased risk of PVL (OR: 41,  $p = 0.005$ ). CHD fetuses with WME had high incidence of histologically abnormal placenta (OR: 6.1,  $p = 0.04$ ) (Fig. 1d).

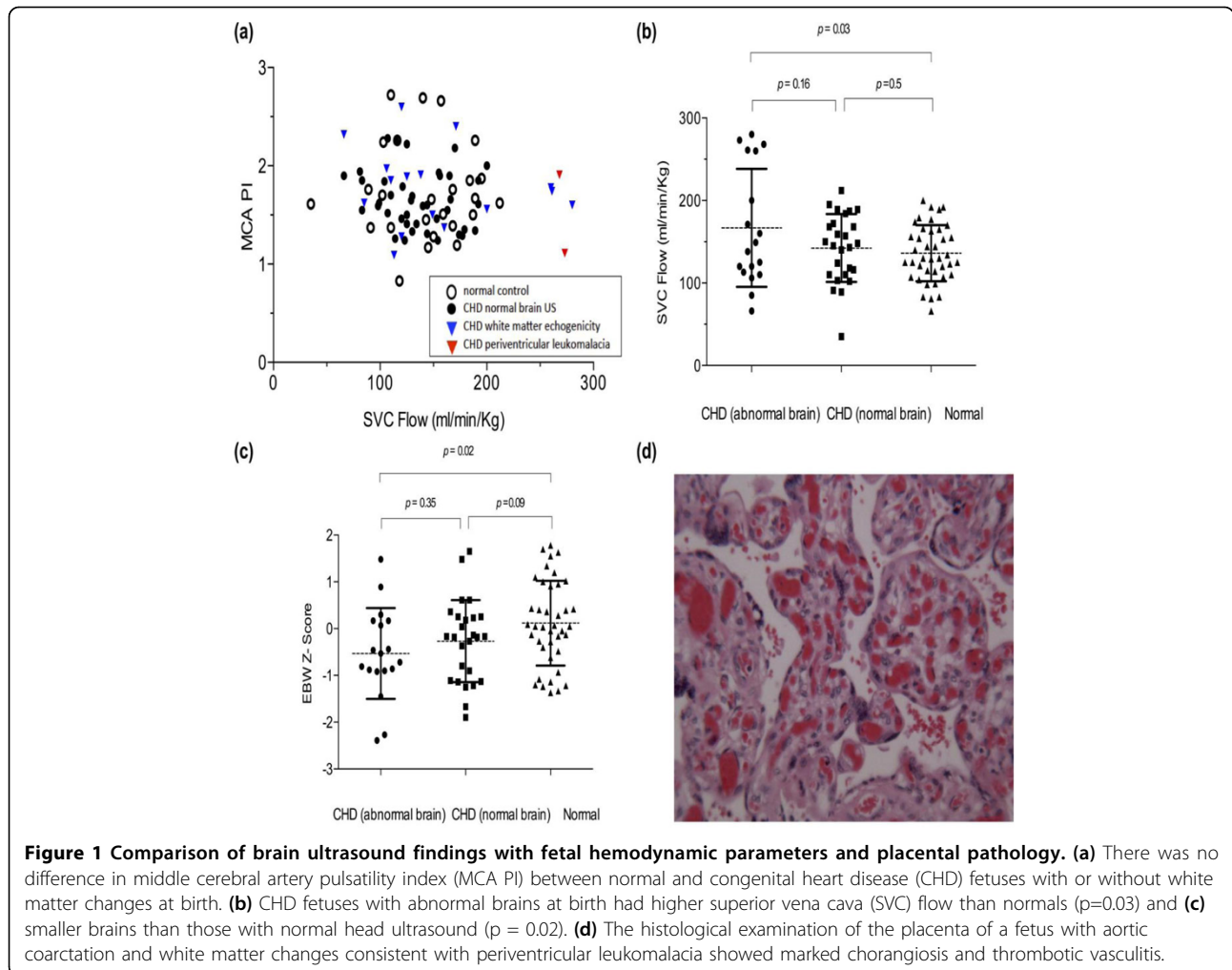
## Conclusions

In keeping with previous studies, we found smaller brains and a high incidence of WME and PVL in newborns with CHD. Abnormally high SVC flow, in keeping with “brain-sparing physiology” appears to be highly associated with PVL, suggesting that the combination of CHD and placental disease may be dangerous for the immature white matter in CHD newborns. Abnormal SVC flow by fetal cardiac MRI may be a more useful indicator of increased risk for white matter injury than MCA PI during late gestation, and could indicate early delivery by Cesarean section.

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**Table 1 Head ultrasound findings, fetal hemodynamic parameters and placental abnormalities.**

	CHD Abnormal Brain	CHD Normal Brain	Normal
EBW z-score	-0.53 ± 0.23 (n=18)	-0.27 ± 0.17 (n=26)	0.12 ± 0.14 (n=40)
SVC Flow (ml/min/kg)	166.9 ± 16.86 (n=18)	142.5 ± 8.08 (n=26)	136.1 ± 5.38 (n=40)
MCA PI	1.75 ± 0.1 (n=18)	1.74 ± 0.1 (n=25)	1.63 ± 0.05 (n=40)
Placental Abnormalities (%)	81.3	59.1	N/A

CHD: Congenital heart disease; EBW: Estimated brain weight; SVC: Superior vena cava; MCA PI: Middle cerebral artery pulsatility index

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