

Peripheral flows in fetuses with ductal-dependent congenital heart defects

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Introduction: Abnormal flows in fetal middle cerebral artery (MCA) and umbilical artery (UA) are generally associated with placental insufficiency, but they may also be due to changed hemodynamic conditions in various congenital heart defects (CHDs). Our aim was to examine the influence of ductal-dependent CHD type on MCA and UA flows, as well as on the fetal head growth.

Methods: We retrospectively reviewed echocardiograms of normal fetuses (n=1897), cases with hypoplastic left heart syndrome (HLHS; n=373) and CHD associated with pulmonary atresia (PA; n=190). Fetuses with major extracardiac defects, chromosomal abnormalities, small for gestational age, from multiple pregnancy, and of mothers with pregestational diabetes and hypertensive disorders were excluded from the study. We measured fetal head circumference (HC), pulsatility indices (PI) in UA and MCA and cerebroplacental ratio (CPR = MCA PI/UA PI).

Group	Normal (n=1897)	HLHS (n=373)	PA (n=190)
Gestational age [weeks]	13-40 (median 26)	13-40 (median 26)	14-40 (median 27)
No of measurements			
MCA PI	1346	348	171
UA PI	1451	356	175
CPR	1333	343	166
HC	1885	366	188

Results: In HLHS fetuses lower MCA PI and CPR and higher UA PI values were observed ($p < 0.05$). In the group with PA, UA PI and CPR values were higher, ($p < 0.05$), and MCA PI were comparable to normal. UA PI was increased more in HLHS than in the PA group. Head circumference was slightly lower, yet within normal limits in HLHS group (borderline significant), with no difference between normal and PA groups.

Conclusions: Observed peripheral flows in HLHS and PA groups are consistent with hemodynamic changes in these CHDs. Increased UA PI values are probably secondary to diastolic steal, which is more pronounced in case of HLHS than in PA (cerebral vascular resistance lower than pulmonary vascular resistance). Cerebral flow is impaired in HLHS both in systole and diastole (filling only retrograde through hypoplastic aortic arch), which gives another reason for decreased MCA PI, and is not the case in PA fetuses. Observed difference in head growth in HLHS fetuses, however mild, may be due to impaired blood flow to the head region.