Analysis of Neonatal Cardiac Function in Infants with and without Patent Ductus Arteriosus

Kathryn M Broadhouse[1,2], Anthony N Price[1,2], Giuliana Durighel[1], Anna E Finnemore[1,2], David J Cox[1], A David Edwards[1,2], Joseph V Hajnal[1,2], and Alan M Groves[1,2]

[1]Imaging Sciences Department, MRC Clinical Sciences Centre, Imperial College, London, United Kingdom, [2]The Centre for the Developing Brain, Imaging Sciences & Biomedical Engineering Division, King's College, London, United Kingdom

Target Audience

Neonatologists, Cardiologists.

Background

Persistent patent ductus arteriosus (PDA) (an extra-cardiac shunt remnant of fetal circulation) remains a common clinical presentation in preterm infants and is clinically apparent in around 60% of infants born before 28 weeks gestation [1]. Using phase contrast MRI we have previously shown that shunt volumes can be up to 74% of left ventricular output[2] (LVO), that even in the presence of large shunt volumes total systemic blood flow can be maintained and that it is done so by a significantly increased LVO. From observation, infants with PDA that result in large shunt volumes appear to have enlarged hearts (fig1), but the extent of remodelling and its impact on cardiac function is yet unknown.

Objective

The aim of this study was to quantify ventricular dimension and function in "healthy" neonates using 2D SSFP stacks. Then compare PDA infants to this normative range to determine the impact of shunt volume.

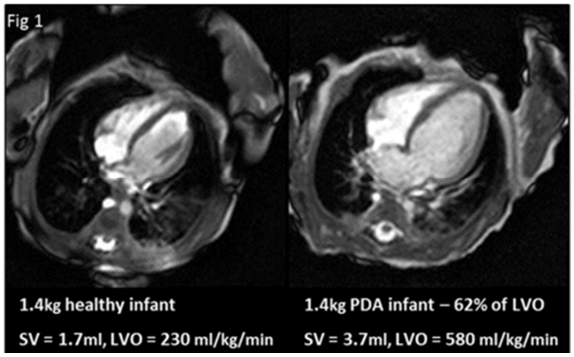

Figure 1- 4 chamber view in a 1.4kg control infant (left) and PDA (right) infant with a shunt volume of 62% of LVO. Figure shows *the apparent increase in left ventricular dimensions.*

Methods

2D SSFP stacks providing full coverage of the left ventricle (LV) were acquired in neonates with and without PDA. PDA shunt volume was calculated from phase contrast (PC) sequences[2] acquired during the same MRI scan. Scans were performed at 3T (Philips Achieva system) using a specialised 8 channel pediatric body receive coil for infants above 2kg and a small extremity receive coil for infants below 2kg. Infants were scanned with ear protection, routine monitoring and without sedation/anesthesia[3]. Optimized 2D SSFP short axis 10-slice stacks[4] (in plane resolution = 1x1mm, slice thickness = 4mm, TR/TE = 3.8/1.9 ms) were placed over the heart and aligned with the mitral valve using previously acquired pilot scans. Negative slice gap was adjusted for individual infants so that the 10 slices covered apex to base of left ventricle. Between 2 and 8 averages were obtained depending on size and calmness of infant, resulting in acquisition times between 2 and 8 minutes. Segmentation and quantification was carried out using freely available Segment[5] software, and the 17 sector AHA model for wall analysis. Description of shunt volume quantification is described previously [2]. Left ventricular (LV) mass, end diastolic volume (EDV), wall thickness and ventricular function parameters of stroke volume (SV), LVO, ejection fraction (EF), and fractional thickening (%thick) were quantified for both "healthy" (control) and PDA infants. The 2 groups were compared using unpaired student t-tests. Impact of shunt volume was analysed with multiple linear regression, p-values ≤ 0.05 being significant. Statistical analysis was carried out in Excel (Microsoft). Frank Starling curve was plotted to determine effect of PDA.

Results

27 control infants median(range) corrected gestational age (GA) $34^{+4}(28^{+3}\text{-}39^{+3})$weeks, weight at scan 1730(790–3050)grams and 12 PDA infants (determined by echo prior to MRI) corrected GA $29^{+5}(27^{+3}\text{–}36^{+1})$weeks, weight at scan 1100(660–2400)grams were scanned. Shunt volume ranged from 7-74% of LVO[2]. t-tests showed significant difference in SV, EDV, ED wall thickness and LV mass between control and PDA infants when normalized by weight at scan. There was a significant association between shunt volume and increased LV mass when correcting for postnatal age and corrected GA. However there was no significant difference in EF and %thick between the two groups (p-value=0.6 and 0.75). The group estimated Frank Starling curve showed no evidence of reaching any maximum, i.e. even PDA babies with very large SV remained on the same linear trend line (fig2).

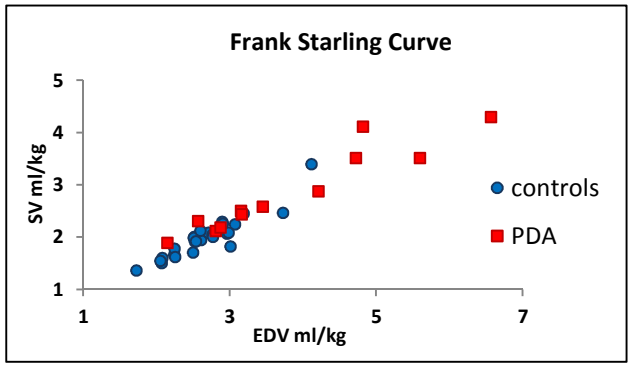

Figure 2- population Frank Starling curve for controls and PDA

Conclusion

Although LVO, EDV and LV mass are significantly increased in PDA infants, EF and %thick are not. In addition none of the PDA infants appear beyond the peak of the Frank Starling curve. This would suggest that function is maintained in these enlarged hearts.

References

[1] – K.Broadhouse *et al*. proc ISMRM 2011, 1195
[2] - J. Wyllie. Seminars in Neonatology 2003; 8:425-32.
[3] – N.Merchant *et al*. *Early Human Development* 2009. 85:779-83.
[4] – A.N.Price *et al*, MRM 2012 in press
[5] – E.Heiberg *et al*. BMC Medical Imaging 2010, 10:1