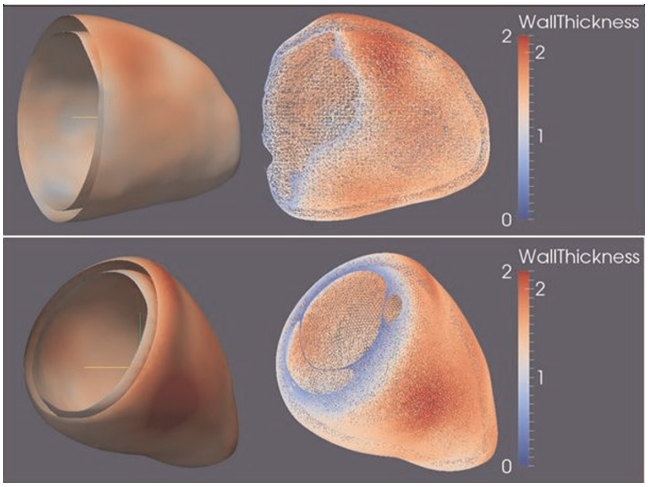

Abstract PS-011 Figure 1

Results Weight-corrected left ventricular mass (LVM, g/kg) and end-diastolic volume (EDV, cm^3/kg) for the preterm cohort at term-corrected age (LVM - mean 1.89, 95% CI 1.89 ± 0.21; EDV – mean 3.42, 95% CI 3.42 ± 0.34) were significantly greater than both the preterm cohort at birth (LVM 1.05, 1.05 ± 0.08, p = 0.0002; EDV 4.89, 4.89 ± 0.59, p = 0.0008) and healthy term controls (LVM 0.95, 0.95 ± 0.18, p = 0.001; EDV 2.16, 2.16 ± 0.38, p = 0.0006).

Conclusions Neonatal MRI with manual ventricular segmentation quantifies preterm gross ex-utero left ventricular growth, highlighting differences from in-utero cardiac development. Increases in preterm LVM and EDV may represent pathological remodelling or physiological ex-utero adaptation.

We have constructed provisional computational atlases that currently allow visual comparisons of size and shape, but which after further analysis will enable more sophisticated quantification and characterisation of preterm ventricular growth and remodelling.

PS-012 LEFT HEART STRUCTURE AND FUNCTION IN 6-YEAR-OLD CHILDREN BORN EXTREMELY PRETERM

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Background and aim Preterm birth has been associated with myocardial remodelling, arrested vascular growth, higher blood pressure and ventricular hypertrophy later in life. The aim of this study was to evaluate left heart structure and function in 6-year-old children born extremely preterm.

Method Children born extremely preterm (EXP; <27 weeks of gestation) in Sweden 2004 to 2007 and matched controls born at term were included. Left ventricular mass index (LVMI), left ventricular end diastolic diameter (LVED) and fractional shortening (FS) were determined by echocardiography. Blood pressure, weight and height were also measured.

Results EXP-children (n = 88; mean GA 25.1 w; BW 817 g) were significantly shorter than controls (mean heights 117.8 and 122.8 cm, p < 0.001). LVMI was 72.1 g/m^2 in EXP and 79.6 g/m^2 in controls (p < 0.01). LVED in EXP was (43.8 mm/m^2) and in controls (42.3 mm/m^2; p < 0.05), unadjusted EXP (35.9 mm) and controls (38.7 mm: p < 0.001). FS was 36% in EXP and 35% in controls (n.s). Unadjusted systolic blood pressure was 2.2 mmHg lower in EXP compared to controls (p < 0.05) but this difference disappeared after taking length into account.

Conclusion Although the shape of the heart differed (larger LVED in EXP), there was no left ventricular hypertrophy or other obvious signs of myocardial dysfunction in 6-year-old children born extremely preterm as compared to age-matched controls born at term. Further cardiac follow-up at older age is warranted and analyses of myocardial strain using two dimensional speckle tracking are underway.

PS-013 ARTERIAL-VENTRICULAR COUPLING IN PRETERM INFANTS BELOW 30 WEEKS OF GESTATIONAL AGE

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Background and aim The model of arterial-ventricular coupling (AVC) describes the interaction of the left ventricle (E_{LV}) with the arterial system (E_A) by the AVC-ratio (AVC = E_A/E_{LV}). Aim was to apply the model to preterm haemodynamics and to analyse time courses of AVC, E_A and E_{LV} in sick preterms with either pulmonary hypertension (PH-group) or haemodynamically significant patent ductus arteriosus (hPDA-group) and in stable preterms with uncomplicated postpartal course (control-group).

Methods Study period was from 10/2009 to 12/2012. Patient recruitment criteria were as follows: anti-PH treatment due to (supra-) systemic pulmonary pressure on echocardiography (PH-group); presence of PDA with an enddiastolic maximal velocity in the left pulmonary artery (LPAdia) ≥ 0,2 m/s and negative history of PH (hPDA-group); neither anti-PH treatment nor catecholamines, PDA with an LPAdia < 0,2 m/s and a ratio of the left atrium/aorta < 1,4 (control-group). AVC was calculated from blood pressure and M-mode measurements. Selected time points were set from days 1–3, 4–7 and 8–30 respectively.

Results Twentyone preterms were recruited to the PH-group, 19 to the hPDA-group and 63 to the control-group. AVC was lower in the PH- and hPDA-group than in the control-group (p = 0,05). E_{LV} was higher in the PH-group (p = 0,007) and both E_A and E_{LV} were lower in the hPDA-group (E_A: p = 0,0002; E_{LV}: p = 0,02).

Conclusion The AVC-ratio was lower in sick preterms. Higher E_{LV} in PH results from interventricular interdependence with decreased LV-filling. Lower E_{LV} and E_A in PDA result from LV-volume-overload and systemic steal-effect. Applying the AVC-model may facilitate explaining preterm haemodynamics.