Background Preterm infants and especially very low birth weight (VLBW) preterms are prone to suffer from cardiac stress due to bronchopulmonary dysplasia (BPD) or hemodynamically significant patent ductus arteriosus (hsPDA). Tissue-Doppler imaging (TDI) based strain and strain rate measurements are ultrasound techniques that so far have not been used to assess cardiac function in this population.

Aim of this study was to assess TDI based strain and strain rate by in VLBW infants and their correlations with the infants’ clinical courses within the first 28 days of life.

Methods We conducted ultrasonic measurements on days 1, 7, 14 and 28 of life in 119 preterm infants with a birth weight below 1500 g. We assessed peak systolic strain (PSS) and strain rate (PSSR) and compared these parameters depending on weight, weight at examination and heart rate as well as the presence of a PDA or development of BPD.

Results PSS and PSSR of the right ventricle were significantly lower than left wall strain on days 14 that only increased insignificantly after closure of the PDA. Incipient BPD was associated with significantly lower PSS in the right wall on days 14 and 28 of life.

Discussion Although BPD and hsPDA are highly intercorrelated, significant differences of strain and strain rate could be shown between groups. Benefits of clinical applications, however, remain to be assessed.

Background Sick preterm neonates may have significant cardiac dysfunction. Blood pressure (BP) may be a surrogate marker however mean BP alone does not indicate the nature of myocardial dysfunction.

Aim To analyse biventricular myocardial velocities and myocardial performance indices (MPI) using tissue Doppler imaging (TDI) in preterm neonates <30 weeks gestation, with and without hypotension, in the first 24 h of life.

Methods 25 preterm neonates were recruited: 15 were normotensive and 10 were hypotensive. The hypotensive group (HT) received between 1 and 5 interventions (fluid and inotropes) till the first 24 h of life. Methods 25 preterm neonates were recruited: 15 were normotensive and 10 were hypotensive. The hypotensive group (HT) received between 1 and 5 interventions (fluid and inotropes) till the first 24 h of life.

Results The left ventricular (LV) MPI was significantly higher in the normotensive group compared to the hypotensive group (p = 0.01), suggesting left ventricular dysfunction. Biventricular MPIs decreased significantly when hypotension was corrected, indicating an improvement in myocardial function (RV p = 0.01, LV p = 0.05). Trans-mitral E’ also showed an improvement following intervention for HT suggesting improvement in left ventricular relaxation (p = 0.02).

Conclusion Although our study is small we have demonstrated that hypotensive preterms have impaired left ventricular function. Myocardial function improved after intervention in the hypotensive group. More studies are needed to investigate the application of TDI as an adjunct in clinical decision making when managing preterm babies with hypotension.

Background Early cardiac re-modelling in preterm animals and increased left ventricular mass (LVM) in young adults born preterm have been reported. We investigated LVM in infants for early adaptational myocardial changes during 6 months after preterm birth.

Method Longitudinal echocardiographic study measuring LVM in 25 preterm infants (GA 26–30) directly after birth, at term and 3 months post-term, and comparison to 30 age-matched term children after correction for body surface area (m²).

Results LVM/m² increased with 78% during the first three months after preterm birth (37.43 to 66.73 g/m²) compared to 13% in controls (49.39 to 55.70 g/m²). At term, LVM/m² was significantly higher in the preterm group (66.73 vs 49.39 g/m², p < 0.001). Preterm infants developed even more absolute LVM (12.79 vs 10.79 g, p = 0.02) although they were slightly lighter (3.18 vs 3.45 kg).

At three months of corrected age, relative LVM decreased, and no significant differences could be shown between the groups.

Conclusion Preterm infants develop an immediate but transient increase in LVM. Premature myocardial maturation, increased afterload and a narrower vascular tree might be responsible for left ventricular hypertrophy. The impact on short and long term left ventricular function is still unclear and has to be explored.