Background and aims Neurodevelopmental disorders are common in children with congenital heart disease (CHD) and largely ascribed to prenatal factors such as impaired cerebral growth. It remains to be established whether this is due to impaired intratermic cerebral blood flow or genuine genetic causes. Down syndrome (DS) is a known cause of CHD, neurodevelopmental disorders and microcephaly. Hence, studies on DS may provide insight into the causes of impaired cerebral growth in CHD. We aimed to assess the risk of microcephaly in children with DS and CHD compared to children with DS and no CHD.

Methods Children with DS (n = 389) and specific birth characteristics were identified in national registries. Head circumference and the risk of microcephaly (head circumference < -2SD) was compared between children with CHD (n = 168) and children without CHD (n = 221) by linear and logistic regression analyses (unadjusted and adjusted for gender and gestational age).

Results There was no difference in head circumference between the groups, 0.0 cm (95% CI -0.4–0.4). Adjustment did not significantly alter the results. The risk of microcephaly was slightly higher in newborns with CHD, OR 1.4 (95% CI 0.8–2.6). Adjustment did not significantly alter the results.

Conclusions We did not find indications of impaired head growth in children with DS and concomitant CHD. There might be a slight increase in the risk of microcephaly. We suggest that the most common types of CHD in DS i.e. atrioventricular septal defects, ventricular septal defects and atrial septal defects do not impair prenatal cerebral growth in children with DS.

Background and aim The waveform amplitude produced by pulse oximeters can be expressed as an index of pulsatile vs. non-pulsatile signal. This perfusion index (PI) has been shown to correlate with cardiac output, stroke volume, and superior vena cava flow. The aim was to gather PI reference data in preterm infants and to explore if the PI is associated with common clinical parameters.

Patients/methods The PI was recorded in 312 neonates <32 weeks GA during the first 72 h of life. Mixed-effects modelling was applied with PI as the dependent variable and the individual clinical parameters.

Results Mean GA was 28.5 weeks (SD ± 2.1). A quadratic model (0–24 h) combined with a linear model (24–72 h) provided the best fit. The lowest PI was reached 12–18 h after birth, thereafter gradually increasing until 72 h postnatal age. For the first 24 h PI was associated with gender (coefficient 0.05, p = 0.04), inotrope administration (-0.123, p < 0.0001), pulse pressure (0.014, p < 0.0001), SaO2 (-0.015, p < 0.0001), MABP (-0.013, p < 0.0001), and GA (0.014, p = 0.0168). After the first day, only associations with, inotrope administration (-0.17, p < 0.0001), pulse pressure (0.007, p < 0.0001), MABP (-0.014, p < 0.0001), and SaO2 (-0.01, p < 0.0001) remained. No association was found with IVH, PDA, fluid boluses, or birth weight.

Conclusions The evolution of PI values over time probably reflects transitional physiology. The associations with pulspressure, MABP, and inotrope administration suggest that the PI might have an application in blood pressure management.