Conclusions: Prediction of neonatal neurological morbidity might be greatly improved by fetal cardiovascular evaluation, influencing prenatal and neonatal management. The association of prenatal cardiovascular parameters with long-term neurodevelopment merits further investigation.

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ASSESSMENT OF GESTATIONAL AGE IN VERY PRETERM NEONATES USING CEREBELLAR MEASUREMENTS AT CRANIAL ULTRASOUND - WHAT IS THE BEST APPROACH?

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Background and aims: Clinical assessment of gestational age (GA) can be challenging. Several ultrasound approaches to estimate GA using cerebellar measurements have been reported, claiming to be simpler and more accurate than clinical assessment. We compare the accuracy of 3 previously described cerebellar measurements for estimating the GA in VLBW infants.

Methods: We studied prospectively VLBW infants under 32 weeks GA defined by certain dates, IVF date or early fetal ultrasound. We excluded infants with IUGR, neurological problems or scan abnormality except isolated GLH/transient flares. Ultrasound acquisition and cerebellar measurements were made by author AG. Measurements of the vermis anterior-posterior diameter (APD, Cuddihy 1999), transverse cerebellar diameter via anterior (TCDa, Makhoul 2000) and mastoid fontanelles (TCDm, Davies 2001) were obtained. Estimated GA was calculated using published equations, and compared to known GA using intraclass correlation coefficient (ICC).

Results: We studied 60 infants. Mean birth GA was 28.4 weeks and mean postnatal age at scan was 1 week. ICC was 0.801 for APD, 0.123 for TCDa and 0.802 for TCDm ($p < 0.001$). Best estimates of GA were obtained with TCDm (difference SD 1 week) and APD (difference SD 1.3 weeks).

Conclusions: APD and TCD gave good estimates of GA and can be recommended. We obtained similar measurements of TCD via the two approaches, but only the equation for the mastoid fontanelle gave a good estimate of GA with excellent ICC. Studies assessing the use of cerebellar measurements for estimating GA in growth restricted, neurologically abnormal and older infants are needed.

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POST-HAEMORRHAGIC VENTRICULAR DILATATION AND ADC MEASUREMENTS IN THE WHITE MATTER IN PRETERM BORN INFANTS AT TERM EQUIVALENT AGE

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Objective: Post-haemorrhagic ventricular dilatation (PHVD) might cause additional white matter (WM) injury due to increased pressure on surrounding tissue. The extent of associated WM injury seems to be the main predictor for an adverse neurodevelopmental outcome.

Aim: Assessing WM injury in preterm born neonates with PHVD at term-equivalent age (TEA) by measuring the ADC on diffusion-weighted MRI (DW-MRI).

Methods: In this retrospective patient-controlled study, 23 preterm infants with PHVD (median GA 27.3 weeks (range 25.6-30.6)), admitted to our neonatal intensive care unit (NICU), were matched to 23 control patients for gender and GA (median GA 27.4 (range 25.3-30.9)). DW-MRI was performed in all neonates at TEA. Regions of interest were drawn manually on the ADC-map with equal size and location in the frontal, parietal and occipital WM bilaterally.

Results: Results are presented in table 1. PHVD was associated with slightly higher ADC-values in the occipital WM ($p < 0.05$). No significant differences in ADC-values of the frontal and parietal WM were observed between both groups.

ADC ×10-3mm2/s (mean ± SE)	PHVD N=23	No PHVD N=23	p-value
Frontal	1.566±0.020	1.572±0.023	0.830
Parietal	1.632±0.022	1.594±0.025	0.288
Occipital	1.549±0.024	1.468±0.024	<0.05

[Table 1]

Conclusion: Preterm born neonates with PHVD showed slightly higher ADC-values of the occipital white matter using DW-MRI at TEA. The lack of a more striking difference may be due to early treatment of PHVD at our NICU, initiated before the P97 +4mm line is crossed.

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IS CEREBRAL OXYGEN SUPPLY COMPROMISED IN PRETERM INFANTS UNDERGOING CLOSURE OF PATENT DUCTUS ARTERIOSUS (PDA)?

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Background: A hemodynamically important PDA is associated with increased morbidity and should be closed pharmacological or surgical. Studies showed that surgical closure contains a risk for adverse neurodevelopmental outcome.

Objective: To monitor cerebral oxygenation by near-infrared spectroscopy (rScO2) in 3 groups of preterm infants: 9 controls without PDA (CTRL); 9 infants with pharmalogical closure (INDOI); and 9 infants with surgical closure (SURG). Monitoring started before treatment up to 48h after treatment. Infants were matched for GA, BW and severity of illness. Infants had volumetric 3D-MRI at 40wks to calculate cerebral tissue volumes.

Results: GA and BW of three groups were 26.9±0.6, 26.8±1.1 and 26.4±1.0wks, and 928±185, 917±155 and 896±141g respectively. Lowest mean rScO2 values±SD before/during treatment were 58±6% for INDO and 53±7% for SURG vs CTRLs: 65±5% (reference values: 63-71%), p< 0.001. Brain volumes are shown in table1. Linear regression between ventricle volume and rScO2 showed a negative correlation: r=-0.59, p< 0.01 and r=-0.74, p< 0.02 for SURG only.

	Ventricle volume	white matter	gray matter	total volume
CTRL	11 ± 3	158 ± 22	160 ± 36	390 ± 33
INDO	12 ± 2	143 ± 15	161 ± 14	374 ± 20
SURG	15 ± 7*	153 ± 18	153 ± 22	383 ± 29

*[table1: * p*

Conclusion: Lowest rScO2´s were found in the SURG group, whereas ventricle volumes tended to be larger. This, and the reverse relation between rScO2 and ventricle volume in the SURG group may indicate hypoxia-induced brain tissue atrophy which (partly) explain the higher incidence of adverse outcome.

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CELLULAR IMMUNE RESPONSE AFTER HYPOXIC ISCHEMIC BRAIN INJURY IN NEONATAL MICE PERSISTS FOR MONTHS

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Background and Aims: Hypoxic ischemia (HI) induces inflammation in the brain which can aggravate the damage. Immune cell infiltration into brain after hypoxic ischemia has previously been studied, but the timing and temporal interaction of these cells in the immature brain have not been investigated in detail before. Our aim was therefore to characterize the innate and adaptive (specific) immune response after HI.**Methods:** Using FACS and immunohistochemistry, innate (microglia, dendritic monocytes and neutrophils) and adaptive (B and T) cells in the brain parenchyma and spleen after brain injury were investigated. We used the Vannucci model to induce HI by unilateral electrocoagulation of the common carotid artery and subsequent hypoxia (10% O_2 for 60 min) in 10-day-old mice. We used a number of activation, innate and adaptive cell markers at 24, 48, 72h, 1, 2 weeks and 3 months after the insult.

Results: Activation of the innate immune cells was mainly seen up to one week after HI in the damaged brain hemisphere and in the spleen. However, in the spleen, a long term activation of CD11b+cells was also found. The adaptive (specific) immune response was activated mainly at later timepoints (72 hrs up to 3 months after HI) both in the brain and in the spleen.

Conclusions: The adaptive immune response sustained for months after brain ischemia. The functional consequences of this activation need to be studied further, but detrimental autoimmune effects or protective neuromodulation are two possible suggestions.