

**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 (rScO<sub>2</sub>) in 3 groups of preterm infants: 9 controls without PDA (CTRL); 9 infants with pharmacological 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 rScO<sub>2</sub> 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 rScO<sub>2</sub> 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 rScO<sub>2</sub>’s were found in the SURG group, whereas ventricle volumes tended to be larger. This, and the reverse relation between rScO<sub>2</sub> 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<sub>2</sub> 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.