Conclusions: Computerized WM training may be an effective intervention to reduce learning and memory deficits in ELBW adolescents.

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THE COURSE OF APPARENT DIFFUSION COEFFICIENT AFTER PERINATAL ARTERIAL ISCHEMIC STROKE

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Introduction: After a perinatal arterial ischemic stroke (PAIS), ischemic tissue can be clearly visualised using diffusion weighted imaging (DWI) and the derived apparent diffusion coefficient (ADC) map. Aim of this study was to quantify the course of ADC values of the stroke throughout the first week.

Methods: Term born infants born between 2003 and 2010, whose PAIS was confirmed using DWI within the first week of life were included. A second scan was acquired at the age of three months and included an IR and T2 sequence.

Regions of interest were drawn on the ADC map of the first scan in the area which evolved to an area of cavitation on the second scan. Linear regression analysis was performed to assess the relationship between postnatal age (hours) and the ADC value.

Results: Forty-two infants were scanned. Territories involved included MCA main branch (10), MCA cortical branch (17), MCA lenticulostriatal (1), PCA (7) and watershed areas (7). Mean postnatal age at scan was 105h (range: 57-167h). When analyzing the whole group the postnatal age correlated with the ADC value (R^2=0.38,p< 0.001). This correlation was also present in the MCA main and MCA cortical branches subgroups (R^2=0.54,p=0.02 and R^2=0.58,p< 0.001 respectively).

Conclusion: Between day 2 and 7 a significant correlation between postnatal age and ADC value of the stroke area was found in the core of the ischemic tissue. Although a small number of scans was used, this correlation might be useful to estimate the timing of the onset of stroke.

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EVALUATION OF DEXTROMETHORPHAN AS NEUROPROTECTIVE STRATEGY IN NEONATAL HYPEROXIC BRAIN INJURY

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Background and aims: Hyperoxia is a high risk factor in the pathogenesis of preterm brain injury. Oxygen, at supraphysiological concentrations has been shown to cause an increase in free radical formation, inflammatory response and apoptotic cell death and subsequently contributes to brain injury. Dextromethorphan (DM) has been shown to be protective against inflammation-mediated, hypoxic-ischemic and excitotoxic brain injury. This study focuses on potential effects and underlying mechanisms of DM in a neonatal animal model of hyperoxic brain injury.

Methods: On postnatal day six (P6) rat pups were randomly injected intraperitoneally with i) DM 5µg/g body weight, (bw), ii) DM 25µg/g bw and iii) PBS as control. Subsequent to the injection rats were exposed to either normoxia (21% O2) or hyperoxia (>90% O_2) for 24 hours. Endpoint was set at P7. Western Blot (WB) and immunohistochemistry analysed the effect of DM on apoptosis by activation of caspase-3 and on pro- and anti-inflammatory cytokines (interleukin (IL) 18 and 10).

Results: A single dose of DM (25µg/g bw) significantly reduced the number of caspase-3 positive cells after hyperoxia in temporoparietal, parietofrontal and retrosplenial cortex and in occipital and frontal white matter. Preliminary data indicate that IL-10 levels analysed by WB are decreased by hyperoxia and restored to normal levels by administration of DM. DM showed no effect on interleukin 18 expression.

Conclusion: This study shows a protective effect of DM against hyperoxia-induced apoptotic cell death and inflammatory responses in the newborn rat brain.