

not the neonatal scan, we speculate that NS may have a negative effect on hippocampal development. However, these results could also be explained by unmeasured neonatal hypoxic-ischemic injury. The findings are in keeping with studies that suggest NS are an independent risk factor for adverse neurodevelopmental outcome. Further studies are needed to confirm whether seizures harm newborns.

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1087 INVESTIGATE OF S100B PROTEIN IN SERUM AS PROGNOSTIC MARKER FOR BRAIN INJURY IN TERM NEWBORN INFANTS WITH HYPOXIC ISCHEMIC ENCEPHALOPATHY

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Background Further to investigate whether increased S100 levels in serum are correlated with the grade of HIE after perinatal asphyxia, mechanical ventilation in some severe cases of the asphyxiated infants and more specifically whether increased S100 predicts the cerebral injury and subsequent cerebral palsy.

Methods All risk neonates with severe asphyxia, within 24h of injury were included. Serum S100 was measured on postnatal days 1–3–7 in 62 term infants with birth asphyxia. S100B levels were measured using ECLIA method.

Results The average serum S100B levels for the control group (N=48) was 0.12 microg/L (cut-off point). S100B levels were significantly higher in asphyxiated term neonates N=29; M=0.64. Infants with moderate and severe HIE had significantly higher S100 levels on postnatal day 1 ($p = 0.031$) and day 2 ($p = 0.008$) than infants with mild or no HIE. The levels of S100 decreased on days 2 and 3 in all infants with HIE. The median S100 level on postnatal day 1 was higher in nine infants who died neonatally and in 10 infants who developed cerebral palsy (CP), compared with 43 infants with no signs of impairment at follow up, 14.0 µg/L, 20.7 µg/L and 5.5 µg/L, respectively. A level of S100 above 12 µg/L the first day of life was significantly more frequent in infants who died or developed CP than in infants with no impairment at follow up ($p = 0.02$).

Conclusion Early determination of serum S100 may reflect the extent of brain damage in infants with HIE after asphyxia.

1088 VENTILATION MODALITY AND CEREBRAL DAMAGE IN PRETERM INFANTS

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Background and Aims Several studies on high frequency oscillatory ventilation (HFOV) have demonstrated a high relation between this method and the incidence of hemorrhagic or ischemic brain injuries. In this study, we meant to verify the incidence of intraparenchymal hemorrhage (III–IV stadium) and cystic periventricular leukomalacia (CPVL) in subjects submitted to different ventilation (HFOV vs CMV).

Materials and Methods We have examined 120 newborns (mean gestational age 30±1.85 weeks, mean birth weight 1314±404.14 grams). Group A: 60 infants ventilated in HFOV; Group B: 60 infants ventilated in CMV. All received surfactant.

All infants underwent monitoring of cardiovascular function with evaluation of parameters such as fractional shortening, ejection fraction, size of rooms, the ductal shunt, pulmonary artery pressure and cardiac output and blood pressure.

Results Infants ventilated in HFOV have required a maximum value of MAP significantly lower ($p < 0.05$) versus those ventilated in CMV; In group A, 8 (13.3%) experienced a hemorrhage of III–IV degree against 12 (20%) of subjects in group B.

They presented CPVL 14 (7.23%) infants in group A compared with 16 (26.6%) in group B. There were no differences in cerebral blood flow and resistance index of the anterior cerebral artery. There were no differences in cardiac function.

Conclusions Our data show an increase, not statistically significant, of PIVH and CPVL in newborn infants treated with conventional ventilation than oscillatory ventilation. This is attributable to the use of a MAP “optimal” able to obtain a good alveolar recruitment without causing hyperexpansion.

1089 VISUAL EVOKED POTENTIALS IN TERM AND PRETERM INFANTS

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Background and Aims Evoked potentials are a useful non-invasive method for the assessment of neurological status in term and preterm infants at risk for perinatal CNS damage. The present study intended to gather reference values of visual evoked potentials (VEP) for preterm and term neonates and identify neonates at risk for perinatal CNS lesions using VEPs.

Methods 23 healthy preterm and term neonates (group A) and 16 infants with perinatal brain injury (group B) were examined for this study. Groups were classified according to their post-conceptual age (A1/B1: 42–45 weeks, n=4/6; A2/B2: 38–41 weeks, n=5/8; A3: 36–37 weeks, n=12; A4/B3: < 36 weeks, n=2/2). Stimulation was triggered by stroboscopic flashes (1 Hz/10 µs) and P1 and N2 waves were analyzed.

Results Latencies significantly correlated with post-conceptual age (P1: $p < 0.001$, N2: $p < 0.05$) and gestational age (P1: $p < 0.01$). The average latency values (mean±SD) of the subgroups were: group A1 (P1: 165.7ms±33.5; N2: 211.5ms±29.9), A2 (P1: 199.6ms±34.2; N2: 255.6ms±25.8), A3 (P1: 223.8ms±14.7; N2: 272.1ms±13.5), A4 (P1: 240 resp. 209ms; N2: 242 resp. 233ms). Average latencies of term infants with or without perinatal injury differed significantly for P1 (228.8 ms±30.9 vs. 165.7ms±33.5; $p < 0.05$) and N2 (266.0 ms±21.1 vs. 211.5ms±29.9; $p < 0.01$).

Conclusions The present study adds to the knowledge on normal VEP values during early development. Present data showed a negative correlation with post-conceptual age for central latencies as an equivalent of progressing myelination regardless of extra- or intra-uterine maturation. Term infants with perinatal brain injury showed significantly prolonged VEP latencies compared to healthy children.

1090 RELATIONSHIP BETWEEN NEUROTROPHINS AND BRAIN STRUCTURE IN PRETERM GROWTH RESTRICTED BABIES

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Background Neurotrophins are responsible for the growth and survival of neurons during early brain development. Intrauterine growth restriction (IUGR) leads to alterations in brain structure.

Aim To explore the relationship between neurotrophins and brain structure in preterm IUGR babies.