

compared to adults control and APC did not reduce this effect. Neutrophil TLR4 expression was significantly increased in response to LPS in NE infants on D3 compared to adults ($p<0.001$) and has been reduced by APC ($p=0.03$). LPS induced monocyte TLR4 was only significantly increased in NE infants D7 ($p<0.001$). Neutrophil ROI was significantly increased in Adults ($p<0.001$) and NE infants on D3 ($p=0.021$) following LPS and this response were significantly reduced by APC.

Conclusion Neutrophil activation and production of ROI may mediate tissue damage in NE infants. APC modified LPS responses in adults and NE infants on D3 of life. APC may reduce the inflammatory responses secondary to hypoxia and possibly benefit these patients at high risk of inflammatory multiorgan dysfunction.

1098 CARDIAC OUTPUT MEASUREMENTS IN PRETERM NEONATES REQUIRING RESUSCITATION AT BIRTH

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Background The effect of perinatal asphyxia on cardiac output and flow patterns in asphyxiated preterm neonates is less well understood.

Objectives To study the cardiac outputs (left and right ventricle – LVO and RVO) and superior vena cava (SVC) blood flow patterns in asphyxiated preterm neonates in first 24 hours of age.

Subject and Interventions Serial echocardiography was done in preterm neonates < 34 weeks who required resuscitation, at 6 ± 2 , 12 ± 2 and 24 ± 4 hours using color Doppler (Sonosite). LVO, RVO and SVC flow velocity were calculated.

Results Functional Echo was done in 68 neonates with mean gestation and weight of 31 ± 1.6 weeks and 1343 ± 361 g. Median SVC flow, LVO and RVO at 6, 12 and 24 hrs of age were 109 (70–137), 103 (85–150) and 132 (92–181); 381 (287–493), 421 (337–510) and 408 (324–557); 327 (214–435), 328 (259–467) and 381 (280–501) ml/kg/min respectively. The differences in these three measures between three time points were not statistically significant. A statistically significant increase was seen between SVC flows at 6 versus 24 hours. No difference was observed in these measurements in 21% vs 100% oxygen groups.

Conclusions LVO, RVO and SVC flow showed an increasing trend from 6 hrs of age to 24 hrs of age. A significant increase was observed in the SVC flow between 6 and 24 hours of age suggestive of hypoperfusion-reperfusion phenomena. Resuscitating with 21% or 100% oxygen did not show any difference in these measurements.

1099 NORMATIVE LEVELS OF INTERLEUKIN 16 IN UMBILICAL CORD BLOOD

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Background and Aims The need for early and accurate prediction of outcome in Hypoxic-Ischemic Encephalopathy (HIE) remains critical. We have previously demonstrated that Interleukin 16 (IL-16) is raised in the umbilical cord blood of infants with moderate and severe HIE and has the potential to be developed as a predictive biomarker. Normal reference ranges for IL-16 in umbilical cord blood have not been previously described. The aim of this study was to determine normative levels of IL-16 in full term neonates using cord blood following uncomplicated deliveries.

Methods Full term infants were recruited as part of an ongoing birth cohort study, the Cork BASELINE Birth Cohort Study. All had cord blood drawn and bio-banked at -80°C , within 3 hours of birth. Samples were chosen based on Apgar scores (≥ 8 at 1min, ≥ 9 at 5min), duration of ruptured membranes < 24 h, temperature in

labour $\leq 37^{\circ}\text{C}$, gestational age ≥ 37 weeks and birthweight centile $\geq 10\%$. Analysis was performed on plasma EDTA, using ELISA Quantikine® (R&D Systems, Europe).

Results The study consisted of samples from 48 infants with two different modes of delivery; unassisted vaginal delivery ($n=12$ male, $n=12$ female) and pre-labour elective caesarean section ($n=12$ male, $n=12$ female). The range of all samples was normally distributed between 87.0 and 114.6 pg/ml. Mean (SD) for IL-16 was $103.1 (\pm 21.9)$ pg/ml. Levels were not affected by gender or mode of delivery.

Conclusion For the first time we have described the expected range of cord plasma IL-16 levels in healthy term infants.

1100 C-REACTIVE PROTEIN CONCENTRATIONS IN NEONATES WITH HYPOXIC-ISCHAEMIC ENCEPHALOPATHY AND EFFECT OF TOTAL BODY HYPOTHERMIA

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Background and Aims Production of C-reactive protein (CRP), an acute phase reactant of hepatic origin, may be affected by perinatal asphyxia. This study tested hypotheses that circulatory CRP concentrations correlate with clinical severity of hypoxic-ischaemic encephalopathy (HIE) and that total body hypothermia modulates CRP response.

Methods Clinical records in three centres were reviewed for neonates ≥ 36 weeks' gestation admitted between 01/07/06 and 30/06/11 with HIE of any severity (grades 1–3 Sarnat-Sarnat). Participating centres adopted routine cooling at different dates. Data extracted included CRP concentrations in the first postnatal week measured during routine clinical practice, clinical HIE grading, and reception of therapeutic hypothermia. Proportions with raised CRP (>10 mg/L), and maximum CRP concentrations were assessed according to HIE grade and whether cooled.

Results A raised CRP was present in 150/259 (58%) neonates during the first postnatal week (HIE1: 30/73[41%], HIE2: 83/129[64%], HIE3: 37/57[65%], $p=0.003$) but elevated maximum concentrations (peaking median day 3) did not differ between HIE grades (median [range] HIE1: 31.3 [10.0–188.1] mg/L, HIE2: 32.5 [10.0–305.9] mg/L, HIE3: 34.0 [10.2–346.5] mg/L, $p=0.48$). A raised CRP was present in 117/187 (63%) cooled and 33/72 (46%) non-cooled infants ($p=0.02$), but their peak CRP concentrations did not differ (median [range] CRP cooled vs. non-cooled: 31.9 [10.0–346.5] mg/L vs. 53.0 [10.4–188.1] mg/L, $p=0.26$).

Conclusion A raised CRP is a common finding in the first postnatal week in neonates admitted with HIE and is found in most infants with moderate-severe HIE. Peak CRP concentrations did not differ with clinical HIE grade and whole body hypothermia did not significantly affect peak CRP concentrations.

1101 ARE LACTAT DEHYDROGENASE AND NEURON SPECIFIC ENOLASE ANALYSES GOOD DIAGNOSTIC TOOLS FOR ASSESSING EXTENSION OF PERINATAL HYPOXIC-ISCHAEMIC BRAIN INJURY?

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