plane. Transverse cerebellar diameter (TCD), 4th ventricle width (4VW) and length (4VL) were assessed via mastoid fontanelle.

Results Sector probe gave significantly smaller measurement results than convex transducer. Left side measurements were significantly greater than right side ones. VI, VW, BW, TCD and 4VW were dependent on birth weight, birth length, head circumference and gestational age. ACSP, FHW, TOD and JR were independent of those factors. No relationships were found between gender and measured diameters or calculated ratios.

Conclusions Reference values for cranial measurements, ER and JR were determined for examined population of premature newborns. Described indices may be useful for everyday clinical practice in neonatal units.

1066 ABNORMAL HEART RATE CHARACTERISTICS ARE ASSOCIATED WITH ABNORMAL BRAIN ULTRASOUND AND MRI IN EXTREMELY LOW BIRTH WEIGHT INFANTS

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Background and Aim Brain injury may lead to autonomic nervous system dysfunction reflected by abnormal heart rate characteristics (HRC). A HRC monitor was developed to analyze heart rate variability and decelerations and predict catastrophic illness in NICU patients (HeRO monitor). We tested the hypothesis that the HRC index would be abnormally high in preterm infants with abnormal brain ultrasound and MRI.

Methods We collected HRC data on extremely low birthweight infants who underwent brain MRI as part of a study of neurodevelopmental outcomes. Brain ultrasounds (US) were performed in the first week and as clinically indicated, and brain MRI was performed near discharge. US were scored as normal/mild (including grades 1–2 IVH) and moderate/severe (grades 3–4 IVH or cystic periventricular leukomalacia). MRI was scored 0–3 for gray and white matter injury.

Results 45 ELBW infants with MRI performed had HRC data available. MRI was classified as 0 (normal, n=22), 1 (mild, n=14), 2 (moderate, n=5) or 3 (severely abnormal, n=4) and US as normal/mild (n=36) or moderate/severe (n=9). Average HRC index in the first 28 days after birth (aHRC28) was highly correlated with severity of abnormal brain US and MRI. aHRC28 for patients with normal/mild US abnormalities was 1.37 ± 0.48 (mean $\pm$ SD) and with moderate/severe 2.53 ± 0.69 ($p < 0.001$). For MRI, aHRC28 for patients with classes 0–3 was 1.24 ± 0.44 , 1.59 ± 0.53 , $2.49 \pm 0.68^*$, $2.72 \pm 0.82^*$, ($*p < 0.001$ versus class 0). Differences persisted after adjustment for gestational age and birthweight.

Conclusion HRC monitoring may be a useful adjunct test for severity of brain injury in NICU patients.

1067 INITIAL EXPERIENCE OF EFFECT OF SELECTIVE HEAD COOLING IN NORMOTHERMIC AND HYPOTHERMIC INFANTS

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The Aim of this Study To evaluate the effect of selective head cooling (introduced 3 months ago in the Neonatal Intensive Care Unit at the University Children's Hospital in Skopje-Macedonia) on S100 B protein levels, previously measured only in normothermic infants after perinatal asphyxia and the preliminary neurodevelopment outcome at the age of 3 months.

Methods All risk neonates with severe asphyxia admitted within 24h of injury were eligible for inclusion in the study. One serum

blood sample was obtained from each patient the first day of admission, and 48h and 72h hours after admission. S100B levels were measured using ECLIA method (Electro-Chemil-Luminescence Immuno Assay-Elecsys 2010-Roche Diagnostic).

Results The average serum S100B levels for the control group (N=48) was 0.12 microg/L (–1) (cut-off point). Serum S100B levels were grossly elevated in both HT and NT groups of infants with asphyxia. The differences were statistically significant as follows: a) between the first (24h) and second (day 4) time interval significant at $p < 0.05$; b) between the second (day 4) and third (day 7) time interval significant at $p < 0.005$; c) between the first (24h) and third (day 7) time interval significant at $p \leq 0.001$. Serum S100B values were lower in HT (selective head cooling infants) compared to NT infants ($p = 0.049$ at 48 hours).

Conclusion Serum S100B levels were lower in the HT group after 72h, and strongly correlated with the neurodevelopment impairment. S100B levels are highly elevated following asphyxia. Serum S100B levels are lowering in the HT and strongly correlate with the early neurodevelopment outcome.

1068 CAN LOSS OF SLEEP-AWAKE CYCLICITY AT AEEG PREDICT POSTHEMORRHAGIC HYDROCEPHALUS IN PRETERM INFANTS WITH INTRAVENTRICULAR HEMORRHAGE?

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Background and Aim Intraventricular hemorrhage (IVH) is the most common brain injury in preterm infants. Among infants with IVH 30% develops posthemorrhagic hydrocephalus (PPH). Actually there is no predicting factor for the developing of PPH. We observed if loss of sleep-awake cyclicality, at aEEG evaluation, is predictive for PPH.

Methods 6 preterms of gestational age between 25 and 30 weeks were detected for IVH (II degree to IPH) at cerebral ultrasonography. Cerebral background activity was continuously performed by CFM (Brainz US).

Results Sleep-awake cyclicality was observed in all 6 infants in the first 24 hours after the detection of IVH. But in 2 of these we noted the loss of cyclicality few days after the diagnosis of the bleeding. These 2 patients developed posthemorrhagic hydrocephalus whereas the other 4 infants didn't develop PPH.

Conclusions Loss of sleep-awake cyclicality, at aEEG, has a high positive predictive value for the developing of PPH in preterm infants with IVH; therefore study of cerebral background activity and in particular of sleep-awake cyclicality can be used as a early prognostic tools in patients at risk of PPH.

1069 DOES CEREBRAL OXYGENATION AID IN OUTCOME PREDICTION IN ASPHYXIATED NEWBORNS SUBMITTED TO HYPOTHERMIA?

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Background and Aims Neurological outcome in asphyxiated newborns submitted to hypothermia is being predicted by aEEG and MRI. The aim of this study was to assess the value of regional cerebral oxygen saturation (rSO₂) in early outcome prediction and its relation with aEEG and brain MRI.

Methods rSO₂ was measured by NIRS INVOS monitor. Newborns were simultaneously monitored with aEEG during hypothermia and rewarming period. Values of rSO₂ were analyzed in three groups of predicted outcome (normal, intermediate or abnormal) according

to both aEEG pattern at 48h and MRI performed during the second week. Comparisons were done with ANOVA, SPSS19.

Results 21 newborns were monitored. During hypothermia there was a trend towards higher rSO₂ in newborns with predicted abnormal outcome. During rewarming there was a significant difference between the normal and abnormal outcome groups ($p<0.02$).

Abstract 1069 Table 1 rSO₂ values during hypothermia and rewarming

	rSO ₂	Normal (n=9)	Intermediate (n=4)	Abnormal (n=8)
Hypothermia	12 hours	76,3	74,5	79,4
	24 hours	83,1	77,0	88,1
	48 hours	84,6	88,0	89,5
	72 hours	82,1	79,0	87,4
Rewarming	34.5°C	77,3	84,0	89,8
	35.5°C	75,8	73,5	88,4
	35.5°C	74,6	77,8	87,4

Conclusions NIRS monitoring may improve the early recognition of newborns with abnormal outcome. Our data suggests that a rSO₂ value of 85 during rewarming can be used as a cutoff for predicting abnormal outcome.

1070 CEREBRAL BLOOD FLOW CHANGES IN PRETERM NEONATES REQUIRING RESUSCITATION AT BIRTH

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Background Hypoxia in the perinatal period causes increase in cerebral blood flow followed by a decrease. CBF data is not available for preterms.

Objectives To study CBF changes in Anterior and Middle cerebral arteries (ACA & MCA) of asphyxiated preterm neonates (< 34 weeks).

Setting Level III Neonatal unit in Northern India, PGIMER, Chandigarh.

Subject and Interventions This is a subgroup analysis of a RCT comparing 21% vs 100 % oxygen for resuscitation. CBF was measured in ACA and MCA at 6±2, 12±2 and 24±4 hours. Peak systolic velocity (PSV) cm/s, End diastolic velocity (EDV) cm/s, Resistance index (RI), Pulsatility index (PI), Systolic diastolic ratio (S/D) and Velocity time integral (VTI) were measured using Philips ultrasonography machine.

Results CBF was measured in 57 neonates. Mean gestation and weight was 31±1.3 weeks and 1400±358 g. HIE was seen in 24 babies (42%). PSV and VTI showed an increasing trend whereas S/D showed a decreasing trend as age increased. PSV [29.2 (23.7–34.2), 28.3 (23.9–33.4) and 33.9 (29.6–37.8) cm/s, $p=0.02$], VTI (6, 6.5 and 8 cm, $p=0.002$) and S/D (4.1 (3.07–6.6), 4.03 (3.3–5.4) and 3.8 (3.1–4.7), $p=0.01$) in the MCA showed significant trend at 6, 12 and 24 hrs respectively. No such trend could be observed in the ACA. On comparing 21% and 100% oxygen groups no difference were observed.

Conclusion CBF indices (PSV, VTI & S/D) of MCA showed a significant increase from 6 to 24 hrs. No difference was observed on comparing 21% with 100% oxygen.

1071 AEEG DURING THE FIRST 72 HOURS AFTER BIRTH IN INFANTS WITH PRENATALLY DIAGNOSED CONGENITAL HEART DEFECT

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Background and Objective Survival in infants with congenital heart defects (CHD) is associated with neurologic morbidity. Hypoxic-ischemia after closure of the ductus arteriosus is probably one of the factors resulting in brain damage. Prenatal diagnosis makes it possible to prevent closure of the ductus arteriosus and therefore cerebral perfusion can be preserved. Amplitude integrated EEG (aEEG) is a method to evaluate brain injury. Abnormal (a)EEG patterns and epileptic activity (EA) are frequently observed in infants with CHD. Our aim was to determine the course of aEEG patterns in infants with prenatally diagnosed CHD.

Methods Retrospective cohort study of infants prenatally diagnosed with CHD. aEEGs were assessed by pattern recognition: background pattern (BP), presence of sleep wake cycling (SWC) and EA were appraised.

Results Twenty infants (mean GA 39 wks; birth weight 3416 g) were included. Eleven infants had transposition of the great arteries, 7 infants had hypoplastic left heart syndrome, and 2 had aortic valve stenosis. At 6h after birth 80% of infants had normal BPs (continuous normal voltage (CNV)). Only 2 infants had severely abnormal BPs (continuous low voltage). At 24h, 94% had CNV. None of the infants showed EA. SWC was present in 85% and emerged at a median postnatal age of 10.4 hrs.

Conclusions aEEGs of infants with a prenatally diagnosed CHD are normal in the majority of infants, with normal emergence of SWC and absence of EA. This indicates that prenatal diagnosis can prevent brain damage in infants with CHD.

1072 REVIEW OF CURRENT OPINION AND AVAILABILITY OF CEREBRAL FUNCTION MONITORING AND THERAPEUTIC HYPOTHERMIA IN UK NEONATAL UNITS

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Background and Aims Therapeutic hypothermia has been established as an effective treatment modality for term neonates that have suffered an acute perinatal hypoxic ischaemic event. This is a time-critical procedure which increases pressure to commence active cooling as soon as possible after the injury.

This study aims to define the current levels of ownership of CFM and cooling equipment and capture some staff perceptions around cooling matters in neonatal units in the UK.

Methods Telephone survey conducted over March–November 2011 with the 205 active neonatal units listed on the British Association of Perinatal Medicine website.

Results 100% response-rate (205/205). 141 (68.8%) responders were Lead Nurse/Sisters, 59 (28.8%) were Nurses and 5 (2.4%) were Doctors. Overall, CFM was available at 106/205 (51.7%) units and 89/205 (43.4%) had cooling equipment. Equipment ownership was high in NICUs and progressively decreased in LNUs and SCBUs. The majority of responders were positive to the idea of therapeutic hypothermia as a standard of care whilst some asked for further research; only a small minority were negative. 91.6% were keen to consider a scheme where therapeutic hypothermia was initiated at the referring centre and continued in a dedicated cooling centre.

Conclusions Therapeutic hypothermia is widely recognised as effective. Availability of cooling equipment and CFM has increased in all levels of units in the UK. The time-constraints in initiating therapeutic hypothermia might mean that a scheme of initiating therapeutic hypothermia locally, continuing this during transport and completing it in a designated cooling centre is the way forward.

1073 EARLY EEG IN INFANTS WITH HYPOXIC ISCAEMIC ENCEPHALOPATHY: POSSIBLE INFLUENCES OF THERAPEUTIC HYPOTHERMIA

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