Abstract PO-0473 Table 1

	Light NREM	NREM	REM	Sleep stage shifts (n/hour)	Awakenings (n/hour)	Average REM period (min)
Before caffeine	21%	36%	43%	27	13	9
After caffeine	22%	36%	42%	29	12	10

PO-0473 CAFFEINE TREATMENT HAS NO SIGNIFICANT EFFECT ON SLEEP QUALITY IN PRETERM INFANTS

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Background and aims Sleep stages begin to emerge from 20 weeks' gestational age. Typical EEG changes appear at 28–30 weeks with periods of rapid eye movement (REM) and non-REM (NREM) sleep. Sleep patterns develop into near adult like patterns by 3–5 months of age. Sleep cycles (REM/NREM) are essential for infant brain development. Caffeine is routinely used for treatment and prevention of apnea of prematurity. Its effect on sleep organisation in preterm infants has been controversial. This study aimed to find differences in sleep organisation in preterm infants before and after initiation of caffeine treatment.

Methods Polysomnography was recorded in 10 preterm infants [GA 27+2–36+6 (mean 30+1) weeks, BW 790–2875 (mean 1465) g] at 34–41 (avg. 36) weeks' GA before and the day after administration of caffeine citrate loading dose (20 mg/kg). The analysis was done with visual scoring.

Results Sleep quality was variable. Some infants had very interrupted sleep structure. Uninterrupted phasic REM sleep periods seemed to be more readily identified after caffeine. However, we were unable to show this by using standard indexes. There was no difference in sleep stage distribution, number of awakenings, number of sleep stage transitions or average REM period (Table 1).

Conclusions We did not find any evident effect of caffeine on sleep quality in preterm infants.

PO-0474 IMPACT OF GLUTAMATE TRANSPORTER HAPLOTYPES AND CLINICAL COURSE ON FUNCTIONAL BRAIN NETWORKS IN PRETERM INFANTS

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Reducing adverse neurodevelopmental outcomes associated with preterm birth is a major challenge facing neonatal medicine, as abnormalities engendered during the perinatal period have lifelong implications. The pathological mechanisms leading to abnormal neurodevelopment in preterm infants involve several pathways. Many direct and indirect effects of preterm birth on neural development occur at the micro structural or neurochemical level, meaning that current methods for assessing neurological injury in preterm infants provide only limited mechanistic and prognostic information. A promising alternative approach is the use of resting state functional MRI (rs-fMRI) to infer integration of neural activity across brain regions (functional connectivity (FC)). Adults who were born preterm show persistent differences in FC, and early detection of such changes offers potential insights into the pathophysiology of preterm brain injury. These functional changes may be influenced by both neonatal course and underlying susceptibilities to abnormal development, including genetic factors.

We utilised rapid multiband sequence rs-fMRI acquisition at 3 Tesla, to characterise functional brain connectivity in 30 infants born at <32 weeks gestation, scanned at term. DNA was extracted and sequenced for EAAT2 glutamate transporter haplotypes associated with adverse preterm neurodevelopmental outcomes. Using a multivariate model we identified dissociable and interacting influences of demographic, genetic and clinical variables on functional infant brain networks. We are the first to describe the influence of genetic variability in cerebral glutamate homeostasis on neonatal brain connectivity. We discuss the impact on understanding preterm brain injury, and the potential for predicting neurodevelopmental outcome by non-invasive measurement of functional brain connectivity.

PO-0475 THE EFFECT OF CO2-INSUFFLATION ON CEREBRAL OXYGENATION IN THORACOSCOPIC REPAIR OF ESOPHAGEAL ATRESIA IN NEONATES

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Aim of the study Infants undergoing neonatal surgery for thoracoscopic esophageal atresia repair are at high risk of adverse neurodevelopmental outcomes. Increasing concerns have been raised about the incidence of perioperative brain injury, this is suggested to be due to haemodynamic instability and hypoxia perioperatively. We evaluated the effects of CO2-insufflation on regional cerebral oxygen saturation ($rScO_2$) during thoracoscopic esophageal atresia repair.

Methods Observational study of 20 neonates undergoing thoracoscopic esophageal atresia repair. During surgery mean blood pressure (MABP), FiO_2, arterial saturation and the cerebral oxygen saturation ($rScO_2$) were continuously monitored.

Four periods of 10 min were selected: (T=0) during anaesthesia, (T=1 and T=2) during CO_2-insufflation and (T=3) after desufflation.

Main results Complete registration was obtained in 14 neonates (median GA 36.9 [30.6–41.9], birth weight 2358 g [1395–4490]) and were included.

After CO_2-insufflation the FiO_2 increased from 41% to 58%, whilst the saturation decreased from 96.3% to 92.5%($p < 0.05$).

The arterial pCO_2 (mmHg) changed from 47 ± 6.9 to 56 ± 13($p < 0.05$) after CO_2-insufflation and remained stable at the end of insufflation and after desufflation.

The $rScO_2$ did not change after CO_2-insufflation or desufflation.

Conclusion Intrathoracic CO_2-insufflation causes a decrease in arterial saturation and an increase in arterial pCO_2. However, more importantly these changes did not result in significant fluctuations in cerebral oxygenation throughout the procedure. The insufflation of CO_2 with 5 mmHg during thoracoscopy seems to be safe in neonates, since the cerebral oxygenation was preserved during the procedure.