Background Prothrombotic risk factors (PRF) are suggested to be involved in the pathogenesis of PAIS. However, most of published studies are retrospective, they vary in the PRF tested and parents are not usually investigated.

Objective To determine the impact of parental and infant thrombophilia on neonatal cases diagnosed of PAIS in a prospective case-control study.

Methods Factor V (G1691A mutation), prothrombin G20210A variant, MTHFR C677T genotype, antithrombin, protein C, protein S, lipoprotein (a), homocystein (Hcy) and anticardiolipin antibodies were investigated in 45 infant-parent pairs with PAIS and in 85 controls. Blood samples were drawn within the first week of life.

Results All thrombophilic factors investigated were similar or even less frequent among patients with PAIS and their parents compared to controls. The most frequent PFRs were Hcy > 11 and MTHFR homozygosity in cases (7.5% and 5.3%, respectively) and in controls (24.4% and 13.1%, respectively). Thirteen neonates diagnosed of PAIS (28.9%) had at least 1 PFR, compared to 39 subjects (44.3%) in the control group (OR/95% CI, 0.51/0.23 to 1.10) ($p < 0.001$). Twenty three mothers of infants with PAIS (51.1%) were positive for thrombophilia markers, compared to 49 (55.7%) controls ($p = .617$). In 8 mother-infant pairs (17.8%), at least 1 PFR could be identified for either mother or infant, compared to 19 controls (21.6%). Fifteen neonates with stroke (33.3%) had at least one PFR compared to the 57 subjects (64.8%) in the control group ($p < 0.001$).

Conclusion Our data do not support that PFR play a major role in PAIS.

PO-0407 PERINATAL FACTORS AND PERINATAL ARTERIAL ISCHAEMIC STROKE (PAIS): A PROSPECTIVE CASE-CONTROL STUDY

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Background Perinatal factors (PF) have been implicated in the pathogenesis of PAIS. Hypoxia has been suggested to be involved as a potential cause of AIS although prospective case-control studies are still warranted.

Objective To determine the impact of PF on neonatal cases diagnosed of PAIS in a prospective case-control study.

Methods 45 neonates were diagnosed of PAIS within four weeks of life and 85 controls were investigated for the following PF: retarded intrauterine growth restriction, sentinel event, epidural and general anaesthesia, presentation at delivery, type of delivery (vaginal, instrumental, emergency caesarean section), abnormal cardiotocographic monitoring, Apgar score at 1 and 5 min, arterial umbilical cord pH <7.0 and <7.20, and need of advanced resuscitation.

Results The univariate analysis indicated the following associations with PAIS: emergency caesarean section (48.9% vs 14.8%; $p < 0.001$); Apgar score at 1 min < 7 (22.2% vs 4.6%; $p = 0.005$) and < 5 (15.6% vs 1.1%; $p = 0.002$); arterial cord pH, mean $\pm$ SD 7.19 ± 0.12 (CI95% 7.15,7.23) in infants with PAIS compared to 7.25 ± 0.1 (7.23,7.27) in controls ($p = .005$); arterial cord pH <7.20 (45.2% vs 17.8%; $p = 0.006$).

A multivariate analysis did not show any independent factor associated to PAIS, except for arterial cord pH < 7.20 (OR 2.89,

CI95% 1.01, 8.31). Emergency caesarean section and Apgar score <5 at 1min, showed a tendency to be associated with PAIS but without statistical significance: OR 2.61 (95% CI 0.66, 10.2) and 10.02 (CI95% 0.46, 215.8).

Conclusion Our data indicate that PF do not appear to play a major role in PAIS.

PO-0408 CAUSES AND OUTCOME OF NEONATAL CONVULSIONS

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Background and aims Convulsions occur more frequently in the neonatal period than at any other time.

Aims Study the clinical, etiological, therapeutic aspects and the outcome of neonatal convulsions.

Methods We report a retrospective analysis of 51 cases of neonatal convulsions between 2009 and 2013.

Results The sex-ratio was 1.1. Prematurity was found in 8 patients. The Apgar score was lower or equal to 7 in the first minute in 16 cases. The average age of the first convulsion was 9 days. The motive of admission was convulsion in 35 cases (66%). It was a convulsive status epilepticus in 4 cases. The neurological examination was pathological in 27 cases. A metabolic balance sheet, lumbar puncture were realised in every case, cranial ultrasound scan (34 cases), cerebral CT scan (19 cases), electroencephalography (29 cases) and MRI (16 cases). Causes of neonatal convulsions were: hypoxic-ischaemic encephalopathy (13 cases), hypoglycemia (7 cases), hyponatraemia (6 cases), hypocalcemia (4 cases), pyridoxine dependency (1 case), meningitis (5 cases), hypertension (1 case), ischaemic stroke (1 case), cerebral haemorrhage (3 cases), anomaly of the urea cycle (1 case), West syndrome (2 cases) and idiopathic neonatal convulsions (7 cases). The treatment was etiological in 23 cases. The outcome was favourable in 37 cases, 4 patients died and variable sequelae were found in 10 cases.

Conclusion This study emphasises the variability of aetiology of neonatal convulsions and the necessity of the etiological diagnosis on which will depend the prognosis.

PO-0409 WITHDRAWN

PO-0410 PROPYLENE GLYCOL (PG) DETECTION BY BRAIN MAGNETIC SPECTROSCOPY (MRS) IN INFANTS WITH NEONATAL ENCEPHALOPATHY

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Background PG is a commonly used excipient contained in several medications to control neonatal seizures (Phenobarbitone (PhB) (79%) Phenytoin (Ph) (40%) and Clonazepam). On MRS the appearance of a doublet at 1.1 ppm in some spectra is attributed to PG. The lower safe level of short term exposure to PG

in neonates is 35 mg/kg/24 hrs however safe brain levels are unknown.

Objective To determine the PG levels in MRS of infants with NE.

Design/methods MRS between July 2010 and August 2013 were reviewed in infants with NE (PRESS sequence TE: 288ms) All MRS spectra were reviewed by an MR Physicist. Cases with an observable doublet at 1.1 ppm were reprocessed with Tarquin V4.3.2 using a simulated basis set the included PG and referenced to unsurpassed water signal to obtain institutional units of concentration.

Results 29 infants with NE. MRI was performed at mean age of 120 hrs (35–197 hrs) Diagnosis HIE (27), Congenital lactic acidosis (1) GBS meningitis (1) MCA infarction (1) PG was present in 24% of infants (n = 7). The mean level on MRS was 10.76 mM (2.11–26.48).

All infants with PG peaks received 40mg/kg PhB, and 18 mg/kg Ph and 6/7 received Clonazepam. No PG group required less anticonvulsants (13.6% no treatment, 63% PhB, 23% PhB and Ph).

Conclusions PG is detected on MRS in NE infants.

The level may correlate with underlying diagnosis. PG accumulation may have clinical implications which need to be further investigated. Additionally PG must be correctly differentiated from lactate on MRS.

PO-0411 QUANTITATIVE AMPLITUDE INTEGRATED ELECTROENCEPHALOGRAPHY ANALYSIS IN VERY LOW BIRTH WEIGHT INFANTS ON FIRST DAYS OF LIFE

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Background and aims Amplitude integrated electroencephalography (aEEG) is a tool for continuous brain function monitoring in NICU patients. The aEEG classification related to pathology and visually assessed by examiners is fully described. Nevertheless the quantitative analysis of these signal is still not well defined.

The aim was to check if a quantitative analysis of an aEEG can be a useful tool in early diagnosis of morbidities such as intraventricular haemorrhage (IVH), periventricular leukomalacia (PVL) and hemodynamically significant persistent ductus arteriosus (PDA).

Methods Very low birthweight newborns admitted to NICU in a first day of life were included. On 1st, 3rd and 10th day of life patients had been monitored with aEEG for 2 h. A 1 h of stable recording was analysed. The aEEG power was analysed in 3 ranges (below 5uV, between 5uV and 40uV and above 40 uV). The study group consisted of sick newborns with either IVH (III and IV grade), PVL or PDA (with surgical closure of PDA). The control group were children without these abnormalities. Groups were cross-matched according to gestational age. U Mann-Whitney test had been used.

Results There were 12 newborns in each: study and control group. There were 15 samples of an aEEG power from each patient. The aEEG power for children in study group was significantly lower on 1st day of live for a range above 40uV (p < 0,000000). On a 3rd day the aEEG power was significantly

lower in a study group for range below 5uV (p < 0,000000) and higher for ranges between 5uV and 40uV and above 40uV (p < 0,000000). On a 10th day the power above 40uV was significantly lower in a study group (p = 0,000006).

Conclusion Quantitative analysis of an aEEG could be a useful method of identifying high risk neonates on a first and 10th day of live. Diminished power above 40uV reflects decreased CNS activity described as electrical bursts.

PO-0412 RELATIONSHIP BETWEEN CEREBRAL AND SYSTEMIC PERFUSION, AND SHORT-TERM OUTCOMES IN INFANTS WITH PERINATAL ASPHYXIA

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Background and aims The effects of haemodynamic changes on cerebral and systemic perfusion in infants with perinatal asphyxia are not well understood. We investigated the relationship between cerebral and systemic perfusion, and short-term outcome in infants with asphyxia.

Methods Ten infants (gestation age >35 weeks) with asphyxia (Apgar score <7 at 1 min) were divided into 2 groups: those with hypoxic-ischaemic encephalopathy (HIE; HIE group, n = 4) and those without HIE (non-HIE group, n = 6). Cerebral tissue oxygenation index (TOI) and cerebral fractional tissue oxygen extraction (FTOE) were measured by near-infrared spectroscopy (NIRS) at 12, 24, 48, and 72 h after birth. Superior vena cava (SVC) flow and left ventricular cardiac output (LVCO) were simultaneously measured by echocardiography.

Results TOI was significantly higher and FTOE was significantly lower in the HIE group (n = 4) than in the non-HIE group (n = 6) at all measurement time points. Although SVC flow and LVCO were not significantly different between the 2 groups, they were consistently higher in the HIE group than in the non-HIE group at all measurement time points. We found a positive correlation between SVC flow and LVCO in both groups, and between SVC flow and TOI in the non-HIE group.

Conclusions Combined bedside monitoring of TOI and FTOE by NIRS and SVC flow may be useful for evaluating secondary energy failure and disrupted regulation of brain circulation in infants with asphyxia.

PO-0413 TRIGEMINAL ODOURS RELEASED BY HEALTHCARE PRODUCTS ACTIVATE OLFACTORY AND PAIN CORTICAL AREAS IN PRETERM AND FULL TERM NEWBORNS

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Background/aim Hospitalised newborns are highly exposed to nosocomial odorous substances (OS) triggering possibly the intranasal trigeminal subsystem. Irritation of the nasal mucosa can induce pain and activations in pain processing areas in adults. We aimed to evaluate cortical activation in trigeminal/olfactory and pain areas following OS exposure in newborns.

Methods Forty-four newborns (17 full-terms, 12 preterms at term PMA and 15 preterms <33 weeks PMA when tested) were