September 20th, 2023 13:30 - 15:00

SHORT ORAL PRESENTATION SESSION 1 – BRAIN 1

ID 439. Utility of early cranial ultrasonography to time injury in hypoxic ischaemic encephalopathy

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Introduction

Given increased availability and improved quality of MR imaging, use of cranial ultrasonography (cUS) in the evaluation of hypoxic ischaemic encephalopathy (HIE) has fallen out of favor. cUS is primarily used in HIE to screen for alternative causes of encephalopathy and contraindications to therapeutic hypothermia. We hypothesise that cUS has a role in prognosis and timing of injury.

Aims

To assess whether cUS can help predict timing of injury. To assess the correlation between neuroimaging scoring systems (cUS and MR) which can be used in HIE to predict later neurodevelopment.

Methods

This was a retrospective study of infants with HIE admitted to a single tertiary NICU between 2010 and 2021. Validated scoring systems to predict later neurodevelopment in infants with HIE were used to evaluate neuroimaging performed as part of routine clinical care.[1][2]. Statistical analysis was performed using StataSE 17. Distribution

was assessed using Shapiro–Wilks test, significance was 0.05. Spearman rank was used to evaluate relationship between variables.

Results

One hundred and five infants with cUS suitable for scoring were included, of which 94 had MR imaging suitable for scoring. Seventy–four (70%) had moderate encephalopathy and 31 (30%) had severe encephalopathy. Fifty–seven (40%) infants had evidence for an acute sentinel event. Ninety–six (91%) infants underwent therapeutic hypothermia.

There was a strong correlation between cUS injury within 12 hours (n=28) of birth and first pH after birth. Infants with a higher pH were more likely to have white matter injury (r=0.39, p=0.039), suggesting a subacute/chronic insult as the likely mechanism for their encephalopathy. There was no correlation between grade of encephalopathy and cUS injury within 12 h of birth (p=0.45).

The strength of the relationship between cUS and MR total injury scores differed depending on timing of cUS (Table 1).

Conclusion

This study provides evidence for the utility of early cUS to time injury in HIE. White matter injury seen on early cUS, performed with 12 hours after birth, supports a sub–acute prenatal injury associated with HIE. cUS performed from 48 hours after birth correlates well with MR assessment of injury in HIE and may be useful adjunct to prognosis.

Table 1 Correlation between total injury scores on cUS and MR

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Authors have no conflict of interest to declare.

ID 215. A Machine Learning Method To Enhance The Recognition Of Brain Injury In Newborns

Mr. Jad D. Raad[1], Professor Ratna B. Chinnam[1], Dr. Suzan Arslanturk[1], Professor Jeong–Won Jeong[1], Dr. Swati Mody[2], **Professor Sidhartha Tan**[1]

[1]Wayne State University, Detroit, United States, [2]C. S. Mott Children's Hospital, Ann Arbor, United States

Background:

MRI is used to diagnose severity of brain lesions in neonatal encephalopathy. Machine learning to enhance the recognition of brain injury by neuroradiologists is now being actively investigated. Autoencoders are unsupervised learning methods based on neural networks, and essentially compresses the data and reconstructing the input without the redundancies. With the focus on finding new brain anomalies missed by the human eye, the objective of the study was to identify anomalies and improve prognostication of neonatal brain injury. We hypothesize that a framework using a particular autoencoder architecture (AE) would be able to discriminate between normal and abnormal in neonatal brain structure.

Methods

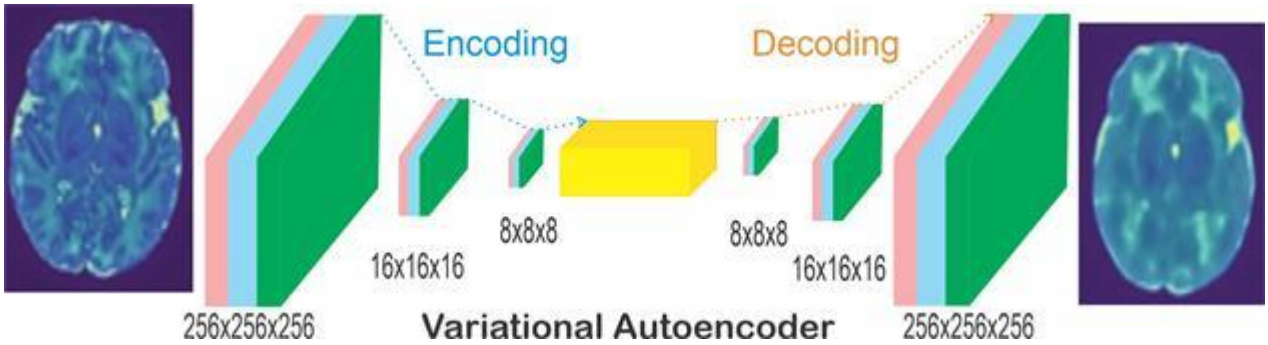
We tested the hypothesis on the Developing Human Connectome Project (dHCP) dataset with 97 patients, scored by their radiologists as 1 normal, 2, 3, 4 increasingly abnormal and 5 most abnormal. The model was trained with 80 normal, validated on 9 normal, and tested on 8 abnormal (score 5) scans. After preprocessing, we resized each image to 256x256x256 pixels. AE was used with a classification input, and compared to Variational AE (VAE), using a specific training method and analysis (mean square error, clustering, Z–scores, binning of standard deviations). Our neuroradiologist reading was the gold standard and ROC curves were determined.

Results

Reduction of 256x256x256 to 8x8x8 was superior to 16x16x16. VAE more effectively distinguished normal from abnormal and localized abnormalities better than AE, up to 83% of cases on ROC curve in 8x8x16. VAE also delineated anomalous regions in abnormal brains. Also, new brain anomalies were identified that were originally missed, and corroborated by our neuroradiologist.

Conclusions:

An unsupervised convolutional learning architecture such as our VAE may be a promising method to identify new brain areas of mostly term newborn brain injury not identified by the human eye. Our VAE can compress to 1/16,384–1/32,798 size and not lose the distinctive nature of the brain injury. The same approach of finding new anomalous regions and hitherto unrecognized abnormalities needs to be tested in a large trial using neurobehavioral outcome as a classification input and we speculate that VAE may be promising in prognostication.



Autoencoder Architecture

Autoencoder Architecture

None declared

ID 874. A direct comparison between intracranial volume measurements using fetal MRI and 3D ultrasound scans

Mrs Inge Van Ooijen[1,2], Mrs Sonja de Zwarte[3], Mrs Elise Turk[1], Jalmar Teeuw[3], Maaike Nijman[1], Daniel Cromb[7], Mireille Bekker[5], Alena Uus[6], Serena Counsell[7], Hilleke Hulfhoff Pol[3,4], Manon Benders[1]

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Background

Over the past decade, fetal brain volumes and growth derived from MRI have been studied in various conditions including critical congenital heart disease. Recent technical advancements have enabled calculation of intracranial volumes using 3D ultrasound. If comparable volumetric results can be achieved with 3D ultrasound when compared to MRI, this would be beneficial since ultrasound is a cheaper and more broadly available tool. To our knowledge, there have been no studies comparing the intracranial volumes derived from both techniques within 24 hours of each other. This paper aims to compare the intracranial volume measurements between 3D ultrasound and 3D MRI.

Methods

We performed both fetal 3D ultrasound and fetal MRI within 24 hours of each other in 15 healthy controls from the YOUth Baby and Child Cohort Study from the Utrecht University and the University Medical Center Utrecht. The 3D Ultrasound images were used as input for a 3D ultrasound–specific in–house developed convolutional neuronal network (CNN) to automatically segment and calculate intracranial volume. The MRI scans in three directions were combined to one 3D MRI slice–to–volume reconstruction (SVR). Each SVR was segmented using an automated tool based on the Human Connectome Project and a UNet CNN to enable measurement of intracranial volumes. We compared both methods by using a paired samples T–test and an intraclass correlation coefficient (ICC).

Results

Ten subjects (six male; four female) were included after MRI and ultrasound quality control. Average GA during MRI scans was 30.77 (SD=0.485) and during US scans was 30.78 (SD=0.461). The average intracranial volume, did not significantly differ between MRI and US measurements (mean intracranial volume 273.9 and 274.4 ml, respectively; p=0.93 and a single measures ICC of 0.766).

Conclusion

The fetal intracranial volume measured with 3D ultrasound is comparable to the intracranial volume measured with MRI. In the future, this could be beneficial since 3D ultrasound is a cheaper and more broadly available tool. Further research should incorporate more brain regions.

None declared.

ID 811. Fetal brain dimensions in congenital diaphragmatic hernia: relationship with fetal cardiac dimensions

Doctor Anna Claudia Massolo[1], Doctor Isabella Fabietti[1], Doctor Flaminia Pugnaloni[1], Doctor Milena Viggiano[1], Doctor Chiara Vassallo[1], Doctor Paola Giliberti[1], Doctor Irma Capolupo[1], Doctor Sara Savelli[2], Doctor Neil PATEL[3], Doctor Andrea Conforti[1], Doctor Leonardo Caforio[1], Professor Pietro Bagolan[1]

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Background:
Fetal cardiac hypoplasia is a recognised component of congenital diaphragmatic hernia (CDH). Recent investigations have separately demonstrated altered prenatal brain morphometry in CDH. This study now aims to examine the relationship between brain measurements and cardiac measures in fetuses with CDH.

Methods: Retrospective study of fetuses with isolated left CDH who underwent brain Magnetic Resonance Imaging (MRI) at a median gestational age (GA) of 32 (24–36) weeks. Insular fissure (IF), insular depth (ID), cingulate fissure (CF), and brain fronto–occipital diameter (BFOD) were collected. Fetal echocardiography was serially performed in all fetuses at 20–24 weeks, 25–28 weeks, and 33–38 weeks of gestation. Mitre–valve diameter (MVdm), Tricuspid–valve diameter (TVdm), left and right ventricular length (LVL, RVL) were measured and Z–scores were calculated. LV and RV end–systolic and end–diastolic area (LVESA, LVEDA, RVESA, RVEDSA) were analysed. Simple linear regression analysis was performed.

Results: 67 cases were considered for analysis, 10 fetuses met inclusion criteria.Significant correlations included: LV EDA and LV ESA measured at 33–38 weeks gestation correlated with IF and ID on both right and left sides of the brain and with DFO (all r2>0.4, P<0.05), Table 1. MV Z score correlated with Left IF at 33–38 weeks gestation (p0.04, r20.4). At 25–28 weeks LVL correlated with left CF (r20.5, P 0.02), right CF (r20.4, P 0.04) and right IF (r20.4, P 0.04).

Conclusions: Prenatal cardiac dimensions are significantly associated with reduced brain cortical measurements, particularly insular fissure, insular depth, cingular fissure and brain fronto–occipital diameter in left–sided CDH. These results support a mechanistic relationship between cardiac dysplasia and altered brain development in CDH fetuses.

Table1: Correlation between selected cardiac dimensions and brain measurements in left sided CDH fetuses.

	BRAIN MEASUREMENT						
CARDIAC DIMENSION	Left CF	Right CF	Left IF	Right IF	Left ID	Right ID	DFO
LV ESA mm^2							
20-24 weeks GA	p0.63, $r^2$0.02	p0.7, $r^2$0.02	p0.57, $r^2$0.04	p0.95, $r^2$0.01	P0.34, $r^2$0.1	P0.29, $r^2$0.1	p0.2, $r^2$0.1
25-28 weeks GA	p0.01, $r^2$0.6	p0.15, r^2 0.3	p0.97, $r^2$0.01	p0.48, $r^2$0.07	p0.04, $r^2$0.4	p0.08, $r^2$0.4	p0.05, $r^2$0.42
33-38 weeks GA	p0.52, $r^2$0.05	p0.6, $r^2$0.03	p0.14, $r^2$0.2	p0.002, $r^2$0.5	p0.04, $r^2$0.6	p0.001, $r^2$0.5	p0.004, $r^2$0.6
LV EDA mm^2							
20-24 weeks GA	p0.28, $r^2$0.1	p0.01, $r^2$0.5	p0.14, $r^2$0.2	p0.18, $r^2$0.2	p0.57, $r^2$0.04	p0.23, $r^2$0.2	p0.4, $r^2$0.08
25-28 weeks GA	p0.06, $r^2$0.4	p0.03, $r^2$0.51	p0.22, $r^2$0.2	p0.12, $r^2$0.2	p0.16, $r^2$0.2	p0.11, $r^2$0.3	p0.12, $r^2$0.3
33-38 weeks GA	p0.51, $r^2$0.05	p0.54, $r^2$0.04	p0.01, $r^2$0.5	p0.04, $r^2$0.4	p0.0003, $r^2$0.6	p0.0003, $r^2$0.7	p0.001, $r^2$0.8

None declared

ID 292. REDUCED INSULAR GREY MATTER VOLUME IS ASSOCIATED WITH LOWER FULL-SCALE IQ FOR CHILDREN BORN EXTREMELY PRETERM AT TEN YEARS

Miss Hedvig Kvanta[1], Dr Nelly Padilla, Prof Ulrika Ådén

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Introduction: Children born extremely preterm (EPT) have altered brain volumes already at term age, and the volumetric differences persist later in childhood. Children born EPT also have impaired neurodevelopment, with lower scores on cognitive assessments compared to term–born controls. The relation between volumetric alterations and cognition for children born EPT remain still needs further investigation. The aim of this study was to examine how grey matter volume for children born EPT relate to full–scale intelligence quotient (IQ).

Materials and methods: Out of children born EPT (<27 weeks) in Stockholm between 2004–2007 41 had high–quality MRI examinations at 10 years of age accompanied with cognitive data at 12 years of age. Voxel–based morphometry was performed on pre–processed T1 weighted images using SPM12. The association between full–scale IQ at 12 years and grey matter (GM) volume at 10 years was analyzed in a multiple regression model, adjusted for sex. Results are presented at voxel level $p<0.001$, family–wise error corrected at $p<0.05$ at cluster level.

Results: We found that GM volume was significantly related to IQ in areas with peak coordinates in insula in both hemispheres. These clusters were 1588 voxels large on the right side, 962 voxels on the left side. No significant negative associations were found between grey matter volume and IQ.

Conclusions: Reduced insular volume was associated with lower full–scale IQ for children born EPT. The insula is known to subserve a variety of functions, from sensory and affective processing to cognition. This region could be a potential neuroimaging biomarker for neurodevelopmental problems in this patient group.

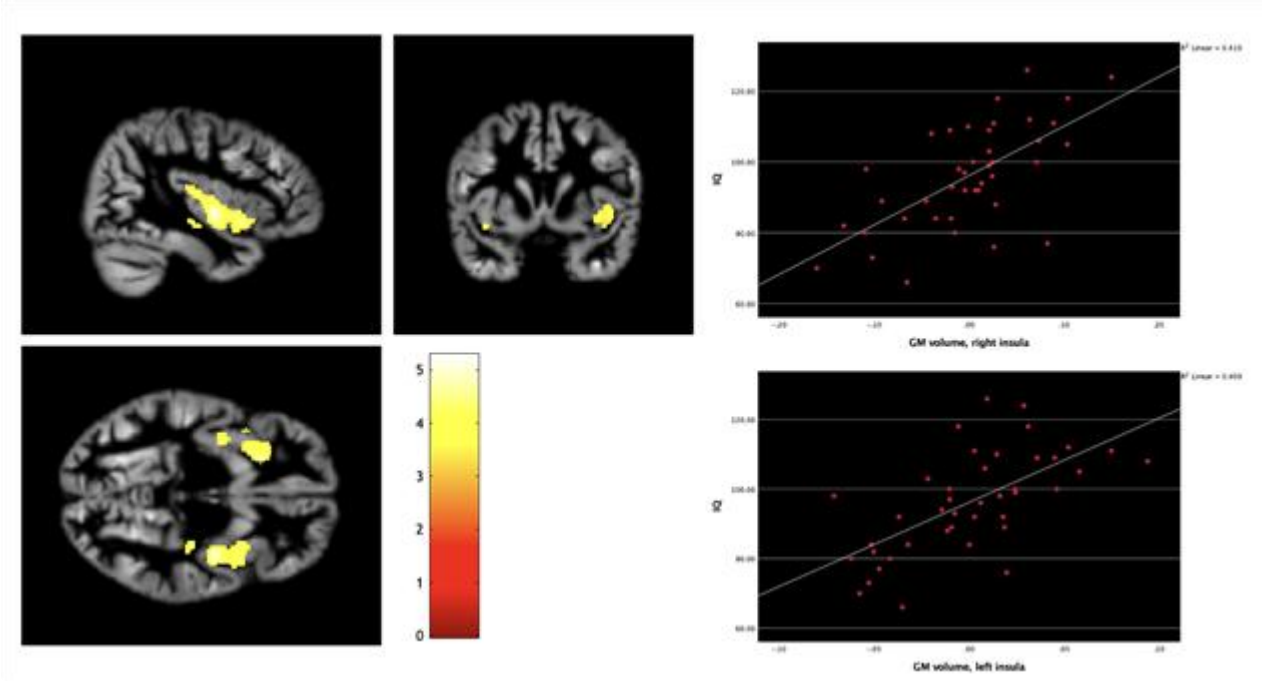

Figure 1: Areas where grey matter (GM) was positively related to the intelligence quotient (IQ) score, with peak coordinates in bilateral insula. Y axis: standardized GM volumes

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None declared

ID 718. No sign of crossed cerebellar atrophy (CCA) in infants with perinatal arterial ischemic stroke (PAIS) at three months of age

Miss Lisanne M. Baak[1], Ms Nienke Wagenaar[1], Mr Floris Groenendaal[1], Mr Jeroen Dudink[1], Ms Maria Luisa Tataranno[1], Miss Cora HA Nijboer[2], Ms Manon JNL Benders[1], Mr Niek E van der Aa[1]

[1]Department of Neonatology, University Medical Center Utrecht Brain Center and Wilhelmina Children's Hospital, Utrecht University, Utrecht, the Netherlands, [2]Department for Developmental Origins of Disease, University Medical Center Utrecht Brain Center and Wilhelmina Children's Hospital, Utrecht University, , the Netherlands

Background

Perinatal arterial ischemic stroke (PAIS) is caused by a disruption of arterial blood flow and often results in a significant cerebral lesion. Crossed cerebellar atrophy (CCA) is a form of diaschizis of the cerebellar hemisphere contralateral to the cerebral lesion, and has been described in adult and pediatric stroke, and in preterm infants with cerebral hemorrhage. We aimed to investigate the presence of CCA after perinatal arterial ischemic stroke.

Methods

We selected infants born ⩾36 weeks of gestation diagnosed with unilateral PAIS by MRI within 10 days after birth, and with a follow-up MRI performed around 3 months of age. Experienced neuro-neonatologists scored the arterial stroke territory, stroke size (small: perforator, or cortical stroke; medium: posterior, anterior, or middle branch of the middle cerebral artery (MCA), or posterior cerebral artery; or large: main branch MCA), and corticospinal tract (CST) involvement (posterior limb of the internal capsule and/or cerebral peduncle). Cerebellar volumes were determined quantitatively using semi-automatic brain tissue segmentation from the Developing

Human Connectome Project. Segmentation quality was inspected visually, and poor quality segmentations were excluded. The ratio between cerebellar hemispheres (contralesional/ipsilesional) was calculated with values <1 indicating CCA.

Results

Fifty–three patients (51% female) met inclusion criteria and had brain segmentations of sufficient quality available. Mean±SD gestational age at birth was 40.06±1.46 weeks with a birthweight of 3383±465 grams. The most frequent stroke arterial territory was a posterior trunk MCA (26%), followed by cortical (23%), perforator (21%), and main MCA stroke (9%). Twenty infants (38%) had CST involvement on the neonatal DWI, and 10 (19%) had asymmetrical myelination of the CST on the follow–up MRI. Mean cerebellar ratio at follow–up MRI performed at 13.58±1.44 weeks was 0.996±0.028, and did not differ across stroke size, stroke territory, or CST involvement.

Conclusion

In contrast to children and preterms with unilateral cerebral lesions, we found no signs of CCA in infants with unilateral PAIS at three months of age. Larger populations with longer follow–up periods are required to make definite statements about the development of CCA after PAIS.

This study was funded by ZonMw, the Netherlands, and the Wilhelmina Research Fund, the Netherlands. The authors have no conflicts of interest to declare.

ID 576. Age-dependent brain perfusion alteration in intrauterine growth restricted neonates at term equivalent age

Miss Seline Coraj[1,2], Alexandra De Silvestro[3,4,5], Ninib Yakoub[1,2], Thi Dao Nguyen[2], Cornelia Hagmann[3,6], Andras Jakab[4], Tilman Reinelt[1,2], Ruth O'Gorman Tuura[3,4], Giancarlo Natalucci[1,2]

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Background

Infants with intrauterine growth restriction (IUGR) are at risk for neurodevelopmental impairment. In IUGR altered cerebral blood supply may contribute to suboptimal brain maturation. Moreover, the concurrence of preterm birth by itself affects brain perfusion and later neurodevelopment. Yet, we still lack a complete understanding of cerebral perfusion alterations in IUGR. Here, we aimed to compare cerebral perfusion in neonates with IUGR and intrauterine growth appropriate for gestational age (AGA) by arterial spin labeling (ASL) MRI at term equivalent age (TEA).

Methods

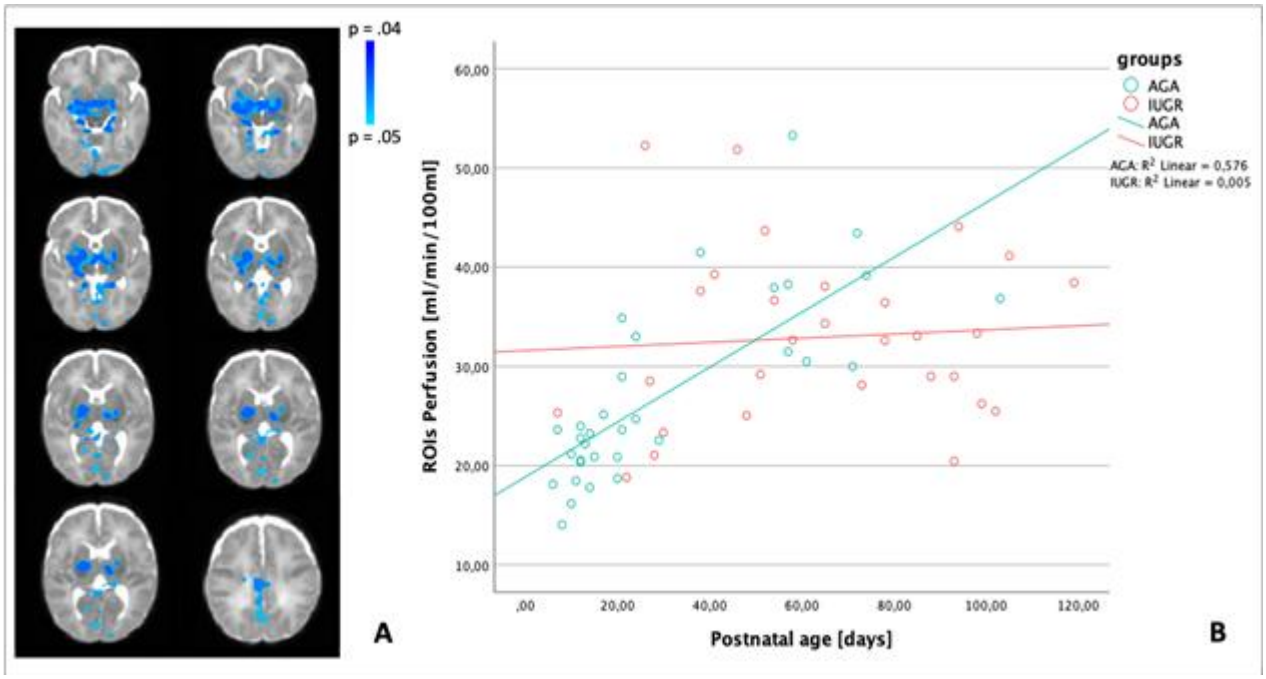
Our interim analysis of an ongoing prospective cohort study included 29 IUGR [15 female; postnatal age at MRI in days (mean±SD) 64.2±30.0; gestational age (GA) at birth in weeks 32.0±4.1; postmenstrual age (PMA) at MRI in weeks 41.1±1.3] and 33 AGA [16; 30.2±25.2; 36.7±4.2; 41.0±1.4] infants. IUGR was defined based on consensus criteria. Perfusion images were acquired without sedation on a 3T GE MR750 scanner (3D background–suppressed pCASL sequence), reconstructed and registered to a neonatal perfusion template. Next to whole brain grey matter (GM) perfusion, we compared perfusion voxelwise across groups (alpha = 0.05, TFCE corrected), including postnatal age at MRI, GA at birth and PMA at MRI as covariates. The interaction effect of postnatal age at MRI by group on perfusion was investigated separately. Regional perfusion values were extracted in the regions (ROIs) where we found significant interaction effects.

Results

No significant perfusion difference between groups but increasing voxelwise perfusion and whole brain GM perfusion with increasing postnatal age in both groups was found. Differences in the slope of postnatal age–dependent perfusion increase (AGA>IUGR) were found bilaterally in deep GM clusters extending into basal ganglia, thalamus, hippocampus, occipital and midline parietal cortices.

Conclusion

Findings in AGA are in line with previous literature. In IUGR the lower rate of CBF increase over time may be explained by unmet metabolic demands during a period of intrauterine and early postnatal insults. Difference in postnatal age between groups and the cross–sectional design limit result interpretation and hinder inference about brain maturation related perfusion trajectories. Nevertheless, our findings suggest altered brain perfusion in IUGR which is dependent on postnatal age.



A) Voxelwise correlation analysis between perfusion and groups accounting for postnatal age*group interaction.

B) Association between perfusion in the ROIs showing a significant interaction effect and postnatal age over groups.

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B) Association between perfusion in the ROIs showing a significant interaction effect and postnatal age over groups.

None declared

ID 724. Assessment of cerebral perfusion using transfontanellar contrast-enhanced ultrasound in neonates with vein of Galen malformation

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Background

Vein of Galen aneurysmal malformation (VGAM) is a very rare congenital cerebrovascular Malformation with high morbidity and mortality in neonatal manifestation. The aim of this study was to dynamically assess the influence of neuroendovascular therapy on cerebral perfusion using transfontanellar contrast-enhanced ultrasound.

Methods

At present, this prospective, monocentric study (DRKS00030052) included n=6 neonates. All subjects received an intravenous ultrasound contrast agent (UCA) bolus of 0.08 ml/kg SonoVue® at three study time points (T1 = within 24h before endovascular therapy, T2 = within 24h after therapy, T3 = one week after therapy).

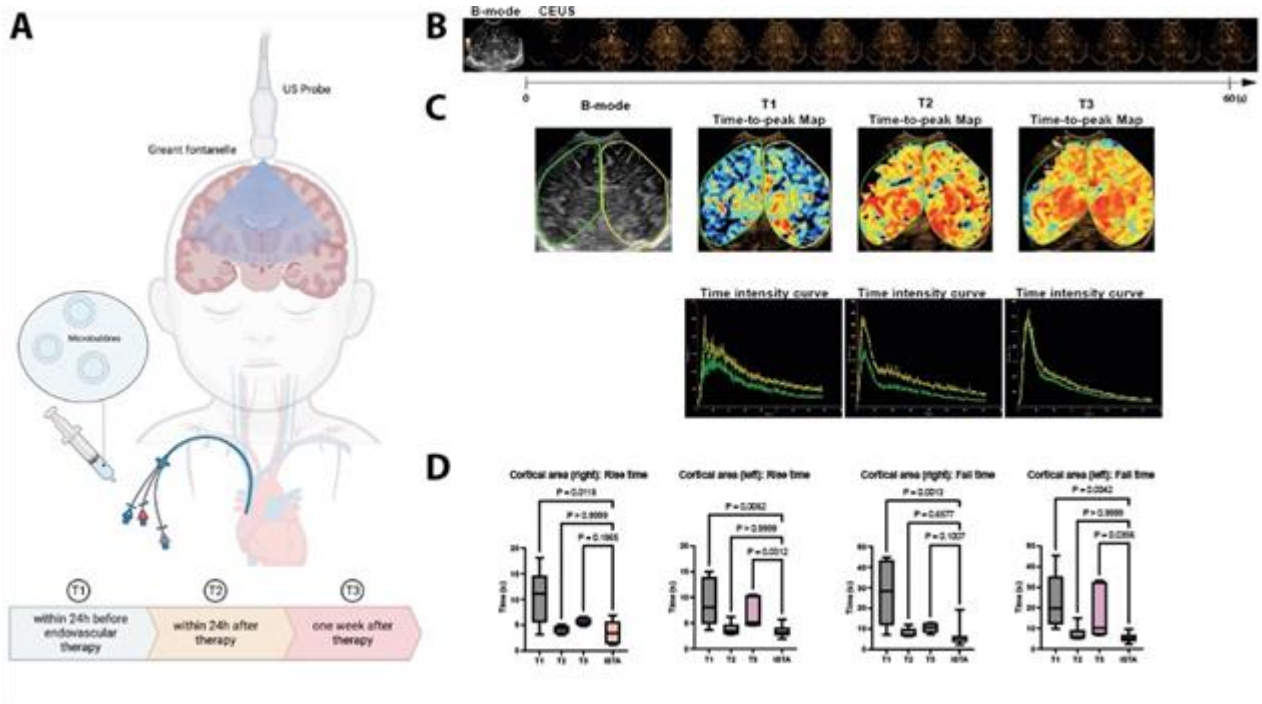
Time intensity curves of the UCA were quantified on standard middle coronal sections using VueBox® software (Bracco, Italy). Results were compared to existing control data of patients with coarctation of the aorta (ISTA).

Results

Neonates with VGAM were included at an age of 3±2.5 days after birth and with a weight of 2932±315g. One infant died before the time point T3. In total, n=15 datasets were analyzed. When compared with baseline values (T1) the analyses of the hemispheres revealed changes in the time intensity curves after therapy (T2, T3). The time intensity curves at T1 showed a late peak (rise time (RT) right: 8.3±2.1s) and a slow washout (fall time (FT) right: 37.3±13.9s) and after therapy an early, steep peak (RT right T2: 4.3±1s, P=0.01; T3: 5.8±1.3s, P=0.004) and a faster washout (FT right T2: 13.3±10s, P=0.01; T3: 14.7±9.7s, P=0.01). Analysis of smaller regions of interests revealed an increase in rise time (RT right: 10.6±5.3s, P=0.0118) in the cortical areas (cortical grey and subcortical white matter) at T1 compared to the ISTA control group (3.6±2.2s), which normalized at T2 (4.2±0.8s, P=ns) and T3 (5.7±0.7s, P=ns). In this regions fall time was also significantly increased compared with the ISTA control group (6.7±5.7s) at T1 (FT right: 27.4±15.3s, P=0.001) and reduced at T2 (8.0±2.2s, P=ns) and T3 (10.5±2.5s, P=ns). Smaller treatment effects could be detected in periventricular white matter and deep gray matter.

Conclusion

Transfontanellar contrast–enhanced ultrasound is suitable for quantifying hemodynamic cerebral changes and for mapping endovascular therapy effects in VGAM.



A:Transfontanellar contrast–enhanced ultrasound. B:Representation of ultrasound contrast agent wash–in and wash–out.

C:Time–to–peak maps and time intensity curves at T1–T3.

D:Evaluation of cortical areas at T1–T3 compared with the control group.

A:Transfontanellar contrast–enhanced ultrasound. B:Representation of ultrasound contrast agent wash–in and wash–out.

C:Time–to–peak maps and time intensity curves at T1–T3.

D:Evaluation of cortical areas at T1–T3 compared with the control group.

None declared.

ID 943. DISTINCTIVE ANATOMICAL PATTERNS OF THE PREMATURE BRAIN GROWTH AT TERM EQUIVALENT AGE

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Background

Defining the structural maturational patterns of early brain development in preterm infants is a critical step to identify the divergent developmental trajectories that may appear even in absence of brain pathology. These patterns may be associated with neurodevelopmental disorders that could arise later in life.

Since many significant developmental changes occur during the third trimester of gestation, specific anatomical brain atlases for premature infants are crucial for the study of brain development in this population.

Aim

To describe anatomical patterns of brain development at term equivalent age in 3 groups of preterm infants without major brain injury and compare them to a group of healthy newborns born at term.

Methods

We use a high–quality, publicly available, parcellated brain atlas of term infants [1] to quantify volumes of several brain structures. Additionally, we adapted an adult cerebellum parcellation [2] in order to account for potential differences in this structure, which develops considerably during the third trimester of pregnancy.

Cerebro–spinal fluid volume in subarachnoid spaces and within the ventricles was also obtained.

Results

Four templates were created from selected MRI (59 out of the initial 114 brain images passed quality control) at 40 ±2 weeks corrected age: 15 from infants born at 24–26 weeks of gestational age (wGA), 15 from 27–29 wGA, 16 from 30–32 wGA, and 13 from 40–42 wGA. Total brain volume and volume from different brain regions were segmented: total white matter, cortex grey matter, central grey matter, hippocampal–amygdala complex, cerebellum, and ventricles (Figure 1).

Conclusions

Preterm birth is associated with brain volume loss with respect to infants born at term. We observe that volumetric deficits increase proportionally to the degree of prematurity, except ventricle volume, which was much larger in all premature groups. Some brain regions showed differentially larger volume deficits in preterm: corpus callosum, cerebellum and hippocampal–amygdala complex, which warrants further investigation for associated function impairments.

[1] Chen L, et al. A 4D infant brain volumetric atlas based on the UNC/UMN baby connectome project (BCP) cohort. Neuroimage. 2022; 253:119097.

[2] Diedrichsen, et al. A probabilistic atlas of the human cerebellum. Neuroimage; 2009. 46(1), 39–46.

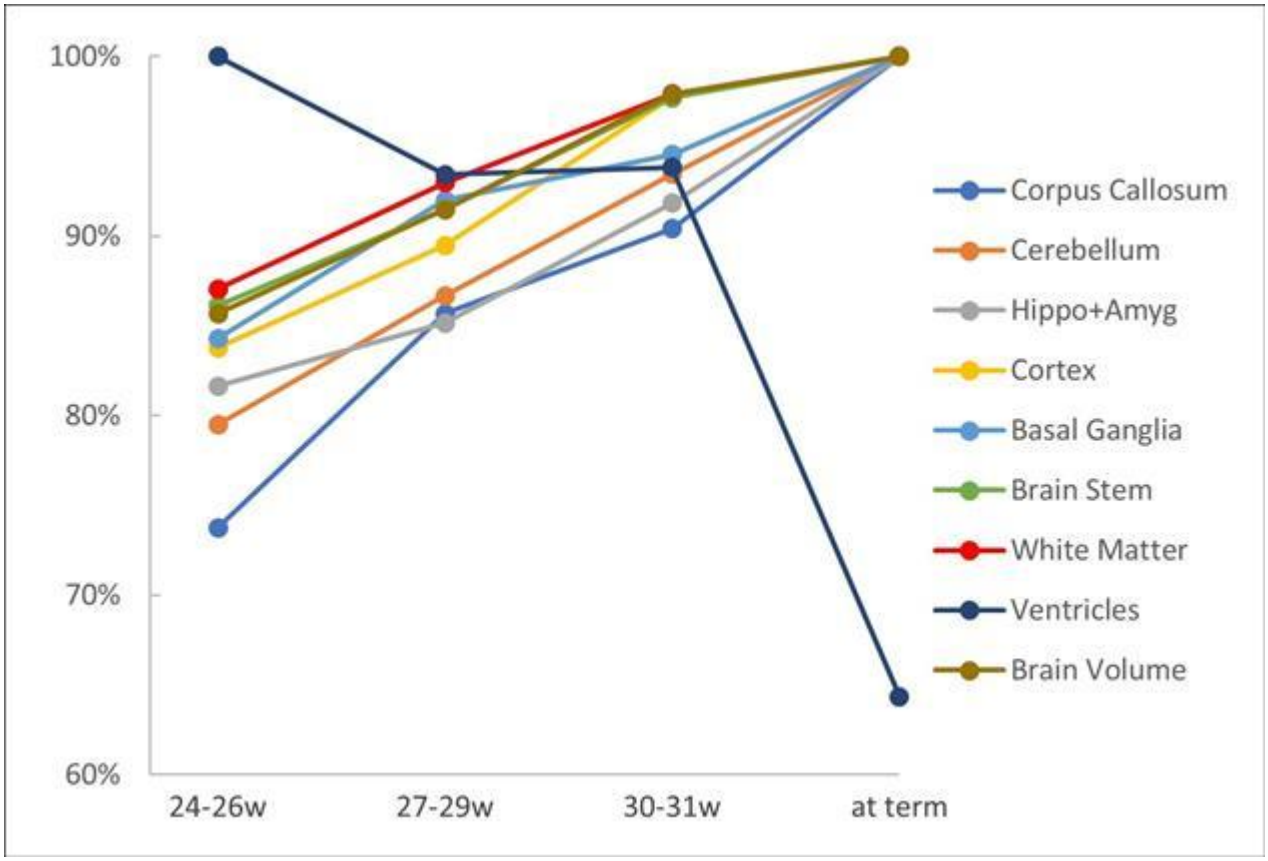

Figure 1 Brain structure relative volumes of the three groups of preterm with, respect to term newborn. For ventricle, the most premature group (with larger ventricles) is used as reference.

Figure 1 Brain structure relative volumes of the three groups of preterm with, respect to term newborn. For ventricle, the most premature group (with larger ventricles) is used as reference.

This work was supported by a Grant PI08/00110 from the Instituto de Salud Carlos III co-funded by the European Regional Development Fund.

ID 638. LONG-TERM IMPACT OF POSTHEMORRHAGIC VENTRICULAR DILATATION ON CEREBRAL OXYGENATION IN PRETERM INFANTS WITH INTRAVENTRICULAR HEMORRHAGE

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Background:

Intraventricular hemorrhage (IVH) and its associated complication, post–hemorrhagic ventricular dilatation (PHVD), continue to be a major cause of morbidity and mortality in preterm infants. While previous studies have shown that IVH reduces regional cerebral oxygen saturation (rScO2) in the short–term, little is known about long–term effects, and in particular the impact of developing consecutive PHVD. The objective of this study was to examine the long–term impact of PHVD on rScO2 in infants with IVH.

Methods:

In this prospective study preterm neonates with a gestational age <33 weeks born from 2013 to 2023 were investigated. Peripheral and regional oxygen saturation (rScO2) were monitored repetitively at the time of IVH diagnosis for 10 weeks of life. Following error correction, cerebral fractional tissue oxygen extraction (cFTOE) was computed. The median values of rScO2 and cFTOE were statistically analysed by postnatal age and injury group (IVH without PHVD; IVH with PHVD) utilizing non–parametric statistical methods.

Results:

This study comprised 146 preterm neonates with a median gestational age of 25.7 (23.3–28.1) weeks and a diagnosis of IVH (grade I: 5.2%, II: 19.1%, III: 40.4%, IV: 35.3%). Among them, 53% (n=77) were diagnosed with PHVD. The results demonstrate a significant distinction between neonates with and without PHVD. Specifically, infants diagnosed with IVH and PHVD displayed lower rScO2 levels (55.0% vs. 60.3%, p⩽0.001) but higher cFTOE levels (0.42 vs. 0.38, p=0.006) during the first 10 weeks of life compared to infants without PHVD.

Conclusion:

A significant association between PHVD and a protracted decline in regional cerebral oxygen saturation coupled with an elevation in cerebral fractional tissue oxygen extraction was demonstrated, indicating the severity of the inflicted injury.

None declared

ID 517. BRAIN INJURY PATTERNS AND NEURODEVELOPMENTAL OUTCOME IN PRETERM INFANTS WITH PERINATAL ASPHYXIA.

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Background: Brain injury patterns on magnetic resonance imaging (MRI) following perinatal asphyxia are well-described for (near-)term infants. However, data on those born prematurely is scarce.

Objective: To explore cranial MRI and 18-24 month neurodevelopmental outcome among preterm infants with perinatal asphyxia.

Methods: A retrospective single-centre study including infants with gestational age (GA) 24-36 weeks and perinatal asphyxia, defined as ⩾2 of the following criteria: signs of fetal distress, umbilical cord pH ⩽7.00, and/or 5-minute Apgar ⩽5. Injury was separately analysed for infants with GA <32 weeks (group 1) and 32-36 weeks (group 2). MRI performed <36 weeks postmenstrual age (early MRI) was categorised

according to predominant injury pattern. MRI performed around (near–)term–equivalent age (TEA, between 36–44 weeks) was analysed using the Kidokoro scoring system. Adverse outcomes included death, cerebral palsy, epilepsy, severe hearing/visual impairment, or neurodevelopmental delay at 18–24 months (Bayley–III motor and/or cognitive composite score <85, or Griffiths developmental quotient <88).

Results: Of 173 eligible infants, n=18 died before MRI and n=75 had no MRI <44 weeks postmenstrual age. N=80 with early MRI (group 1: n=35, group 2: n=22) and/or MRI around TEA (group 1: n=38, group 2: n=18) were included (median GA 30.6, IQR 5.2 weeks). The main injury patterns on early MRI were intraventricular and/or intraparenchymal haemorrhage (supratentorial 28%, infratentorial 11%), white matter (WM) and/or watershed injury (26%), deep grey matter (DGM) injury (7%) and near–total injury (5%). Group 1 more often demonstrated haemorrhage (n=18, 51%), while group 2 more frequently showed WM/watershed injury as main abnormality (n=10, 45%, p=0.022). Kidokoro global brain abnormality scores (GBAS) for MRI around TEA for the overall group were normal, mild, moderate and severe in respectively 29%, 36%, 21% and 14%. Kidokoro GBAS and sub–scores were comparable between groups 1 and 2. Thirty–five infants (44%) had an adverse outcome. DGM injury on early MRI (p=0.001), and Kidokoro WM (p=0.034), DGM (p=0.009) and GBAS (p=0.008) were associated with adverse outcomes.

Conclusions: Extremely–very preterm infants with perinatal asphyxia mainly demonstrate haemorrhages, and moderate–late preterm infants predominantly show WM injury on cranial MRI. MRI findings, particularly DGM abnormalities, were associated with adverse neurodevelopmental outcomes.

None declared

ID 196. Erythropoietin for Neuroprotection in Very Preterm Infants

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Background: Currently the use of promising neuroprotective technologies in premature newborns are increasingly discussed. A number of studies have shown that high doses of erythropoietin (rhEPO) have a neuroprotective effect, but the benefits and safety of this therapy in premature infants have not been established.Objective: To study the safety and effectiveness of the use of rhEPO as neuroprotective therapy in premature infants with extremely low birth weight (ELBW).

Methods:A single-center randomized study was conducted, which included 190 ELBW infants who received various regimens of rhEPO. The gestational age of the children ranged from 26 to 30 weeks. The children were divided into 3 groups depending on the treatment regimen: group 1 (n=63) — premature newborns who received 600 IU/kg per week subcutaneously; group 2 (n=76) — received 1200 IU/per week subcutaneously; group 3 (n=51) premature infants who did not receive rhEPO — the control group. The neurological development of children at the time of discharge from the hospital, as well as from 22 to 30 months of life, was evaluated. 168 children were included in the analysis after discharge: 122 received rhEPO according to various schemes and 46 did not receive rhEPO.

Results: There were no significant differences regarding short–term outcomes such as intraventricular hemorrhage, periventricular leukomalacia, retinopathy, necrotizing enterocolitis, and bronchopulmonary dysplasia, or death or in the frequency of serious adverse events. There was no significant difference between the rhEPO groups and the control group in the incidence of severe neurodevelopmental impairment at 22–30 months of life (relative risk, 1.06; 95% confidence interval 0.66 to 1.84; p=0.735).

Conclusion: Treatment with different doses of rhEPO in ELBW infants 600 IU per week vs 1200 IU per week vs control group did not result in a lower risk of severe neurodevelopmental impairment at the age of 22 to 30 months of life. The effectiveness of erythropoietin therapy, the time of its start and various treatment regimens remain controversial issues that require further study.

None declared

ID 692. WEARING CPAP CAPS IS ASSOCIATED WITH HEAD GROWTH RESTRICTION AND ALTERED SKULL MORPHOLOGY IN NEWBORN INFANTS.

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Background. In infancy, occipitofrontal head circumference (OFC) is routinely measured as a surrogate of skull volume and brain size. Subnormal postnatal head growth is associated with impaired neurodevelopmental outcome and may not be compensated after infancy. Premature infants, who need CPAP often fall below OFC trajectories, while vertical head distortion has been observed. As OFC disregards vertical head growth, ear–to–ear distances (EED) have been suggested as complimentary measurements, and growth charts for transfontanellar EED (fEED), transvertical EED (vEED) and a derived head volume index (HVI) have been established.

Methods. Weight, length, OFC, fEED and vEED of 4591 infants on our neonatal wards were measured repeatedly between birth and discharge. The mode of ventilation was continuously recorded. Z–Scores of anthropometric data were compared by repeated–measures ANOVA for groups of cumulative cap time in increments of 15 days. Geometric indices for head eccentricity and head volume were derived from OFC, fEED and vEED. Growth patterns were analyzed by panel regression models. 367 infants were followed up after 6 months for assessment of neurodevelopmental outcome by Bayley Scales of Infant and Toddler Development (3rd Edition). Associations between Bayley–III scores and postnatal growth patterns were analyzed by Pearson’s correlation, multivariate regression models and confirmatory factor analysis.

Results. Z–Scores of OFC, fEED, vEED and HVI are significantly lower in groups with longer cumulative CPAP cap time. This observation was confirmed in panel models controlling for gestational age, gender, weight and length. Cap time positively influences head eccentricity indices, while weight and length are not significant and do not contribute to model fit. Catch–up growth after cessation of wearing of the cap was not observed for the duration of the hospital stay. Bayley–III scores were not associated with HVI development, which was confirmed by factor analysis. However, cumulative cap time was negatively associated with Bayley–III scores, indicating confounding variables such as disease severity.

Conclusion. Wearing tight–fitting CPAP heads is associated with head growth restriction (OFC and head volume) and an increase in head eccentricity in newborn infants. This altered growth pattern is not associated with the neurodevelopmental outcome at 6 months of age.

	OFC	vEED	fEED	HVI	vECC	fECC	2vEED/OFC-index	2fEED/OFC-index
cap time	-0.337***	-0.068***	-0.071**	-324.469***	0.003***	0.002***	0.002***	0.003***
	(0.017)	(0.018)	(0.035)	(26.654)	(0.0004)	(0.001)	(0.0003)	(0.001)
ga	-0.061***	-0.108***	-0.158***	-206.441***	-0.007***	-0.007**	-0.005***	-0.007*
	(0.019)	(0.023)	(0.059)	(34.755)	(0.002)	(0.003)	(0.001)	(0.004)
male gender	0.007***	0.004***	0.0001	31.903***	-0.0005***	-0.001***	-0.0004***	-0.001***
	(0.001)	(0.001)	(0.002)	(1.251)	(0.00005)	(0.0001)	(0.00004)	(0.0001)
weight	0.573***	0.407***	0.830***	1,110.755***				
	(0.032)	(0.040)	(0.101)	(51.093)				
length	0.478***	0.245***	0.164***	532.975***				
	(0.005)	(0.006)	(0.015)	(7.698)				
Constant	6.872***	5.390***	9.649***	-15 875.540***	0.767***	0.831***	1.283***	1.463***
	(0.211)	(0.245)	(0.643)	(357.072)	(0.011)	(0.021)	(0.009)	(0.029)
Observations	11,451	6,178	1,566	12,517	11,278	2,442	11,279	2,442
R^2	0.944	0.886	0.864	0.836	0.712	0.721	0.920	0.850
Adjusted R^2	0.944	0.885	0.863	0.836	0.712	0.720	0.920	0.850
F Statistic	38,459.690***	8,584.389***	1,196.416***	26,318.730***	219.164***	62.640***	218.228***	87.959***

Linear panel regression. Cap time, gestational age (ga), gender, weight and length are response. OFC, vEED, fEED, HVI, and indices of transvertical (vECC), transfontanellar eccentricity (fECC), and EED/OFC–indices are predictors.

Linear panel regression. Cap time, gestational age (ga), gender, weight and length are response. OFC, vEED, fEED, HVI, and indices of transvertical (vECC), transfontanellar eccentricity (fECC), and EED/OFC–indices are predictors.

None declared

ID 1026. Influences of medical parameters on automated derived sleep-wake architecture in preterm infants

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Background: Integration of sleep assessment in the NICU as standardized clinical care is gaining attention. Curtailment of sleep by different neonatal perturbations such as noxious stimulation, cardiorespiratory instability, or exposure to the extra–uterine environment, may be detrimental for further brain maturation and neuronal plasticity. The challenge is to distinguish medical and perinatal stressors that have the highest impact on sleep physiology. The aim of this study is to identify risk factors for impaired sleep organization in preterm and sick babies by automated reporting of neonatal sleep parameters.

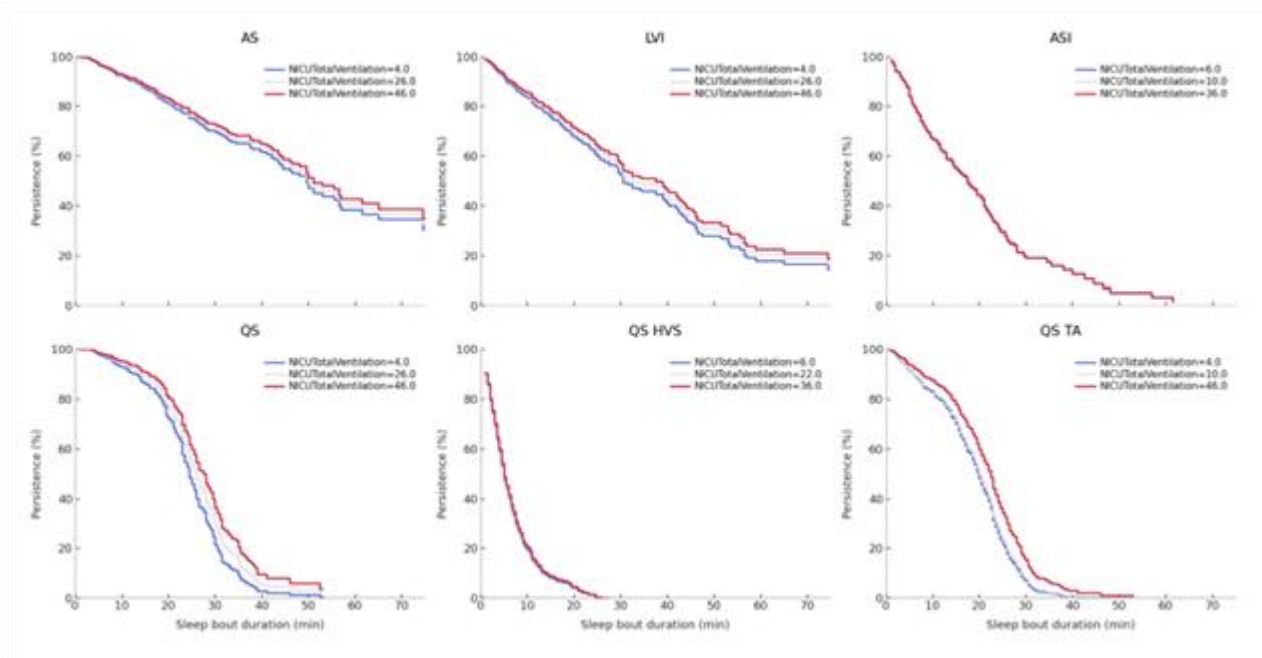
Methods: In 79 preterm infants (GA 23 6/7 and 33 6/7 weeks), we monitored overnight multichannel–polysomnography between 36 to 45 4/7 weeks PMA, before discharge. All infants have a normal BSID–III score at 24 months. The sleep staging algorithm runs the EEG in five sequential steps: artefact detection, data cleaning, sleep state classification, reliability analysis and hypnogram construction, and classifies each 30s EEG segment as one of four sleep states: LVI, ASI, QS–HVS or QS–TA. We constructed Kaplan–Meier survival curves of sleep—wake bout durations and investigated the effect of PMA, Kangaroo Care (KC), total number of skin

breaking procedures, total days of ventilation and CRIB–II score as covariate with the Cox–regression model.

Results:
A significant effect of PMA on the sleep bout duration is found for AS, QS, QS–HVS and QS–TA. The durations of QS–TA bouts decrease, while the duration of HVS bouts increase with PMA. Moreover, with more KC hours during NICU stay, AS bout durations last longer, whereas a higher CRIB–II score gives shorter ASI bouts. Total days of ventilation is negatively correlated with the total percentage of sleep and gives rise to longer QS–TA bouts, the more immature QS state.

Conclusion: Normative modelling approaches can characterize typical population variation in a data–driven fashion. By mapping datasets from different neonatal cohorts, we can detect perinatal factors that have the highest impact on neonatal sleep architecture, which can support hypothesis–driven interventions and serve as tool for post–outcome measures.

AS: Active–Sleep
QS: Quiet–Sleep
ASI: Active–Sleep stage I
LVI: Low Voltage Irregular pattern or ASII
QS–HVS: QS–High Voltage Slow Wave Sleep
QS–TA: QS–Tracé Alternant



Kaplan–Meier Survival curves with total days of ventilation as covariate on sleep bout durations for AS, LVI, ASI, QS, QS–HVS and QS–TA (with significant longer QS–TA for more ventilation days)

Kaplan–Meier Survival curves with total days of ventilation as covariate on sleep bout durations for AS, LVI, ASI, QS, QS–HVS and QS–TA (with significant longer QS–TA for more ventilation days)

none declared

ID 595. Validation of a Machine Learning Algorithm for Identifying Infants at Risk of HIE in Unseen Data

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Background:
Hypoxic ischaemic encephalopathy (HIE) is one of the leading causes of morbidity and mortality in infants worldwide. Earlier initiation of therapeutic hypothermia can significantly improve outcomes. We have previously reported a machine learning algorithm which may support clinical decision making in neonatal hypoxic ischaemic encephalopathy. We aimed to validate a HIE prediction algorithm to identify infants at risk of HIE immediately after birth using readily available clinical data.

Methods:
A retrospective review of electronic health record data of all term deliveries between January 2017–December 2021. Infants > 36 weeks' gestation with the following clinical variables available were included; Apgar score at one and five minutes, first postnatal pH, base deficit, and lactate values taken within one hour of birth. Previously trained open-source logistic regression (LR) and random forest (RF) prediction algorithms (https://www.infantcentre.ie/predictionapp.html) were used to calculate a probability index (PI) for each infant for the occurrence of HIE.

Results:

Over the study period 1229 infants had a blood gas measurement in the first hour of life. Of these 1093 had a complete dataset available: 76 (6.95%) with HIE and 1017 non–HIE. Of the 76 infants with HIE, 37 were clinically classified as mild, 29 were moderate and 10 were severe. 46 (60.5%) infants received therapeutic hypothermia (7 [19%] mild, 29 [100%] moderate and 10 [100%] severe). Both models performed well, but best overall accuracy was seen with the RF model. Median(IQR) probability index in the HIE group was 0.70(0.52–0.86) vs 0.05(0.016–0.16), ($p<0.001$) in the non–HIE group. AUROC for prediction of HIE = 0.925 (0.893–0.958), ($p<0.001$). Using a PI cut off to optimise sensitivity of 0.30; 947 of the 1093 (86.6%) infants were correctly classified by the algorithm (sensitivity = 86%, specificity 87%). This cut off identified 78.4% of mild HIE, 93% of moderate HIE and 100% of severe HIE cases.

Conclusion:

In a large unseen data set an open–source algorithm could identify infants at risk of HIE in the immediate postnatal period. This may aid focused clinical examination, transfer to tertiary care (if necessary) and timely intervention.

None declared

ID 548. PROGNOSTIC ROLE OF EARLY SERUM PHOSPHATE LEVEL ON PERINATAL MORTALITY IN INFANTS WITH NEONATAL ENCEPHALOPATHY

Doctor Kata Kovacs[1], Doctor Benedek Paszthy-Szabo[1], Doctor Marianna Dobi[1], Doctor Hajnalka Barta[1], Doctor Eniko Szakmar[1], Doctor Agnes Jermendy[1], Professor Miklos Szabo[1]

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Background: Birth asphyxia and neonatal encephalopathy (NE) is a leading cause of perinatal mortality and neurodevelopmental impairment. Multiorgan failure is a frequent complication in neonates with NE and in the most severe cases may lead to perinatal death. Multiorgan involvement can be characterized by several laboratory tests and clinical parameters, however, a single biomarker would be desirable for early risk stratification of infants. Our aim was to describe the possible association between early, routinely measured serum laboratory parameters and perinatal mortality in neonates with NE, treated with hypothermia.

Methods: In this retrospective cohort study, medical records of neonates with NE, born between 2006 and 2017 were reviewed. Patients were cared for in the NICU of the Department of Paediatrics Semmelweis University, Budapest, Hungary. Results of the first, routinely measured 20 laboratory parameters (mostly <6 hours of life) and clinical characteristics of patients were analyzed. The primary outcome was defined as in-hospital mortality. The effects of early laboratory biomarkers on mortality were analyzed by logistic regression modelling which was adjusted for gestational age, initial base excess and initial amplitude-integrated electroencephalography (aEEG) background activity.

Results: A total of 237 neonates were analyzed, the rate of perinatal mortality was 17% in this cohort. Multiple logistic regression analysis showed that for every 1 mmol increase in serum phosphate level the odds of perinatal mortality increased by 5.93–fold (95% CI 1.57–22.41; p=0.009) independently from gestational age, initial base excess and the initial aEEG background activity. ROC curve analysis revealed that a cut–off value of 2.5 mmol/l serum phosphate level excellently predicts early mortality with 88.5% sensitivity and 84.5% specificity (AUC 0.903). Among survivors, neurodevelopmental impairment (using the Bayley–II test) was not associated with the initial serum phosphate level.

Conclusion: Based on our results, increased initial serum phosphate level is associated with higher odds of in–hospital mortality (nearly 6–fold increase for every 1 mmol/l) in infants with NE. This result suggest that early serum phosphate may be a potential biomarker in the future and may additionally be used to determine the severity and short–term prognosis of neonates with NE earlier than other conventional predictors.

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ID 711. Neonatal arterial ischemic stroke: role of brain monitoring and seizure treatment response.

Doctor Veronica Pegoraro[1,2], Geneviève Malfilatre[3], Isabella Mauro[4], Robertino Dilena[5], Cecilia Zardini[6], Renaud Viellevoye[7], Maria Luisa Tataranno[8], Beatrice Desnous[9], Duccio Maria Cordelli[2], Francesca Campi[10], William Rozalen[9], Maria Elena Cavicchiolo[11], Giulia Ventura[4], Stefano Sartori[6], Carla Pittini[4], Mathieu Milh[9], Maria Roberta Cilio[1]

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BACKGROUND: There is a lack of considerable variations in clinical practice regarding neonatal seizures management, but an etiology–specific approach is emerging. We investigate the response to anti–seizure medications (ASMs) in a population of neonates with acute ischemic stroke (AIS).

METHODS: This is a retrospective, multicentric, multinational study of term newborns with an MRI-confirmed neonatal diagnosis of AIS, who presented seizures and were monitored by continuous (a)EEG monitoring. Patients were included if an adequate (a)EEG documentation regarding response to ASMs was available. Choice and doses of ASM were at the discretion of the treating clinician. Treatment response was defined as (a)EEG-confirmed seizure resolution and continuous monitoring until seizure freedom for 24 hours.

RESULTS: Seventy-four neonates referred to ten level III NICUs and diagnosed with AIS had continuous (a)EEG recordings available for review. 23/74 were excluded because of fragmented data about ASM administration, or because the duration of monitoring lasted less than 24 hours after last seizure. Forty-eight patients were included. The most frequently recorded seizure type was focal clonic (76%), although a significant percentage of infants presented with ictal apnea (16%). Most neonates (47/51, 92%) had brain monitoring started at 7.7 ± 12.6 hours after the onset of symptoms. aEEG was the main monitoring tool (60.7%). Mean time to treatment was 8.2 ± 11.8 hours, and the most frequently administered first-line ASM was phenobarbital (85%). Seizures were completely controlled within 24 hours from onset in 54% of neonates. Interestingly, sodium-channel blockers, including IV phenytoin and lidocaine and oral carbamazepine were effective in almost all neonates in whom they were trialed (20/21, 95%), while PB was only effective in 30% of patients (13/43, $p < 0.001$). Furthermore, patients treated with PB required additional loads and significantly higher mean doses than those treated with sodium-channel blockers ($p < 0.001$).

CONCLUSION: This is the first study evaluating the treatment response in a homogeneous cohort of neonates with AIS-associated seizures. Our data provide

evidence for the higher efficacy of sodium–channel blockers over PB and other ASMs. Randomized–controlled studies are needed to confirm our data and move toward an accurate, etiology–specific treatment of neonatal seizures.

Timing of administration of each anti-seizure medication and cumulative response to treatment separately for anti-seizure medication

Timing of administration of each anti-seizure medication and cumulative response to treatment separately for anti-seizure medication

None declared

ID 571. Neighborhoods socioeconomic deprivation and severity of neonatal hypoxic-ischemic encephalopathy

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Background:

Neonatal hypoxic–ischemic encephalopathy (NHIE) is a leading cause of neonatal death and neurodevelopment disabilities. While maternal socioeconomic deprivation is widely recognized as a risk factor of perinatal disease, its effects on neonatal HIE have been little investigated.

Objective:

To investigate the association of deprived neighborhoods with the severity of the NHIE.

Methods

A single–center retrospective and observational cohort study included newborns born between the 1st January 2015 and the 31th December 2020, and admitted for moderate and severe NHIE and who required therapeutic hypothermia. The severity of NHIE was defined by early death or severe brain damage (NICHD, stages 2B and 3). Maternal socioeconomic status was assessed using a geographic Socioeconomic Deprivation Index (SDI). Logistic regression was used to evaluate the association of deprived socioeconomic status and severity of NHIE.

Results

The study included 107 infants; 29 (27%) infants had severe NHIE. Maternal and neonatal characteristics were similar between the “no severe NHIE” and “severe NHIE” groups. 20/29 (69%) infants with severe NHIE were born to mother living in deprived neighborhoods compared to 27/78 (35%) in the other infants group (p=0.01). After adjusting for perinatal characteristics and severity factors of perinatal asphyxia, infants born to mother living in deprived neighborhoods had 4 fold higher odds to have severe form of NHIE (aOR= 4,4 (95% CI, 1.54–12.79; p=0.006).

Conclusion:

Newborns born in a context of perinatal asphyxia and born to mother living in deprived neighborhoods have high risk of developing a severe form of neonatal HIE.

none declared

ID 672.Correlation of Therapeutic Hypothermia for HIE and Pulmonary Hypertension

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Background and Aim

Neonates with perinatal asphyxia and moderate to severe Hypoxic Ischemic Encephalopathy(HIE) are currently treated with Therapeutic Hypothermia(TH) as a part of brain protective strategy. Perinatal asphyxia is a risk factor for development of Persistent Pulmonary Hypertension(PPHN). In animal studies, hypothermia was associated with an increase in pulmonary vascular resistance(PVR). One degree drop in temperature can increase PVR by 1–2%. It was suggested that increasing oxygen requirement during TH is probably attributable to PPHN and may have serious clinical consequences.

The aim of this study is to evaluate any correlation of therapeutic hypothermia and development of pulmonary hypertension.

Method

Retrospective study of all infants who underwent TH for moderate to severe HIE over a period of 3 years(Jan 2020—Dec 2022). Data was collected from Badger(Electronic Patient Record). The diagnosis of PPHN was based on clinical signs (pre and post ductal saturation difference >10%, high oxygen requirement) and/or Echocardiography findings of raised pulmonary pressures. Correlation of TH and PPHN was determined. Ventilation days and length of hospital stay was recorded. Treatment with inhaled nitric oxide(iNO) and inotropes was assessed. Outcome was measured in terms of MRI brain severity and feeding method at discharge.

Result

Total 60 neonates were included in the study who were treated with TH for HIE. 17% were diagnosed with PPHN. 60% developed moderate PPHN and 20% had mild PPHN. All were successfully treated for PPHN. Correlation of TH causing PPHN was found in 60% whereas, 40% who developed PPHN did not had association with TH. Only 50% required Nitric oxide for PPHN management. Table1

Conclusion

In this small cohort of infants who underwent TH for HIE, 10% developed PPHN after initiation of TH. Clinicians treating these infants need to be aware of this potential complication when providing TH for HIE. Neonates with lower Apgar score are more prone to develop PPHN while undergoing TH. These infants need longer ventilation days and length of hospital stay.

No difference in immediate outcomes like MRI brain changes and feeding at discharge are observed.

Large multicentral studies are urgently required to establish this association to provide robust evidence.

Table 1: Comparison of Neonates who developed PPHN vs No PPHN while undergoing Therapeutic Hypothermia for HIE

	TH with PPHN (n=10)	**TH without PPHN (n=50)**
Gestational age (mean ± SD)	39.5 ± 2.1	38.3 ± 1.5
Birth weight, kg (mean ± SD)	3.5 ± 0.98	3.2 ± 0.6
APGAR at 5 min (mean ± SD)	2.7 ± 2.3	3.8 ± 2.7
Inotropes needed (%)	100%	40%
Inhaled Nitric Oxide (%)	50%	0
Ventilation days (mean ± SD)	5.7 ± 1.3	2.8 ± 1.7
Length of hospital stay (mean ± SD)	14 ± 10	12 ± 13.2
Outcome		
MRI findings:		
1. Mild HIE (%)	20%	16%
2. Moderate HIE (%)	10%	28%
3. Severe HIE (%)	30%	22%
4. Normal MRI (%)	30%	34%
Bottle/breastfeeding at discharge (%)	60%	68%
Nasogastric feeding at discharge (%)	30%	24%
Deceased	1	4

None declared