activated microglia populated the area in which neurons had disappeared.

Conclusions Acute development of NEC is associated with neuron loss, microglial activation, and increased IL-8 levels in the hippocampus of preterm pigs. Gut inflammatory disorders and increased intestinal permeability may affect the immature brain and contribute to long term neurological disorders.

Objective The Blood-brain barrier (BBB) is a complex structure that protects the central nervous system (CNS) extracellular fluid from peripheral insults. Understanding the molecular basis and functioning of the BBB has a significant potential for future strategies to prevent and treat neurological disorders. The aim of our study was (1) to investigate BBB alterations in an excitotoxic model and (2) to test the protective properties of melatonin.

Methods The glutamate analogue ibotenate was injected intra-cerebrally in postnatal day 5 (P5) rat pups to mimic excitotoxic injury. Rats were sacrificed at P5+2 h, P5+4 h, P5+18 h. Lesion size and location of tight junction (TJ) proteins were determined by immunohistochemistry and BBB leakage after ibotenate injection by dextran staining. BBB proteins gene expression (TJ’s efflux transporters and detoxification enzymes) was determined on cortex and plexus. A group of pups was treated with melatonin (5 mg/kg, intraperitoneal).

Results Dextran extravasation was found 2 h after the insult, suggesting a rapid BBB breakdown that resolved at +4 h. A significant reduction in extravasation was observed in melatonin-treated pups. Molecular Biology, immunohistochemistry and electron microscopy showed a dynamic BBB modification during the first 4 h, partially reversed with melatonin. Lesion size evaluation confirmed melatonin white matter neuroprotection.

Conclusions Cardiac dysfunction in this vulnerable group of patients can be better identified with TDI compared to FS and promoting axonal regrowth.
LVO. Using TDI may improve the identification of cardiac dysfunction and guide further management.

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O-218 THE EFFECT OF CAFFEINE ON DIAPHRAGMATIC ACTIVITY IN PRETERM INFANTS

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Background Preterm infants born with a GA <32 weeks are at high risk of developing central apnea of prematurity (AOP). Treatment with caffeine reduces central AOP by stimulating the breathing centre. Animal studies suggest that caffeine improves contractility of the diaphragm. We have determined the effect of caffeine on diaphragmatic activity in preterm infants.

Methods Spontaneously breathing preterm infants <32 weeks treated with an intravenous loading dose (10 mg/kg) of caffeine base for central AOP were eligible for the study. Diaphragmatic activity was continuously measured by transcutaneous electromyography (dEMG) starting 30-min before (baseline) until 1-hour after caffeine administration. Diaphragmatic inspiratory activity per breath, expressed as the relative amplitude change of dEMG (logEMGAR), area under the curve (AUC), respiratory rate (RR), as well as tidal volume (Vt) measured by respiratory inductive plethysmography, were calculated at 4 fixed time points after caffeine administration (5, 15, 30 and 60-min) using the average of all breaths in a 30-sec recording and compared to baseline.

Results 30 preterm infants (mean GA 29.1 ± 1.3 wk; birth weight 1237 ± 370 g) were included. 5-min after caffeine administration, diaphragmatic activity significantly increased (median, IQR) compared to baseline; logEMGAR (0.13, 0.09–0.17), corresponding with an amplitude increase of 35% (22–49%). AUC (19%, 11–34%) and Vt (30%, 7–48) also increased significantly. Caffeine did not impact RR. The increased activity was observed at all subsequent time points.

Conclusions This is the first study showing that caffeine treatment, besides stimulating respiratory drive, results in a rapid (within 5-min) and sustained increase in diaphragmatic contractility in preterm infants.

Late Breaking O-219 DEVELOPING AND EVALUATING AN ON LINE PARENT INFORMATION AND SUPPORT APPLICATION TO FACILITATE HOME-BASED CARE BY PARENTS OF LONG-TERM CONDITIONS: A FEASIBILITY RCT

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Abstract O-219 Figure 1