with recurrent seizures during phenobarbital therapy, plasma concentrations were predicted to be < 20 mg/L at the moment of recurrent seizures. This supports a minimal effective concentration of about 20 mg/L.

Conclusion Also during hypothermia we advise an initial 20 mg/kg loading dose. However, clinicians should not be reluctant to administer an additional dose of 10–20 mg/kg, as we have shown that the blood levels were often below the therapeutic range (20–40 mg/L).

132 EARLY LIPID AND HIGH DOSE AMINO ACID ADMINISTRATION INCREASES ANABOLISM IN VLBW INFANTS

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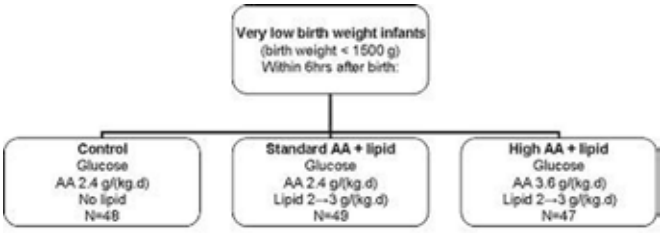
Introduction The beneficial effects of early nutrition in preterm infants are well known. Nonetheless, almost all VLBW infants (BW< 1500g) develop a protein and energy deficit in the first week of life. Consequently, protein balance is impaired. Lipids could aid in ameliorating the protein balance.

We hypothesized that early parenteral lipid and high dose amino acid (AA) administration from birth onwards to VLBW infants is safe and results in a higher protein balance.

Methods Inborn VLBW infants were randomized to one of three different parenteral nutritional regimens (Figure).



Abstract 132 Figure 1

Nitrogen (N) balances and urea rate of appearance ([urea]Ra, subgroup of infants) were measured at day two; biochemistry was recorded daily.

Results Table shows significant differences at day 2.

Abstract 132 Table 1

	Control group	Standard AA + lipid	High AA + lipid
Glucose (mmol/l)	5.5±2.5	7.0±2.9a	6.6±3.1
Urea (mmol/l)	10.0±4.3	8.3±2.5a	11.7±3.2b
Triglyceride (mmol/l)	0.8±0.5	2.0±1.6a	1.8±1.0a
N-balance (mg/kg/d)	93±111	181±111a	251±145a
[Urea]Ra (μmol/kg/h)*	361 (202–520)	413 (233–593)	721 (197–1245)ab

a: sign. different from control group (p<0.05); b: sign. different from Standard AA+lipid group (P<0.05); * median (IQR)
Biochemistry, N-balance and [urea]Ra at day 2

Blood gas, platelet count, electrolytes, and bilirubin were not significantly different between groups.

Conclusion Introduction of 2g lipids/(kg·d) and 3.6g AA/(kg·d) from birth onwards seems safe and results in a higher N-balance and thus increased anabolism in VLBW infants. Urea is more likely a marker of AA metabolism than of AA intolerance.

133 PHYSIOLOGY OF THE AIRWAY AND ITS CONTROL

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Nature, Key Functions, Neural Control & Clinical Impact.

The airway is a dynamic conduit, extending from the nose to the air sacs. Its key functions include protection, volume maintenance and ventilation, which are coordinated with other motor acts. Neural control of motor output provides airway defense as a first priority, with rapid protection of the lower airway being afforded by laryngeal closure and central apnea. During breathing, stability of airway volume (patency) and gas flow with ensuing gas exchange are also controlled centrally via coordination of motor activities that interact with physiochemical (structural) mechanisms. Sensors rapidly relay information about all key motor functions and, if required, this monitoring results in within-breath pattern adaptations. Neural control of the airway is not only dynamic but varied, with many motor output patterns noted during development and in different physiological (e.g. sleep) and pathological states. The clinician uses this knowledge to interpret breathing patterns as normal or abnormal, and uses this synthesis to direct both investigation of the airway and/or its central control and therapy.

Review aims This talk will describe

- nasal functions for protection and airway patency
- obstructive sleep apnea and the effects of CPAP therapy
- coordination of sucking, nutritive and non-nutritive swallowing in breathing
- laryngeal muscle functions in eupnea, sighs, grunting, incremental breathing and gasping
- lower airway patency and hysteresis matching of conducting and parenchymal airways
- central control of breathing and the impact of changes in breathing with behavioral state

134 OBSTRUCTIVE SLEEP APNEA, HYPERTONUS AND ADIPOSITAS

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Obstructive sleep apnea (OSA) is characterized by prolonged partial and/or intermittent complete (apnea) or partial (hypopnea) upper airway obstructions. The disruption of normal ventilation can be associated with hypoxemia and abnormal sleep patterns. OSA occurs predominantly during REM-sleep. Most affected children present with snoring and breathing problems during sleep.

The prevalence of OSA in children is approximately 4%. OSA can be associated with daytime sleepiness and cognitive/behavioral complications like poor school performance and hyperactivity. Cardiovascular complications include pulmonary hypertension, cor pulmonale, and systemic hypertension. There is a significant association between apnea-/hypopnea-index (AHI) and oxygen desaturation index with raised daytime and nocturnal blood pressure.

There is an increasing prevalence of obesity in children. Obesity can interfere with sleep in different ways. A lack of sleep is associated with an increased risk for obesity. On the other hand, obesity can have a negative influence on sleep. An increased soft tissue mass and altered mechanics lead to an increased airflow resistance, causing upper airway obstruction. With the current epidemic of obesity the incidence of OSA due to obesity in younger children may become remarkable. The risk for systemic hypertension caused by obesity is independent from the risk for hypertension caused by obstructive sleep apnea.