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PREDICTING FEEDING PROBLEMS IN INFANTS WITH CENTRAL GREY MATTER DAMAGE FOLLOWING HYPOXIC-ISCHAEMIC ENCEPHALOPATHY (HIE)

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Background and aims: Feeding problems are common following HIE and are a huge burden for infants and families. We aimed to evaluate, in term newborns with HIE and basal ganglia/thalamic (BGT) lesions, the relation between feeding difficulties and early MRI findings.

Methods: From 1993-2007, all term infants with evidence of perinatal asphyxia, HIE and BGT injury seen on MRI within 6 postnatal weeks were included. BGT, white matter, internal capsule, cortex and brainstem lesions were classified by severity. Cerebral palsy (CP) severity was determined from the Gross Motor Function Classification System (GMFCS).

Results: 175 infants fulfilled the entry criteria. 49 died (23 neonatally, 26 in infancy). Feeding problems occurred in all who died, both early and late, and in 65% of survivors; 44% had difficulty with liquids, 48% with solids and 27% only tolerated pureed foods. 33% had a gastrostomy (median age 11 (range 3-48) months). All but 3 of the 82 children with feeding problems had CP, 91% GMFCS level IV/V. In a logistic regression model BGT/mesencephalic injury was associated with feeding problems ($p < 0.001$) and BGT/pons injury with gastrostomy ($p < 0.001$). The need for gastrostomy occurred in only 3.5% of infants with mild/moderate BGT + normal pons but in 90.6% of surviving infants with severe BGT+ pons injury.

Conclusions: The occurrence and severity of feeding problems in neonates with HIE and BGT injury relates to the severity of the BGT/brainstem lesions. These findings enable planning of care and a better approach to nutrition in these children.

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DIETARY SUPPLEMENTATION WITH ARGINYL-GLUTAMINE (ARG-GLN) DIPEPTIDE OR DOCOSAHEXAENOIC ACID (DHA) MODULATE HYPEROXIA-INDUCED BRAIN INJURY IN NEONATAL MICE

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Aims: Using a model of hyperoxia-induced brain injury, we evaluated the potential for Arg-Gln or DHA to counteract the impact of hyperoxia in the developing brain.

Methods: 7-day old pups and their nursing dams were placed in 75% oxygen for five days. Pups were returned to room air on postnatal day 12 (P₁₂) and supplemented by oral gavage twice daily with Arg-Gln (5 g/kg/d), DHA (5 g/kg/d) or vehicle through P₁₇. A group of mice kept in room air served as normoxia controls. On P₁₇, pups were sacrificed and brains collected. Caspase-3 activity was used as a marker of apoptosis. Inflammatory cytokines IL-6 and KC were determined by ELISA. LDH activity served as an index of tissue injury.

Results: Arg-Gln reduced caspase-3 activity by 19% vs. hyperoxia ($p < 0.05$), while DHA was without effect. Exposure to hyperoxia increased LDH activity by 22% over normoxia controls. Importantly, the hyperoxia-induced increase in LDH activity was attenuated with Arg-Gln or DHA, by 22% and 36%, respectively ($p < 0.05$). Although there was a modest induction of IL-6 by hyperoxia, supplementation with Arg-Gln or DHA completely reversed this effect resulting in IL-6 levels that were 19% and 30%, respectively, lower than the hyperoxic-vehicle group ($p < 0.05$). DHA significantly reduced KC levels by 34% ($p < 0.05$) vs. hyperoxic-vehicle, while Arg-Gln had no effect.

Conclusion: These results demonstrate dietary supplementation with Arg-Gln or DHA modulate markers of hyperoxia-induced inflammation and tissue injury, suggesting these nutrients to be of particular importance to the developing brain exposed to supplemental oxygen.