transplants were from living-related donors. At the time of transplantation the mean height z score was -1.15 +/- 1.7 and BMI z score was 0.44 +/- 1.8. Eighteen months after transplantation, catch-up growth was seen in 40% of children, 30% had normal linear growth without any catch-up and 30% had slow growth rate after transplantation. Children with evidence of catch-up growth (growth velocity z score >0) had more growth retardation at the time of transplantation, and were receiving lower doses of prednisone at 1.5 years after transplantation. Younger infants (below 6 months) were most likely to demonstrate catch-up growth after transplantation. In summary, a large proportion of children have growth retardation at the time of liver transplantation. Serum albumin increased significantly after (59.8 +/- 5.2 g/L) vs before (34 +/- 11g/L) transplantation, and Alanine transferase (ALT) decreased significantly from (130 +/- 260U/L) to (30 +/- 15U/L). Poor growth after transplantation occurred more in those receiving higher doses of corticosteroid. This growth retardation is inversely correlated with age. Growth after transplantation is proportional to growth retardation at the time of transplantation and inversely correlated with age. Growth after transplantation occurred more in those receiving lower doses of prednisone.

Methods

Background Hypoxic-ischaemic neonatal encephalopathy is associated with high mortality and morbidity rates worldwide.

Aims To investigate brain haemodynamic, cytochrome-c-oxidase (CCO) and energy-resource changes during transient hypoxia-ischaemia (HI) and recovery using simultaneous broadband near-infrared spectroscopy (NIRS) and phosphorus (31P) magnetic resonance spectroscopy (MRS).

Methods Nine healthy piglets (aged <24 hr) were anaesthetised and physiologically monitored. Transient cerebral HI (duration 20 minutes) was induced by reducing the inspired oxygenation and reversibly inflating bilateral carotid artery occluders. Using 31P MRS we measured inorganic phosphate (Pi)/epp, phosphocreatine (PCr)/epp, and nucleotide triphosphate (NTP)/epp where epp=exchangeable phosphate pool. NIRS measured cerebral concentration changes of oxy-haemoglobin (HbO2) and deoxy-haemoglobin (HHb), and cytochrome-c-oxidase oxidation state changes (Δ[oxCCO]).

Results Simultaneous 31P-MRS and NIRS results are shown. HI rapidly reduced brain oxygenation as shown by changes in haemoglobin difference (Δ[Hbdiff]=Δ[HbO2]-Δ[HHb]) closely followed by a fall in Δ[oxCCO]. PCr/epp fell, and Pi/epp rose, quickly while NTP/epp was buffered initially and only declined when Δ[oxCCO] was significantly lowered.

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Discussion During transient HI, CCO becomes reduced due to oxygen depletion; adenosine triphosphate levels are initially preserved by the creatine kinase reaction leading to PCr decline whereas energy utilisation without oxidative phosphorylation leads to increased Pi. Complementary MRS and NIRS enable better understanding of the cerebral metabolic response to HI and can help evaluate early intervention therapies.