vasogenic edema as part of this secondary brain damage and as predictor of late neurodevelopmental outcome. Both, DWI and MRS provide not only a non-invasive insight into pathophysiological mechanisms of HI brain injury, they add significant improvement to the early diagnostic assessment of the asphyxiated neonate and to the prognostication of later outcome. This is especially important during the acute postnatal phase, when the child often is hardly amenable to clinical judgement.

MAGNETIC RESONANCE SPECTROSCOPY STUDIES OF EXPERIMENTAL PERINATAL ASPHYXIA
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31P MRS studies of infants who have suffered severe intrapartum cerebral hypoxia-ischaemia (HI) often exhibit normal energy metabolism for up to 24 hrs after resuscitation (1). Later, [adenosine triphosphate (ATP)]/[inorganic phosphate (Pi)] and eventually [adenosine triphosphate (ATP)], fall. This secondary energy failure (4) has not been modeled in the newborn piglet (2). Recent studies have used this model to elucidate whether: a) intravenous magnesium sulphate (3) or cerebral hypothermia (4,5) were cerebroprotective; and b) 1H MRS detected metabolic changes similar to those seen in newborn infants (6).

Spectra were acquired continuously from anesthetized Large-White piglets (< 24 hr old) at 7 T using a 2.5 cm surface coil above the parietal lobes (2,6). Transient HI was induced by reversible, bilateral, carotid-artery occlusion and reducing FiO2 to 0.12 until ATP was ~30% of baseline. Quantitation of primary-insult and SEF severity showed magnesium sulphate did not ameliorate SEF (3). However, hypothermia (35°C for 12 hr) commenced at the start of resuscitation reduced both the extent of SEF (4) and the expected delayed rise in lactate (5). Concomitant with SEF development, 1H MRS showed a rise in lactate and a slower decline in N-acetyl-aspartate peak-area ratios. Unlike [PCr]/[Pi], lactate peak-area ratios did not return to baseline during resuscitation.

The newborn-piglet model is useful for testing putative therapies. The clinical viability of cerebral hypothermia as a cerebroprotective is worth exploration. Cerebral lactate may prove important as a clinical marker.

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IN VIVO 1H MR SPECTROSCOPIC IMAGING AND DIFFUSION-WEIGHTED MRI IN EXPERIMENTAL HYDROCEPHALUS

In order to investigate the severity and progression of ventricular dilatation, the occurrence of cerebral edema, and the localization of ischemic metabolic changes in hydrocephalus, in vivo 1H MR spectroscopic imaging (MRSI) and diffusion weighted MRI (DW MRI) were applied in 9 hydrocephalic rats at 1, 2, 4 and 8 weeks after injection of kaolin in the cisterna magna. Adiabatic single voxel 1H MRS and MRSI pulse sequences and multislice DW spin-echo (4 b values) MRI experiments were performed on a 4.7 T magnet, while the rats were under anesthesia and mechanically ventilated. Parametric images of the apparent diffusion coefficient (ADC) revealed a varying degree of ventriculomegaly in all rats, with different patterns of ventricular dilatation in time. Extracellular white matter edema, characterized by an increased ADC near the corpus callosum, was only observed during the early stages of hydrocephalus, most extensively in cases of progressive ventriculomegaly. In gray matter regions of the cortex, caudate putamen and thalamus, ADC values were not changed compared to controls, suggesting that extracerebral edema in hydrocephalus is confined to white matter. In spectra from control rats and one rat with a rapidly declining ventricular volume, no lactate could be detected. All other spectra of hydrocephalic rat brain showed a lactate peak, both in the early and late stages of hydrocephalus. Excessive F2 prolongation of lactate was ruled out as a cause of the increased lactate resonance. In 2 cases of fatal hydrocephalus, lactate was abundantly observed throughout the whole brain. In all other hydrocephalic rats, at all time points after kaolin injection, lactate peaks were only detected in voxels containing cerebrospinal fluid, suggesting the accumulation of lactate in the ventricles, and/or an ongoing periventricular production of lactate as a consequence of cerebral ischemia in experimental hydrocephalus.

CEREBRAL LACTATE OF HYPOXIC FETAL LAMBS MEASURED BY 1H-NMR SPECTROSCOPY
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Introduction: Fetal hypoxia may result in neurological disability in postnatal life. Fetal monitoring has the purpose to prevent fetal hypoxia. It is not known when fetal cerebral metabolism starts to deteriorate under hypoxic conditions. For optimal assessment of the fetal condition, more information is needed about the relation between cerebral metabolism and hypoxia. Based on previous work, we have shown that cerebral metabolism starts to deteriorate under hypoxic conditions and that the relation between cerebral metabolism and hypoxia is different in the fetal lamb compared to the human fetus. In this study, the relation between decreased fetal cerebral lactate and hypoxia under controlled hypoxic conditions is investigated. Methods: Eighteen pregnant ewes of the Dutch Texel breed were operated between 120 and 128 days of gestation (term 147 days).

The fetal head was delivered by hysterotomy and fixed. The fixed head was then used for measurement of fetal cerebral lactate using a 1H-NMR spectroscopy. Measurements were performed at 1.5T. For localized NMR Spectroscopy a STEAM sequence was used (TR=2500 ms, TE=30 ms). The VOI was about 5cm3 located at the thalamus level extending into both hemispheres. A surface coil was located on the vertex of the fetal head to receive the NMR signal in 2.5 min blocks. After baseline measurements the fetal SaO2 was gradually reduced by lowering inspired oxygen of the ewe. Results. In all 5 fetal lambs cerebral NMR spectra showed signals of lactate, ATP, inositol, creatine and N-acetylaspartate (NAA). Lactate level was increased ten times as compared to normoxic control levels of about 0.5 mM/L. Similar results were obtained for the other fetal lambs. Conclusion: Fetal cerebral lactate accumulation appears to be related to the progression of fetal hypoxia. These findings help to understand when fetal cerebral metabolism starts to deteriorate under hypoxic conditions (Supported by Nelleco Puritan Bennett Inc.}