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NEUROLOGICAL ABNORMALITIES IN VERY PRETERM AND VERY LOW BIRTHWEIGHT CHILDREN AT 5 YEARS OF AGE: ASSOCIATIONS WITH OTHER PROBLEMS.

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From a national yearcohort (1983) of 1338 liveborn V Pt (<32 w) and/or VLBW (<1500 g) infants 966 (72%) were alive at 5 years of age. Standardized neurodevelopmental and sensory assessments were performed in 927 children (96%) during homevisits by 3 paediatricians. The impact of an impairment in any assessed area for the functioning of the child was expressed in terms of disabilities as advised by the WHO.
Only 5% (half of the total C(erebral) P(alsy) children) are motor disabled (D-CP), all CP-children, more than half of M(inor) N(eurological) D(ysfunction) children but only 16% of neurologically normal children have a gross motor retardation. The majority of D-CP's is multi-disabled, compared to one third of non-D-CP and MND children and 15% of neurologically normal children. Although motor disabilities are relatively rare, associated impairments in other areas often cause disabilities.

Neurological			Gross motor		Associated	
	n	(%)	n	(row %)	n	(row%)
Normal	630	(70)	99	(16)	92	(15)
MND	176	(20)	101	(57)	55	(31)
Non-D-CP	43	(5)	43	(100)	14	(33)
D-CP	48	(5)	48	(100)	33	(69)
Total	897**	(100)	291	(32)	194	(22)

** major congenital anomalies and incomplete neurodevelopmental assessment excluded.

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GM1 ganglioside given after hypoxia-ischaemia markedly protects the fetal sheep brain from subsequent injuries.

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The purpose of this study was to determine whether GM1 given to the fetus after a hypoxic-ischaemic episode can protect the fetal brain against subsequent insults. Chronically instrumented near-term fetal sheep were subjected to three 10-minute episodes of reversible cerebral ischaemia, repeated at 1 hour apart. Six were given 30 mg/kg of GM1 through the umbilical vein at the end of the first ischaemia for the next 2 hours followed by a continuous infusion of 30 mg/kg/day over 60 hours after ischaemia: these were compared with 7 vehicle-treated controls. The time course of electrocorticographic (ECoG) activity and cytotoxic oedema within the parasagittal cortex were determined with real-time spectral analysis and continuous impedance measurements respectively. GM1 improved recovery of primary oedema and markedly reduced histologic damage ($p < 0.001$) particularly in the striatum, hippocampus and cortex. At 72 hours after ischaemia, ECoG activity had returned to normal in the GM1-treated group but was still depressed ($p < 0.001$) in the control group. These results showed that GM1 treatment initiated immediately after a transient hypoxic-ischaemic episode stabilised membrane function and markedly improved neuronal outcome following subsequent insults suggesting its potential therapeutic value in situations of repeated hypoxia-ischaemia in the perinatal period.

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Changes in extracellular lactate and glucose during the development of parasagittal cortical infarction after hypoxic-ischaemic injury in fetal sheep.

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Objective: Lactic acid accumulation or glucose depletion may contribute to the development of cortical infarction after cerebral ischaemia. The aim of this study was to determine the changes of extracellular glucose and lactate levels within the parasagittal cortex following 30-minute of cerebral ischaemia.
Study Design: Eight chronically instrumented near-term fetal sheep (119-133 days) were studied. Microdialysis probes were implanted in the parasagittal cortex. The relative recoveries for lactate and glucose were 25±6% and 22±3% respectively. Electrocorticographic (ECoG) activity and cytotoxic oedema were measured by continuous spectral analysis and impedance techniques respectively.
Results: Brain lactate levels increased 3-fold (0.36±0.07 to 1.08±0.19 mM, $p < 0.05$) from 1-4h preceding the onset of ECoG epileptiform activity and secondary cytotoxic oedema (8-36h). The maximum lactate concentration was 5.8 mM during 8-16h. Glucose levels rose 2-fold (0.10±0.02 to 0.22±0.03 mM, $p < 0.05$) from 8-64h.
Conclusion: These results suggest a metabolic disturbance during the postischaemic phase. However, the maximum lactate concentration was less than the threshold thought to cause infarction (20 mM). Glucose depletion does not occur during the onset of epileptiform activity and secondary cytotoxic oedema. Thus, tissue availability of glucose and accumulation of lactate are probably not major pathogenetic factors during the development of parasagittal cortical infarction after hypoxic-ischaemic injury in fetal sheep.

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COGNITIVE IMPAIRMENTS AT 7 YEARS OF AGE AFTER PERINATAL ASPHYXIA. Vineta Ruth, Annukka Liikanen, Marit Korkman.

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Aim: In a prospective follow-up study of high-risk newborn infants, to assess if asphyxia causes impairment in cognitive functions at the age of 7 years.
Study subjects: Randomly selected control infants and testable (cerebral palsy [CP] excluded) survivors from 3 high-risk groups: 1) term infants with acute birth asphyxia (5-min Apgar ≤6 or umbilical arterial pH ≤7.05), 2) preterm infants with antenatal distress born to preeclamptic mothers (mean ± SD: 31.8 ± 2.6 gestational weeks [gw], birth weight 1460 ± 570 g), and 3) very low birth weight infants (VLBWI) delivered after uncomplicated pregnancy (29.1 ± 1.7 gw, 1160 ± 220 g).
Methods: blindly assessed IQ with WISC-r and neuropsychological performance with NEPSY (attention and confrontation naming tests) and VMI (copying design test).

Results:	Control (N=47)	Acute Asphyxia (N=43)	Preeclampsia (N=45)	VLBWI (N=49)
IQ	117 ± 13	114 ± 15	106 ± 13*	108 ± 14*
NEPSY	-1.7 ± 4.7	-2.5 ± 7.7	-5.6 ± 7.2*	-3.7 ± 6.5
VMI	-0.4 ± 0.9	-0.5 ± 1.2	-0.7 ± 1.0	-1.0 ± 1.1*

*p<0.05

Conclusion: Survivors without CP after severe birth asphyxia perform as well as controls, whereas children born preterm with or without prenatal hypoxia have lower IQ and a slight impairment in cognitive functions.

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VERY LOW BIRTHWEIGHT (VLBW) INFANTS (<1000 G) : INFLUENCE OF GESTATIONAL AGE (GA) AND INTRAUTERINE GROWTH RETARDATION ON THE INCIDENCE AND SEVERITY OF CEREBRAL ISCHEMIC-HAEMORRHAGIC LESIONS. A PROSPECTIVE STUDY. O. CLARIS, A. LAPILLONNE, D. MIGUET, B.L. SALLE. Department of Neonatology, Hôpital Edouard Herriot, LYON, FRANCE.

Between 1986 and 1991, 148 VLBW infants were prospectively scanned by cranial ultrasound (u/s) in order to determine the incidence of cerebral ischemic-haemorrhagic lesions and their relationship with mortality and neurodevelopmental outcome. The 1st scan was performed as soon as possible after birth, then u/s were repeated at days 2, 3, 5, 7 and then twice a month until discharge. AGA (99) and SGA (49) infants had similar BW (881±102 g vs 853±127 g) but different GA (26,8±1,3 wk vs 30,1±1,9 wk, p<0,001). 17 infants (14 AGA, 3 SGA) died <48 h of life with a normal u/s, and were excluded of this study. U/s were consistently normal in 46/85 AGA and 37/46 SGA infants (p<0,01). Incidence of grades I and II haemorrhages were similar in both groups, but that of grade III was higher in AGA (31%) than in SGA infants (6%, p<0,05), as well as that of parenchymal lesions (19% vs 4%, p<0,05). Abnormal u/s were found in 2/18 28-29 wk SGA infants and in 10/27 wk AGA infants (p=NS) and in 7/28 ≥ 30 wk SGA infants. In the AGA group, incidence of normal u/s decreased with increasing GA, but statistical significance was not found (55% at 24-25 wk, 47% at 26-27 wk and 37% at 28-29 w). In the AGA group, mortality was of 38%, 22/38 (58%) deaths being due to extensive cerebral lesions, versus 58% (p<0,01) and 2/9 (22%, p<0,05) respectively in the SGA group. 45/99 (45%) AGA infants survived with normal u/s vs 34/49 (69%) SGA infants (p<0,01). Conclusion : Incidence and severity of cerebral ischemic-haemorrhagic lesions are higher in AGA than in SGA VLBW infants, explaining the higher mortality rate in the first group. Nevertheless, no significant difference between the 2 groups was found at 28-29 wk GA.

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PLASMA (P) AND CEREBROSPINAL FLUID (CSF) INSULIN (I) CONCENTRATION IS ELEVATED IN PIGLETS WITH EXPERIMENTAL NEONATAL PNEUMOTHORAX (PTX)

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There is a lack of data on the level of I in P and CSF during neonatal cardiovascular collapse. Moreover, disturbances in glucose (G) homeostasis influences brain metabolism profoundly. Therefore, we have studied 10 newborn piglets (Group 1) during the course of PTX measuring I by a RIA method and G concentrations in P and CSF. Stages of the disease were baseline, critical phase 65.0 ± 3.4 min after the beginning of the induction of PTX (MABP 16.8 ± 0.5 mmHg; HR: 54.7 ± 1.4 min^{-1}, $pH_{a,i}$: 6.89 ± 0.04, $pO_{2a,i}$: 25.0 ± 2.0 mmHg), when animals were given 10 ml x b.w.kg^{-1} 4.2 %v.v $NaHCO_3$ *iv* infusion and recovery (samples were taken 0, 120 and 240 min after the beginning of PTX and in critical phase). Data were compared to results taken from sham operated animals without PTX (Group 2, n=9, sampling at 0, 60, 120 and 240 min); all values are means ± SEM), *p < 0.05 compared to values in Group 2

	PLASMA		CEREBROSPINAL FLUID	
	Group 1	Group 2	Group 1	Group 2
GLUCOSE (mmol x l^{-1})				
0 min	9.1 ± 0.9	8.4 ± 0.6	6.7 ± 0.7	6.0 ± 0.6
60 min / critical	10.7 ± 2.3	9.0 ± 0.9	5.0 ± 0.4	5.7 ± 1.0
120 min	7.7 ± 1.4	7.5 ± 1.3	6.5 ± 0.9	5.2 ± 1.1
240 min	4.2 ± 0.9*	6.7 ± 0.7	3.9 ± 1.0	4.2 ± 0.7
INSULIN (pmol x l^{-1})				
0 min	445 ± 135	330 ± 127	37 ± 10	37 ± 5
60 min / critical	658 ± 135*	282 ± 111	30 ± 3	35 ± 4
120 min	1358 ± 495*	132 ± 25	60 ± 13*	37 ± 7
240 min	534 ± 245*	173 ± 34	164 ± 52*	38 ± 5

Conclusion: A significant hypoglycemia develops with a concomitant elevation of insulin levels in P and CSF in piglets with experimental PTX.