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HYPOXEMIA AND REOXYGENATION WITH 21% OR 100% O_2 IN NEWBORN PIGS. MORPHOLOGICAL ASSESSMENT OF THE BRAIN AFTER 4 DAYS

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To evaluate the effect of resuscitation on brain damage hypoxemia (PaO_2 2.3-4.3 kPa) was induced in 17 newborn pigs (2-5 days) by ventilation with 8% O_2 in N_2. When systolic blood pressure reached 20 mmHg, the animals were randomly given either 21% O_2 or 100% O_2 for 20 min followed by 21% in both groups. After 4 days the brains were perfusion-fixed in deep anesthesia. Blinded pathological examination assessed the damage on a 0 to 3 scale. Controls (n=5) showed no damage. Number of animals with damage and the mean (SD) degree of damage (including undamaged animals) are given.

	White matter/Cortex		Cerebellum		Hippocampus CA1	
	Number	Degree	Number	Degree	Number	Degree
21% O_2 (n=8)	8	1.9 (0.8)	6	1.0 (0.8)	2	0.8 (1.4)
100% O_2 (n=9)	9	1.8 (0.7)	7	1.3 (1.1)	4	0.8 (1.1)

The CA1 damage was mainly anoxic, in the white matter/cortex it consisted mostly of small infarctions, while in the cerebellum the damage was a mixture. There were no statistically significant differences between the groups. We conclude that reoxygenation with room air gives no more brain damage than with 100 % oxygen.

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HYPOXEMIA AND REOXYGENATION WITH 21% OR 100% O_2 IN NEWBORN PIGS. CHANGES IN ARTERIAL BLOOD PRESSURE (ABP), BASE EXCESS (BE) AND PLASMA HYPOXANTHINE (HX)

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Reoxygenation after perinatal asphyxia may induce injury through oxygen free radicals. To test if room air is as effective as 100% O_2 in resuscitation, hypoxemia (PaO_2 2.3-4.3 kPa) was induced in 20 newborn pigs (2-5 days) by ventilation with 8% O_2 in N_2. When systolic ABP reached 20 mmHg, the animals were randomly given either 21% O_2 (group 1, n=9) or 100% O_2 (gr.2, n=11) for 20 min followed by 21% in both groups. Mean duration of hypoxemia was 93 and 99 min. Mean (SD) values are shown. Controls (n=5) were stable.

		Hypoxemia	Reoxygenation				
		Before	End of	5 min	15 min	30 min	60 min	180 min
ABP	1	52(6)	20(syst)	61(13)	60(12)	49(7)	42(5)	40(8)
	2	51(7)	20(syst)	63(10)	58(11)	48(10)	42(6)	40(5)
BE	1	2(4)	-29(5)	-31(5)	-28(5)	-24(5)	-17(7)	-3(4)
	2	2(3)	-27(4)	-31(5)	-28(4)	-22(4)	-15(4)	-2(3)
HX	1	31(6)	165(40)	152(38)	136(38)	125(41)	98(47)	45(25)
(µM)	2	28(8)	140(46)	131(42)	118(36)	105(43)	79(38)	41(15)

There were no statistically significant differences.
Conclusion: 21% O_2 is as efficient as 100% O_2 for normalizing ABP, BE and HX after sévere neonatal hypoxemia in pigs.

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BILATERAL PNEUMOTHORAX (PTX) RESULTS IN INCREASED LEVELS OF PURINE METABOLITES IN PLASMA (P), CEREBRO-SPINAL FLUID (CSF) AND VITREOUS HUMOR (VH) OF NEWBORN PIGLETS

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Hypoxanthine (HX), xanthine (X) and uric acid (U) levels were measured by HPLC technique in P, CSF and VH obtained from piglets during 4 hours of experimental PTX (Ref 1.). 6 sham operated animals served as controls (Group 1). The clinical and laboratory parameters were in normal range during the experiments. In Group 2 (n=10), 59.3±3.9 min after the induction of PTX, in the critical phase (MABP=17.9±0.4 mmHg, HR=54.1±1.6 min^{-1}, arterial pH=6.95±0.05, HCO_3^-=10.5±0.9 $mMxL^{-1}$, pCO_2=73±8 mmHg, pO_2=29±3 mmHg, venous pH=6.93±0.04, CSF pH=7.13±0.04), after sampling of P and CSF, the piglets were resuscitated and a recovery period were allowed to them. P and CSF samples were also collected in 0.,120. and 240. minutes; while VH in 240. minutes of the experiments. HX reached its maximal concentration in P in the critical phase (53.8±7.8 μMxL^{-1} vs. the initial 18.8±3.6 μMxL^{-1}, p<0.001), while in CSF it occurred only in the early reoxygenation period (120. min; 43.6±9.6 μMxL^{-1} vs. the initial 17.1±2.2 μMxL^{-1}, p<0.01). X levels in P and CSF, and U concentrations in P increased gradually in the course of PTX indicating a continuous formation of these metabolites. There were elevations in the concentrations of purine metabolites in VH compared with the Group 1 [HX levels were 74.4±22.4 μMxL^{-1} vs. 48.2±18.9 μMxL^{-1} (N.S.), X levels 57.4±17.9 μMxL^{-1} vs. 21.4±8.6 μMxL^{-1} (N.S.) and U levels 150.0±39.9 μMxL^{-1} vs. 55.6±11.3 μMxL^{-1} (p<0.01), respectively.] In conclusion, elevated levels of purine metabolites through the neonatal PTX indicating increased tissue damages due to the formation of free radicals during posthypoxic-reoxygenation period (Ref 2.). (All values are mean±SEM.)

Refs: 1.Temesvári P et al, Neurosci Lett 1990;113:163; 2.Saugstad OD, Pediatr Res 1988;23:143.

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RELATION BETWEEN CEREBRAL HAEMODYNAMICS AND OUTCOME IN BIRTH ASPHYXIATED NEWBORN INFANTS STUDIED BY NEAR INFRARED SPECTROSCOPY (NIRS).

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The purpose of this study was to measure cerebral blood flow (CBF), cerebral blood volume (CBV) and its response to changes in arterial carbon dioxide tension (CBVR) by NIRS in 21 term newborn infants who had suffered birth asphyxia. All had clinical and biochemical (base deficit ≥ 15 mmol/l) evidence of asphyxia and were studied during the first 24 hours. Eight of the 21 infants died and neurodevelopmental examination in survivors at one year of age showed that 7 had major neurological impairments; the remaining 6 infants were normal or had minor impairments. Results (mean ± SEM) were as follows:

	Dead	Major Imp.	Min Imp./Norm
CBF ($ml.100g^{-1}.min^{-1}$)	51.7±18.4 n=3	26.2±7.5 n=3	17.5±5.3 n=2
CBV ($ml.100g^{-1}$)	6.5±1.2 n=7	4.5±0.9 n=7	3.9±0.9 n=4
CBVR ($ml.100g^{-1}.kPa^{-1}$)	0.12±0.05 n=8	0.12±0.05 n=7	0.10±0.06 n=6

Values for CBF and CBV were significantly higher than previously defined normal values and for CBVR lower. The extent of the abnormalities was related to the severity of adverse outcome (ANOVA p<0.05).

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EARLY PROGNOSIS OF POST-ASPHYXIAL ENCEPHALOPATHY IN TERM NEONATES: A CHALLENGE TO NEONATOLOGISTS

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Nowadays, Intensive Care including hyperventilation makes difficult the clinical evaluation of asphyxiated term neonates. In order to precise the early prognosis of perinatal asphyxia, 35 fullterm neonates with moderate or severe post-asphyxial encephalopathy (Sarnat) were studied by means of ultrasonography (US), Doppler and electroencephalopathy (EEG). 18 infants died and the 17 survivors were prospectively followed-up. 4 developed a major handicap (cerebral palsy, seizures). Diffuse US echodensities, Resistance Index < 0.55, isoelectric, low voltage or paroxystic EEG's were considered as abnormal parameters in determing adverse outcome (death or severe handicap).

	Diffuse echodensities	Abnormal RI	Abnormal EEG
Sensitivity	91 %	86 %	95 %
Specificity	85 %	54 %	54 %
Pos.Pred.Value	91 %	76 %	76 %

The high sensitivities allow the clinician to have a high degree of confidence in sequential US, Doppler and EEG's in predicting bad outcome. The reasons for the differencies in specificities and positive predictive values will be discussed.

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99mTc HMPAO BRAIN SINGLE PHOTON EMISSION COMPUTED TOMOGRAPHY (SPECT) AND PERINATAL ASPHYXIA

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This study was designed to rate the clinical value of 99mTc HMPAO brain SPECT in perinatal asphyxia. 12 full-term babies were included. HMPAO SPECT were performed in 2 neonates respectively at 1 and 10 hours after the asphyxial event and repeated 24 hours after. In the 10 remainders HMPAO SPECT were initiated within 1 to 4 days following the asphyxial event. TcPO2, TcPCO2, StaO2, pH values, mean arterial blood pressure, blood glucose levels, pulsed Doppler recordings of middle cerebral artery and ultrasound scans were available at the moment of the SPECT procedures. In acute phase of brain asphyxia (1-10 hours) (2 cases), HMPAO SPECT demonstrated reduction of regional cerebral blood flow (rCBF) in the cerebrum and cerebellum whereas brain stem and basal ganglia were perfused and CBF velocities were significantly reduced. In the subacute phase (24-96 hours), 6 neonates demonstrated high CBF velocities and high rCBF on HMPAO SPECT (two of them belonging to the first group) with total vaso-paralysis. In one case, high rCBF were noted in parasagittal areas. This infant demonstrated cerebral parasagittal lesions on magnetic resonance imaging ; whereas the 4 remainders died. The 6 other neonates showed no abnormalities on rCBF neither on CBF velocities, and were neurologically normal at 6 months of age. These results indicate that 99mTc HMPAO brain SPECT shows a potential clinical value in evaluating perinatal asphyxia. It documents reliably changes in rCBF and demonstrates two distinct features following cerebral asphyxia respectively hypo and hypercerebral perfusion associated with bad outcome.