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EFFECT OF CO_2 CHANGES ON CEREBRAL OXYGENATION AND HEMODYNAMICS DURING ECMO IN PIGLETS

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Extracorporeal membrane oxygenation (ECMO) might alter the cerebral hemodynamics. Using near infrared spectrophotometry (NIRS) concentration changes of oxyhemoglobin (ΔcO_2Hb), deoxyhemoglobin ($\Delta cHHb$) and total hemoglobin ($\Delta ctHb$) in cerebral tissue were measured during induced hypercapnia and hypocapnia in 4 groups of 6 anesthetized, paralysed and mechanically ventilated piglets: Gr.1: normoxemic, Gr.2: hypoxemic, Gr.3: ECMO after normoxemia, Gr.4: ECMO after hypoxemia. ΔcO_2Hb and $\Delta cHHb$ reflect changes in cerebral O_2 supply, while $\Delta ctHb$ reflects changes in cerebral blood volume (CBV). Intracranial pressure (ICP) was also measured.

	$\Delta ctHb$	ΔcO_2Hb	$\Delta cHHb$	ΔICP
	-- µmol/100 g/kPa --			mmHg/kPa
Hypercapnia				
Gr.1	0.06±0.03*	0.10±0.06*	-0.05±0.04*	1.90±1.26*
Gr.2	0.04±0.03*	0.11±0.05*	-0.07±0.03*	1.91±0.85*
Gr.3	0.09±0.08*	0.14±0.08*	-0.06±0.04*	1.21±0.51*
Gr.4	0.11±0.08*	0.16±0.10*	-0.05±0.06	1.25±0.95*
Hypocapnia				
Gr.1	-0.08±0.08	-0.20±0.11*	0.12±0.06*	-0.64±1.07
Gr.2	-0.08±0.09	-0.20±0.21	0.12±0.14	-0.57±1.49
Gr.3	-0.11±0.07*	-0.21±0.10*	0.10±0.08*	0.06±0.98
Gr.4	-0.16±0.13*	-0.55±0.16*	0.39±0.06*	0.00±0.45

Values = mean±SD; * = $p < 0.05$

No significant differences between groups were found, with exception of ΔcO_2Hb and $\Delta cHHb$ in Gr.4 during hypocapnia.

Conclusion: The influences of CO_2 changes on cerebral oxygenation and hemodynamics are not altered during ECMO.

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ALTERATION OF CEREBRAL OXYGENATION AND HEMODYNAMICS CAUSED BY OPENING OF THE BYPASS BRIDGE DURING ECMO

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Repeated opening of the bypass bridge during extracorporeal membrane oxygenation (ECMO) is necessary to prevent clot formation. Using near infrared spectrophotometry concentration changes of oxyhemoglobin (ΔcO_2Hb), deoxyhemoglobin ($\Delta cHHb$) and total hemoglobin ($\Delta ctHb$) in cerebral tissue were measured during a 10 s opening of the bypass bridge during ECMO in 12 anesthetized piglets. ΔcO_2Hb and $\Delta cHHb$ reflect changes in cerebral O_2 supply, while $\Delta ctHb$ reflects changes in cerebral blood volume (CBV). Simultaneously heart rate (HR), arterial O_2 saturation (saO_2), mean arterial blood pressure (MABP), central venous pressure (CVP), intracranial pressure (ICP) and mean blood flow in left common carotid artery (CaBF) were also continuously measured. Most variables showed biphasic changes. The peak values are shown in table (*=p<0.05). HR and saO_2 were unchanged.

Values = mean±SD	1st peak	2nd peak
ΔMABP (mm Hg)	-41.09±13.14*	5.11±3.78*
ΔCVP (mm Hg)	3.65± 1.30*	-0.05±0.15
ΔICP (mm Hg)	-1.32± 1.32*	4.79±3.17*
ΔCaBF (mL/min)	-43.72±18.48*	8.93±5.55*
ΔcO_2Hb (µmol/100 g)	-0.73± 0.29*	0.18±0.12*
$\Delta cHHb$ (µmol/100 g)	0.53± 0.21*	-0.13±0.10*
$\Delta ctHb$ (µmol/100 g)	-0.29± 0.12*	0.17±0.07*

Conclusion: opening of the bypass bridge results initially in decreased CaBF, CBV and cerebral O_2 supply which is compensated by increased cerebral O_2 extraction and vasodilatation, resulting in secondary increased CBV and ICP. The increased CVP might be caused by retrograde bloodflow through the venous cannula. Repeated opening of the bypass bridge during ECMO might contribute to cerebral hemorrhage.

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RELATIONSHIP OF XANTHINE OXIDASE (XO) ACTIVITY TO IMMUNOREACTIVE ENZYME PROTEIN IN DEVELOPING HUMAN LIVER AND INTESTINE.

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XO is a major source of superoxide radicals and hydrogen peroxide, and has been ascribed a role in perinatal ischemia-reperfusion injury. We have previously shown that XO activity increases in the liver but decreases in the intestine during human fetal development. Since reversibly inactive forms as well as endogenous inhibitors of XO are known, our aim was to compare XO activities with immunoreactive XO protein levels in these two organs. XO was purified to apparent homogeneity from human milk. Antibodies to XO were raised in rabbits, purified by adsorption with human IgG and lactoferrin, and used in a sandwich-ELISA assay for XO protein (detection limit 10 ng/mg total protein). In fetal samples (10-40 weeks gestation), XO activities, measured with 14C-hypoxanthine as substrate, were linearly correlated with the XO protein concentrations in the same samples (r=0.74 for liver, r=0.91 for intestine). The slopes of the regression lines were not significantly different for liver and intestine. The constant specific activity (3µmol/min/mg XO protein) in both organs throughout gestation suggests that negligible amounts of inactive XO protein are present.

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PHARMACOKINETICS AND DYNAMICS OF RECTALLY ADMINISTERED SINGLE DOSE ACETAMINOPHEN (APAP) IN PRETERM INFANTS.

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Pain in infants is an important problem. Little is known about pharmacokinetics (PK) and pharmacodynamics (PD) of the analgesic APAP in preterm infants. PK and PD were studied in 15 preterm infants (28-32 weeks gestation, mean ±SD 30.3±1 wk, birthweight 1145±239 g) after a single dose (20mg/kg) of rectally administered APAP. Pain was assessed with a modified painscore. We measured peak plasma concentration(C_m), time to reach C_m (T_m), $T_{1/2}$, time to estimated therapeutic level(10-20 mg/l)(T_t), and excretion in urine (48 h). No changes were noted in heart and respiratory rate during the study. Data are presented as mean±SD or median+range.

C_m 11.9±3.7mg/l	T_m 240 min(90-480)	T_t 90 min(30-360)

Therapeutic levels were reached in 11 infants (73%) and remained high for more than 8 hours in individual infants. There appeared to be good analgesia even at levels below 10 mg/l. Due to long T_m, values for $T_{1/2}$ were not reliable.Over 80 % was excreted in urine as APAP-sulfate. We conclude that 1) APAP in preterms appears to be safe and effective, 2) a higher dose might be needed to shorten T_t interval, and 3) dose interval should be more than 8 hours in case of multiple doses.

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TRIGGER DELAY IN INFANT VENTILATORS

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With new technology it is possible to achieve patient triggered ventilation also in very preterm infants. To avoid accidental high transpulmonary pressures, the time between the start of spontaneous inspiration and ventilator insufflation (trigger delay time) has to be short. AIM: To determine the trigger delay time of three infant ventila-tors. METHODS: The Infant Star ventilator was equipped with an abdominal sensor and V.I.P.Bird and Babylog 8000 with flow sensors close to the proximal end of the endotracheal tube. To get a precise measurement of delay time we recorded phrenic nerve activity and airway pressure in anaesthetized cats and measured the time from the start of phrenic nerve activity to the increase in airway pressure caused by the ventilator.
RESULTS: The shortest delay times were found at the highest sensitivity and during SIMV mode of operation. Trigger delay times of 0.94 ±0.54 s (Infant Star), 0.13 ± 0.04 s (V.I.P.Bird & Babylog 8000) were found.
CONCLUSION: Ventilators equipped with flow sensors have much shorter response times than the ventilator which used an abdominal sensor to trigger inspiration.

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RELATIONSHIP BETWEEN CHROMATIC SENSE AND METABOLIC CONTROL IN DIABETIC CHILDREN: A LONGITUDINAL STUDY.

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Forty children affected by IDDM with HbA1c<9% have been enrolled. Inclusion criteria were: absence of fluoroangiographic signs of diabetic retinopathy, age>10 years, duration of disease>5 years, albumin excretion rate<20 mcg/min. At the beginning of the study all subjects underwent fluoroangio-graphy, monocular colour vision examination (Farnsworth 100 HUE) and glycated haemoglobin. After 4 years the same study protocol has been performed. During the follow-up 18 patients (group A) showed poor metabolic control (mean HbA1c 12.2%) and 22 patients (group B) had good metabolic control (mean HbA1c 8.1%). Colour vision examination as Total Error Score (TES) at the beginning of the study was 63.95(±40.17)in the group B and 68.60(±37.05) in the group A. After 4 years TES was 57.67(±34.56) in group B and 85.67(±42.95) in group A. In patients with HbA1c>9% we observed a significant worsening of chromatic sense(p=0.005) at the end of follow-up. Our study suggests that poor metabolic control can be a risk factor for the impairment of colour vision.