23 Effects of porcine surfactant (*Curosurf*) on cerebral oxygenation and haemodynamics in preterm infants.

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To investigate the cerebral effects of *Curosurf*, nine ventilated infants (5 boys, 4 girls) were studied by near infrared spectroscopy. Their median gestational age was 30 (range 26-34) weeks and they were 5.5 (2.5-13.5) hours old.

Continuous recordings of cerebral oxyhaemoglobin ([HbO_2]) and total haemoglobin ([tHb]) from 5 minutes before to 10 minutes after administation of *Curosurf* showed that both variables fell and then rose above baseline in all infants. The median maximum fall in [HbO_2] was 5.06 (range 1.67-8.08) $\mu mol.l^{-1}$ and in [tHb] was 1.29 (0.53-2.90) $\mu mol.l^{-1}$. The calculated fall in cerebral blood volume (CBV) was 0.08 (0.03-0.17) $ml.100g^{-1}$. [HbO_2], [tHb] and CBV subsequently rose above the initial baseline values by 1.2 (0.35-11) $\mu mol.l^{-1}$, 2.45 (0.29-6.6) $\mu mol.l^{-1}$ and 0.13 (0.02-0.38) $ml.100g^{-1}$ respectively. Measurements of cerebral blood flow (CBF) and oxygen delivery (COD) made in five infants between 12-70 minutes before and 19-80 minutes after *Curosurf* showed that mean CBF was 19 (12-37) $ml.100g^{-1}.min^{-1}$ before and 26 (10-29) $ml.100g^{-1}.min^{-1}$ afterwards; mean COD was 2.2 (1.9-4.2) $ml.100g^{-1}.min^{-1}$ before and 3.4 (1.6-3.7) $ml.100g^{-1}.min^{-1}$ afterwards. Multiple linear regression analysis showed that CBF and COD were significantly related to arterial carbon dioxide tension and that surfactant administration had no independent effect. Surfactant was thus associated with only small and transient changes in cerebral oxygenation and haemodynamics.

24 EFFECT OF SURFACTANT ADMINISTRATION ON CEREBRAL CIRCULATION

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Cerebral blood flow velocities (CBFV) were measured by pulsed Doppler in 16 infants, 26 to 29 weeks gestation, involved in the Exosurf European Trial whose end point was survival without brain lesions. They were randomized to a blinded dose of air or Exosurf, if intubated within the first 2h, with a 2nd dose 18h later. If a respiratory distress syndrom occured, they received 2 rescue doses of Exosurf 12h apart. The area under the curve (V_{mean}), the Resistance Index ($RI=(V_{syst}-V_{diast})/V_{syst}$), and the Pulsatility Index ($PI=(V_{syst}-V_{diast})/V_{mean}$) were recorded at the siphon of the internal carotid artery 15-30 min before and after each blinded dose, or unblinded Exosurf. Ventilator settings, blood pressure and gases were not different. RESULTS (Mean±Std):

		V_{mean}(cm/s)	RI	PI
AIR	Before:	12.45±4.45	0.83±0.15	2.36±1.02
(n= 6)	After:	11.83±3.84	0.86±0.13	2.36±0.82
EXO	Before:	13.57±3.82	0.85±0.12	2.35±0.77
(n=21)	After:	13.26±3.01	0.87±0.10	2.31±0.72

We conclude that Exosurf administration does not modify CBFV within the conditions of the protocol.

25 EFFECT OF UTERINE CONTRACTIONS ON HUMAN FETAL CEREBRAL OXYGENATION AND HAEMOGLOBIN CONCENTRATION MEASURED BY NEAR INFRARED SPECTROSCOPY (NIRS).

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The purpose of this study was to measure by NIRS changes in cerebral oxyhaemoglobin [HbO_2] and deoxyhaemoglobin [Hb] concentrations during labour. Two optical fibres were placed via the cervix 3cm apart on the scalp of 8 term infants. Uterine contractions were divided into those with and without fetal heart rate (FHR) decelerations (a fall in FHR of $\geq$ 15 $beats.min^{-1}$ for $\geq$ 15 secs). In 6 fetuses contractions without decelerations (n=44) were associated with falls in both [HbO_2] and [Hb], by 0.50 ± SD 0.27 $\mu mol.100g^{-1}$ and 0.78 ± 0.60 $\mu mol.100g^{-1}$ respectively. In 5 of these 6 fetuses FHR decelerations were seen (n=20) and were also accompanied by falls in [HbO_2], of 0.87 ± 0.54 $\mu mol.100g^{-1}$, but [Hb] rose by 1.10 ± 0.60 $\mu mol.100g^{-1}$ ($p<0.01$ vs contractions without decelerations, ANOVA). In the remaining 2 fetuses both [HbO_2] and [Hb] rose during contractions (n=18), by 0.71 ± 0.43 $\mu mol.100g^{-1}$ and 1.22 ± 0.55 $\mu mol.100g^{-1}$ respectively: no FHR decelerations were seen. We conclude that: (1) NIRS allows non-invasive monitoring of cerebral oxygenation and haemoglobin concentration during labour; (2) In most infants studied normal uterine contractions were associated with falls in cerebral [HbO_2] and [Hb]; (3) Contractions with FHR decelerations were associated with desaturation of cerebral haemoglobin.

26 CEREBRAL BLOOD FLOW DURING EXPERIMENTAL HYPOXEMIA WITH AND WITHOUT HYPERVOLEMIA IN THE NEWBORN PIGLET.

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The effect of hypoxemia (HO, 10% O2) with and without hypervolemia (HV, blood transfusion 20% 20 min (20′) after HO) on cerebral blood flow (CBF) was studied in 22 newborn piglets with the microsphere method. CBF was measured in brainstem (BS), cerebellum (CBL) and cerebrum (CBR) at baseline (BL), during HO or at baseline 2 (BL2), 20 min after HO (20′) or after HV, and 30 (30′) and 60 min (60′) after HO. Results were (ml/100g/min, mean ± SD.):

		BL	HO	HV/20′	30′	60′
HO	BS	67±15	198±108*	78±19	66±15	77±47
	CBL	73±16	117±71	84±19	74±23	76±34
	CBR	73±16	103±62	81±23	75±23	73±28
HV	BS	71±18	82±23	76±20	62±16	63±19
	CBL	80±24	90±37	81±19	65±18	69±16
	CBR	69±18	76±18	73±17	63±16	68±16
HO+	BS	71±19	252±117#	98±71	58±21	57±29
HV	CBL	88±34	158±104	95±39	63±12	58±20
	CBR	64±16	131±87	91±54	56±9	53±20

* $p<0.05$ from BL, # $p<0.08$ from BL.

HO does not influence the ability to regulate the circulatory response to a volume load.

27 REGIONAL CEREBRAL BLOOD FLOW RESPONSES TO HYPOXIA IN VERY IMMATURE LAMBS.

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As immaturity increases the vulnerability to hypoxic brain injury, this study tested regional cerebral responses of very immature fetal lambs of ≤0.7 gestation, giving emphasis to those brain regions susceptible to damage in the preterm infant. Regional cerebral flow (CBF, $ml.100g^{-1}.min^{-1}$) and oxygen delivery (COD, $mM.100g^{-1}.min^{-1}$) were measured (n=20) in eight lambs using radioactive microspheres. Cerebral regions examined were: cerebral hemispheres, cortex, white matter, hypothalamus, caudate nucleus, choroid plexus, cerebellum, midbrain and pons-medulla. Arterial oxygen content (CaO2) was varied from 1.41 to 4.03 mM. CBF increased at low levels of CaO2 in all cerebral regions with the exception of the choroid plexus. The increase in flow was sufficient to maintain COD only to the pons-medulla, midbrain, hypothalamus, white matter and caudate nucleus. Thus, COD to cerebral hemispheres, cortex, cerebellum and choroid plexus fell with decreasing CaO2. We conclude that in the very immature brain: 1) there are substantial regional differences in the response to hypoxemia; 2) low and absent responsiveness in specific regions may contribute to vulnerability to hypoxic injury before and after birth.

28 EFFECTS OF PHENOBARBITAL ON CEREBRAL HEMODYNAMICS IN PRETERM NEONATES.

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We evaluated the effect of phenobarbital (pb) versus placebo (saline) on cerebral hemodynamics in eleven, clinically stable preterm neonates. The mean (+SD) post natal age at study entry was 12 (3) hours. The mean (+SD) birth weight and gestational age were 1286 (387 g) and 30.5 (3) weeks respectively. We measured cerebral blood flow velocity (CBFV), using a pulsed wave Doppler monitor. Every subject was his own control. Placebo was systematically injected prior to pb (20 mg.kg -1 over 20 min). Simultaneous recording of heart rate, arterial blood pressure, TcPCO2, TcPO2, SaO2 were made before, at the end of the injection, and at 15, 30, 60, 90, 120 min after the end of each administration of either placebo or pb. Results : pb was in anticonvulsant concentrations (16 to 24 µg/ml). No significant changes ocurred in systemic hemodynamics, or in blood gases. CBFV remains stable and no different during placebo or phenobarbital administration.
We conclude that a pb loading dose of 20 mg -1, has no effect on systemic and cerebral hemodynamics during steady state. The cerebral protective action of phenobarbital may not result by decreasing baseline cerebral blood flow as previously suggested.