23 THE EFFECT OF THE MOTHER'S VOICE ON THE PHYSICAL ACTIVITY (A) AND $tcPo_2$ OF VERY PREMATURE INFANTS

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Nine infants of 26-29 w PMGA (BW 780-1270 g) were exposed to the mother's voice from a tape recorder via loudspeaker (at ~70 dB) 5x30' a day between 29 and 38 w PMGA (for $\bar{X}$ 8.2 w) after conditions were stabilized. At 2 days each week the babies' behaviour (applying an A rating from 1 = quiet sleep to 6 = crying) and $tcPo_2$ was recorded every 10" for 1 hour, once without and once with m's voice (30' after 30' "base line"). Results: A and $tcPo_2$ varied significantly during tests in 43-57 percent of "blanc" observations and with m's voice (table), but in the latter case significantly more often A level fell ($\Delta\bar{X}$ -.5 points; $p<.001$) and $tcPo_2$ level rose ($\Delta\bar{X}$ +.4 kPa; $p<.005$) during stimulation. Means of 15' periods of A and $tcPo_2$ correlated inversely ($r = -.55$). Conclusions: 1) Premature infants react to acoustic stimuli after 28 w PMGA, if hearing is unimpaired and/or stronger stimuli do not interfere. 2) M's voice can tranquillize the baby increasingly reproducible causing a transient increase of $tcPo_2$. 3) Recorded m's voice may serve as a substitute, if the mother cannot stay with her baby, to the benefit of both.

Changes (within 1 h) of (variance analysis)	Activity +	Ø	−	$tcPo_2$ +	Ø	−
no stimulation (n)	19	42	13	22	34	18
mother's voice (n) *	6	34	43	36	32	6

* X^2 test $p<.001$

24 PERINATAL HYPOXIA AND VISUAL FUNCTIONS IN VERY LOW BIRTH WEIGHT INFANTS AT 6 WEEKS OF (CORRECTED) AGE.

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A prospective study was started to determine the effects of perinatal hypoxic events on visual development in a group VLBW infants (n=113), born between 8/1/85 and 7/31/86. Binocular visual functions were assessed in 71 out of 93 survivors. Infants with RLF were excluded. In the hypoxic group (n=44) gest.age ranged from 24.8 to 35.0 wks (29.1±5.0; X±SD), bwt from 690 to 1495g (1120±260); in the non-hypoxic group (n=27) gest.age ranged from 28.6 to 34.3 wks (30.6±6.1), bwt from 700 to 1495 g (1145±300). Visual acuity, visual field size and optokinetic nystagmus were assessed at 8.4 to 23.0 wks of age (6.0 wks corrected age). Visual acuity with the acuity card method was significantly lower in the hypoxic group (23 min. of arc ± 0.84 octaves) than in the non-hypoxic group (17±0.47) ($p<0.05$, Student's t-test). Optokinetic nystagmus was asymmetric in 13 hypoxic infants and in 3 non-hypoxic infants ($p<0.05$, X^2-test). No difference was found in visual field size. The data indicate that perinatal hypoxic events are associated with a higher incidence of visual impairment in VLBW infants at 6 weeks of corrected age. Further studies are needed regarding the effects on visual functions at later age.

25 VISUAL ACUITY IN VERY LOW BIRTH WEIGHT INFANTS AT 6 WEEKS OF (CORRECTED) AGE.

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We studied the effect of early visual experience on the development of visual acuity (VA) in preterm infants. We assessed VA using the acuity card method in 63 very low birth weight (VLBW) infants, bwt 700-1495g (1150±240; X±SD), gest. age 25.8-35.0 wks (30.2±4.3). VA was assessed at 11.3-20.3 wks postnatal age (pa) (15.8±2.2), corresponding with 2.1-9.1 wks corrected age (ca) (6.0±1.3). Funduscopy was normal in all infants. The results were compared with those obtained in fullterm low risk infants at the age of 6.8 and 15.9 wks:

	Full term		VLBW	
age:	6.8wks (pa)	15.9wks (pa)	15.8wks (pa) ≙	6.0wks (ca)
VA :	17.7'±0.77 oct*	6.6±0.59	19.4±0.71	

*: min. of arc ± octaves (17.7 min. of arc ≙ 6/106 Snellen equiv.)
The results show that visual acuity assessed with the acuity card procedure is significant lower ($p<0.01$, Student's t-test) in VLBW infants in comparison with low risk infants of the same postnatal age, but is not different when age is corrected for prematurity. The present results fail to demonstrate an acceleration of acuity development by early visual stimulation.

26 CAFFEINE DOES NOT AFFECT CEREBRAL BLOOD FLOW (CBF) IN THE PRETERM INFANT

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Caffeine (C), used in treating idiopathic apnoe of the preterm newborn, has a presumed negative action on CBF, probably due to C-induced hyperpnea with a corresponding decrease of $PaCO_2$, which may contribute to ischemic events like periventricular leucomalacia (PVL). We studied changes in CBF in 25 preterm infants by determining CBF velocity (CBFV), using transcutaneous Doppler technique, before and 24 hours after start of C-medication. CBFV was quantitated by the pulsatility index and area under the velocity curve (cm/min) of the anterior cerebral arteries. $PaCO_2$, PaO_2, blood pH, heart- and respiratory rate, blood pressure and hematocrit were measured simultaneously with the Doppler investigations. In 18/25 infants C-levels were determined which were all in the therapeutic range (10-20) ug/ml). A significant decrease in number of apneic spells occurred ($p<.01$) and the $PaCO_2$ (mean (SD)) was lower after the start of C-medication (4.7(0.7) vs 5.3(0.8) kPa, $p<.01$). CBFV however did not alter during C-medication, in spite of lower $PaCO_2$ values, indicating no gross changes in CBF. It may be that the decrease of $PaCO_2$ after C-medication (d(SD): 0.6(0.7) kPa) was not large enough to alter CBFV. Moreover 21/25 infants had already normal $PaCO_2$ values ($PaCO_2$ <6.0 kPa) before the start of C; it is reported that in preterm infants only marked hypercarbia ($PaCO_2$ >6.7 kPa) and hypocarbia ($PaCO_2$ <2.0 kPa) cause significant changes of CBF from baseline values. It is therefore not likely that C, used in the therapeutic range, alters CBF and contributes to the pathogenesis of ischemic brain damage like PVL.

27 BRAIN DAMAGE AND RETINOPATHY OF PREMATURITY IN VERY PRETERM INFANTS.

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Hyperoxaemia has proved unsatisfactory as a total explanation for retinopathy of prematurity (ROP) in preterm infants. Recently, hypoxic-ischaemic injury to the retinal circulation has been proposed as a pre-requisite to oxygen toxicity in the causation of ROP. We have compared the neonatal ultrasound (US) brain scan findings and the neurodevelopmental status at 12 months of age in 25 very preterm (<33 weeks) infants with ophthalmological evidence of ROP, and 50 unaffected infants matched for gestation and postnatal age. 21 (84%) of the ROP-affected infants had abnormalities on US brain scans, including 11 (44%) with definite or presumed evidence of hypoxic-ischaemic damage or parenchymal haemorrhage (ventricular dilatation, hydrocephalus or cerebral atrophy), compared with 24 (48%) of the unaffected infants ($p<0.01$); and the neurodevelopmental status of 19 (76%) of the ROP-affected infants was abnormal at 12 months of age compared with 7 (14%) of the unaffected infants ($p<0.001$). We conclude that hypoxic-ischaemic injury is associated with ROP and may predispose to the development of ROP in very preterm infants.

28 FIRST RESULTS OF THE FINNISH-BAVARIAN MULTICENTER ARVO YLPPÖ FOLLOW-UP STUDY

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Medical and neurodevelopmental data were/are being collected prospectively in South Bavaria (62000 deliveries/year) and in Uusimaa/SF (15000/y) of all newborn infants over 1 year (1985/86) who needed special care (group I) in 19/6 units and of randomly selected not-transferred controls (group II). Total numbers: Group I: 6288/1409 *(hospital deaths 232/50; after discharge 16/0)*; group II: 742/623 *(0/0;3/1)*. The frequency distribution (%) of individuals with none (-), minor (s/n) or major (S/N) somatic disorders and/or neurologic abnormality after birth and at 5 months of age is shown in the table. Regional differences in group I-spectra are mainly due to different prevalence of prematurity and malformations. There was a close relationship between non-optimal obstetric and neonatal conditions, and both contribute to unfavorable outcome.

state	first week Bavaria I	Bavaria II	Uusimaa I	Uusimaa II	5 months (corrected) Bavaria I	Bavaria II	Uusimaa I	Uusimaa II
-/-	0.2	60.6	0.7	58.7	36.2	51.9	50.5	56.5
s/-	22.0	10.1	37.9	15.7	30.4	34.4	30.0	34.5
-/n	0	20.5	0.7	14.6	7.0	2.6	9.3	4.0
s/n	20.1	5.2	21.9	8.4	15.1	7.1	5.9	4.0
S/-	13.9	0	14.3	0.2	0.3	0.3	0.9	0.6
-/N	0	2.9	0	1.2	2.6	1.0	1.9	0.6
S/n	15.0	0.4	15.5	0.3	0.5	0	0	0
s/N	10.0	0.3	3.3	0.7	7.3	2.6	1.5	0
S/N	18.7	0	6.4	0.2	0.5	0	0	0

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