143 REGULATION OF CUTANEOUS BLOOD FLOW IN NEWBORN INFANTS.

GCM Beaufort-Krol, H Suichies, J Aarnoudse, A Okken. Div. Neonatology, Dept. Pediatrics, Dept. Obstetrics, University Hospital Groningen, Groningen, The Netherlands.

Reactive hyperaemia, which occurs after a short period of arterial occlusion is regulated by the autonomic nerve system. Studies of reactive hyperaemia in newborn infants reported so far are based on limb plethysmography, a method which does not discriminate between skin and muscle blood flow. To determine the regulation of cutaneous blood flow alone, we measured the reactive hyperaemia response using a diode laser doppler flowmeter.
Twentyfive infants with a birthweight ranging from 0.8 to 2.3 kilograms and a gestational age ranging from 28 to 40 weeks were studied.
Results: Cutaneous reactive hyperaemia responses could be elicited in all infants. A good correlation was found between preocclusional cutaneous blood flow and the maximal hyperaemia response. No correlation was found with age, postconceptional age, skin temperature, weight, haematocrit, and transcutaneous oxygen tension.
Conclusion: Autonomic regulation of cutaneous blood flow appears to be equally present in both premature and term newborn infants.

144 OPHTHALMIC ARTERY BLOOD FLOW VELOCITY IN NEONATES: RELATION TO CEREBRAL BLOOD FLOW AND CARDIAC OUTPUT.

Lindner W, Schaumberger M, Versmold H*, Department of Pediatrics and Div. Neonatology Dept Gyn Ob*, Univ. Munich F.R.G.

Ophthalmic blood flow has been discussed in relation to retinopathy of prematurity (ROP). We measured blood flow velocities (BFV) in both ophthalmic arteries (OA), median (MCA) and anterior cerebral artery (ACA), by pulsed Doppler in 18 normokapnic, normotensive neonates. OA's were scanned by sagittal imaging of the orbitae through the eye lids, MCA through the temporal bone, ACA through the fontanel. Cardiac output (CO) was derived from mean BFV in the ascending aorta and aortic root area. Results: right/left OA-BFV 0.99±0.1 (n=18) (mean±SD) *p<0.05

Gest.Age (GA, wks)	n	max Blood Flow Velocity (cm/sec kg) Ophth.Art.	Ant.Cer.Art.	Med.Cer.Art.	Card Output (ml/kg min)
26-36	10	23±11	24±5	32±8	247±21
37-41	8	* 10± 3	* 13±3	* 16±3	217±16

Correlations were significant for OA-BFV/kg vs GA (r=-0.84), vs CO (r=0.80), vs weight (r=-0.81), vs ACA-BFV/kg (r=0.47) and the ratio (OA-BFV/kg)/CO vs GA (r=-0.88). Serial measurements in single sick neonates (hypokapnia, polycythemia) showed low OA-BFV increasing with normoventilation (Pco2 24-40 torr) and after hemodilution (Hct 0.78-0.50; CO +12%, OA-BFV +50%). Conclusion: pathophysiological changes of ophthalmic BFV can be assessed by Doppler sonography, providing a possible tool for future ROP research.

145 AUTOREGULATION OF CEREBRAL BLOOD FLOW (CBF) ESTIMATED BY TRANSFONTANEL DOPPLERVELOCIMETRY (TFDV) OF INTERNAL CAROTID ARTERY (ICA).

Jorch G, Jorch N, Boemelburg T. Department of Pediatrics, University of Muenster, FRG.

Lack of autoregulation of CBF has been shown in preterm neonates. It is an open question, wether this is related to low gestational age (GA) or to low absolute level of mean arterial blood pressure (MAP). We used mean blood flow velocity derived from the outline of the Doppler spectral curve (Vmax) of ICA by TFDV to estimate changes of CBF while MAP (oscillometry) changed due to infusion of plasma or katecholamines. TFDV was performed in 7 hypotensive and 4 normotensive infants < 31 wk, and in 6 hypotensive infants > 30 wk. Δ Vma/ΔMAP was calculated as a measure of autoregulatory dysfunction. Results (median, range, Mann-Whitney-Wilcoxon-test):

	hypot.< 31wk	hypot.> 30wk	normot.< 31wk
GA (wk)	27 (25-30)	36 (31-39)	26 (25-27)
MAP (mmHg)	25 (22-29)	31 (21-36)	39 (36-41)
$\frac{\Delta Vmax}{\Delta MAP}\left(\frac{\%}{mmHg}\right)$	2,6 (1,2-6,4)	0,3 (0,0-1,9)	3,5 (1,9-5,0)
	p<0,025	p<0,025	

Conclusion: Autoregulation of CBF as derived from TFDV of ICA is poor < 31 wk, both at hypotension and normal MAP, but active > 30 wk even at mild hypotension.

146 OPTICAL EVOKED RESPONSES (OER) AND PHOTIC DRIVING (PD) IN VERY SICK PREMATURE INFANTS < 33 WK OF GESTATIONAL AGE (GA).

Jorch G, Schneider G. Department of Pediatrics, University of Muenster, FRG.

ANDERSON & TORRES (Electroenceph. Clin. Neurophysiol. 58:1984) demonstrated OER and PD within normal electroencephalographic (EEG) tracings of healthy very low birth weight infants (VLBW). We investigated this phenomenon in 11 very sick VLBW (BW 600-1200g, GA 26-32wk, age 1-33d) with cerebral complications (hemorrhage, edema, leucamalacia), to see whether the incidence of OER and PD was different in this group indicating cerebral dysfunction. We recorded 6 EEG-channels (Fp1-T3,T3-O1,T3-Cz,Fp2-T4,T4-O2, T4-Cz). Optical stimulation was performed by single flashes and repeated flashes of 1-30/sec lasting 20 sec. Results: OER could be elicited in 10 of 11, PD in 7 of 11 tracings. Thus incidence was similar to healthy VLBW. Conclusion: OER and PD can be elicited even in very sick VLBW. They do not indicate cerebral integrity.

147 Zapadlo,M., Pětová,J., Böswart,J.: Importance of the level of 2,3 DPG in umbilical cord blood for the diagnostics of fetal chronic hypoxia.

In 105 full-term infants the autors have examined the level of serum lactate and that of intraerythrocytic 2,3 DPG. The samples were taken from mixed umbilical cord blood immediately after delivery. In 37 infants whose mothers were at great risk of chronic intrauterine hypoxia, the level of 2,3 DPG was significantly higher than in 68 infants from physiological gravidity. The level of serum lactate was in both groups normal. In the group with acute fetal hypoxia and high level of lactate, there was no increase of intraerythrocytic 2,3 DPG. The 2,3 DPG seems to be a proper metabolite for the detection of chronic intrauterine hypoxia.

148 TRANSIENT HYPERINSULINISM IN ASPHYXIATED NEWBORN INFANTS.

Károly Schultz, Gyula Soltész. University Medical School, Department of Pediatrics, Pécs, Hungary.

Hypoglycemia (H) in birth asphyxiated infants is commonly attributed to glycogen depletion. We have observed 3 term AGA infants with birth asphyxia, who developed hyperinsulinemic hypoglycemia (HH) in the early postnatal life. All had inappropriatelly high serum insulin concentrations for their blood glucose level, and needed glucose infusion rates of>8 mg/kg/min for several days to maintain normoglycemia. All infants recovered spontaneously.

Patients	1	2	3
Gestational age (wk)	39	38	40
Birthweight (g)	2950	3370	3800
Apgar score at 1 min, 5 min	6,7	6,7	5,6
Age at onset of hypoglycemia (hr)	10	4	25
Blood glucose (mmol/l)	0,7	0,8	1,5
Plasma insulin (mU/l)	17	45	101
Maximum glucose inf.rate (mg/kg/min)	16	13	9
Duration of hyperinsulinism (day)	21	10	7

It is important to consider HH in asphyxiated newborns and vigorous treatment of H is needed to prevent CNS damage.