77 NEURODEVELOPMENTAL OUTCOME IN PRETERM INFANTS WITH CEREBRAL INTRAVENTRICULAR HEMORRHAGE.

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A real-time ultrasound scanner was used to examine the brains of 137 infants born at less than 33 weeks of gestation who were admitted to the neonatal unit of Paediatric Department of Padua's University. The overall incidence of intraventricular hemorrhage (IVH) was 32.8%. 98 infants survived, 80 were repeatedly assessed until they had reached a corrected age of 24 months. The Brunet-Lezine test was used and a neurodevelopmental assessment for early diagnosis of neurological lesions. 85% of infants with normal scans or IVH grade I (according to Papile) were normal at follow-up and 10 had cerebral palsy (mild in three cases); 75% of infants with IVH III were normal and all the 6 infants with IVH IV had cerebral palsy. The Brunet-Lezine Developmental Quotient (DQ) was within the normal range in 70% of infants with normal scans or IVH I-II, while only 50% in case of IVH III and at 16.7% in IVH IV were normal. All cases with periventricular leucomalacia or porencephaly had developmental or neuromotor impairment. This suggests, according with most literature, that infants with milder degrees of IVH differ little from preterm infants without hemorrhage; survivors with more severe grades of IVH or parenchimal lesions have poorer neurodevelopmental outcome.

78 FACTORS INFLUENCING CEREBRAL BLOOD FLOW (CBF) IN PREMATURE BABIES DURING THE FIRST WEEK AFTER BIRTH.

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In order to evaluate the role of CBF in the pathogenesis of periventricular haemorrhage and leukomalacia, we measured CBF with the non-invasive Xenon-133 clearance method. Intravenous application of Xenon and registration of its clearance with Cadmium Telluride detectors was performed inside the incubator. Twenty premature babies (mean birth weight, ±SD, 1050 ±180g, gestational age 29 ±2 weeks) had one to three CBF measurements at a postnatal age of < 26h (CBF1), at 26–120h (CBF2), and at 120–206h (CBF3). The results show a mean CBF1 (ml/100g.min,±SD) of 12.4 (±1.5), CBF2 21.4 (±4.1), and CBF3 16.6 (±3.8) in babies with normal ultrasound (US). A similar time course, with a peak CBF2, was found in those with abnormal US. Interestingly, all these values were highest in those with intraparenchymal (±intraventricular) US anomalies: CBF1 19.1 (±6.9), CBF2 23.5 (±3.5), CBF3 21.5 (±7.3). This flow elevation might represent hyperaemia similar to the luxury-perfusion syndrome described by Lassen N.A. (Lancet ii: 1113-1115, 1966) in adults, and could be due to a loss of autoregulation.

79 ENERGY (E) AND NITROGEN (N) BALANCES IN VERY LOW BIRTH WEIGHT INFANTS (VLBI).

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23 E and N balances were performed in 12 VLBI. 4 were fed with a preterm formula (P F) and 8 with expressed breast milk (EBM). The two groups were comparable for birthweight (1133±164 vs 1016±235g), gestational age (28.5±1.7vs28.4±2.8 weeks), age and weight at beginning of the study (17±10vs31±29 days and 1258± 108vs1243±218g) (M±1SD). The E and N content of PF and EBM was 81vs 64±9kcal/dl and 329±8vs 242±46mgN/dl respectively. The two groups received similar milk intakes (187±17vs 184±15ml/kg/d). The PF group showed a higher E retention (117±17vs 97±21kcal/kg/d p=0.03), a higher N retention (377±62vs 297±64mgN/kg/d p=0.009) and a higher weight gain (17±6vs 14±6g/kg/d p=0.03). We found a positive linear relationship between N retention and E retention (r=0.68,p<0.001). The EBM was highly variable in E and N content so the ratio between them was not constant. Using multiple regression analysis we found no effect of E retention on the relationship between N intake and N retention in EBM group (15 balances). Conclusions: 1) VLBI behave in similar manner than low birth weight infants. 2) The most relevant factor affecting N retention is N intake. The E content of EBM and of modern PF is generally well retained by VLBI and it is probable that E is less often the limiting factor in growth than it was previously thought to be. Protein deficiency seems to be the most likely reason for poor growth in human milk-fed infants.

80 RESPONSES OF CEREBRAL VASCULATURE TO CHANGES IN ARTERIAL CARBON DIOXIDE TENSION MEASURED BY NEAR INFRARED SPECTROSCOPY IN NEWBORN INFANTS.

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Responses of the cerebral vasculature to changes in arterial carbon dioxide tension ($PaCO_2$) were studied by near infrared spectroscopy during the first week of life in 12 newborn infants, 11 of whom were receiving mechanical ventilation. 5 of the infants were thought to have normal brains, and 7 had various forms of cerebral injury. In the normal infants, increases in $PaCO_2$ of about 1-2kPa caused cerebral blood volume to increase by 0.5 to 1.8ml.100g^{-1}.kPa^{-1}: the size of the increase appeared to be gestation-dependent. Estimated cerebral blood flow increased correspondingly. In the 7 infants with cerebral injuries the responses to changes in $PaCO_2$ were usually less. In 3 term infants who had been severely birth-asphyxiated they were absent or grossly reduced and all 3 had elevated cerebral blood volumes. 3 of the 7 infants died and all 4 survivors had loss of brain tissue. We conclude that: 1) the sensitivity of the cerebral vasculature to $PaCO_2$ probably increases with gestation, 2) cerebral injury is associated with diminished or absent responses to $PaCO_2$.

81 Doxapram (DX) for apnea (A) of prematurity ; effects on pulmonary function. P. VERT, A. BAIRAM, M. FAULON, P. MONIN. INSERM U.272 UNIVERSITE NANCY FRANCE.

8 preterm infants GA ± SD, 30.5 ± 1.6 wks, BW 1.4 ± 0.2 kg, mean age 4.6 ± 2.6 d, presenting ≥ 3 idiopathic A/24h, ≥ 15s + bradycardia ≤ 100 bpm, received IV DX either 0.25 mg/kg/h (DX1) or 1 mg/kg/h (DX2) the 2nd day of treatment when A persisted. Respiratory and heart rates (RR, HR), TcPO2, TcPCO2, art-pressure were recorded. Occlusion pressure (P0.1) and respiratory function tests were evaluated before DX and during each dose regime (mean ± SD). None of the doses affected RR or HR, art-press. or TcPO2. No side effect was observed. A reappeared at a mean time of 81 ± 59 min after the end of DX, requiring caffeine P.O. These results confirm the efficacy of DX at low doses. The discrepancy between the effects on A and on respiratory function with DX1 suggests a peripheral action. Early recurrence of A at the end of DX may correspond to a very short half-life.

Criteria	Before DX	DX1	P <	DX2	P <
A/100 min	1.53 ± .55	.80 ± .54	.01	.38 ± .36	.001
PO.1 cmH2O	2.54 ± .46	3.00 ± .44	.05	3.49 ± .62	.001
VT ml/kg	6.25 ±1.17	5.96 ± .75	NS	7.85 ± 1.90	.02
V_T/T_I	15.37±1.96	16.09±1.65	NS	18.67±2.89	.01
VE ml/kg	417 ± 57	454 ± 56	NS	511 ± 77	.01
TcPCO2 torr	39.1 ± 3.3	38.7 ± 3.5	NS	36.0 ± 3.1	.02

82 IMMUNOHISTOCHEMICAL STUDY OF THE DISTRIBUTION OF EXOGENOUS SURFACTANT IN THE LUNGS OF SURFACTANT DEFICIENT RABBITS USING A MONOCLONAL ANTIBODY.

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To study the distribution of a natural porcine surfactant in the lungs of surfactant deficient rabbits a monoclonal antibody against surfactant apoprotein was prepared using hybridoma technology. This monoclonal antibody reacts specifically with the instilled porcine and not with the rabbit surfactant protein. In six surfactant deficient rabbits, natural porcine surfactant was instilled endotracheally, six other surfactant deficient rabbits did not receive surfactant (controls). The effects of surfactant instillation included the ability to re-establish spontaneous room air breathing. Porcine surfactant protein was found in both bronchi and alveoli, in upper, middle, and lower lung parts. There was, however, inhomogeneous focal distribution. It is concluded that 3-4 hours after instillation, porcine surfactant appears to be trapped in collapsed lung parts.