31 THE EFFECT OF ADDING LIPIDS TO THE INTRAVENOUS FEEDING OF NEWBORN INFANTS.

P. Sauer, J. Van Aerde, J. Smith, D. Wesson, P. Swyer, P. Pencharz, Depts. Paed., Med. Eng. & Surg., Univ. Toronto; Res. Inst., Hosp. Sick Children, Toronto, Canada. Sponsor HKA Visser.

Previously we showed a decrease in metabolic rate in infants receiving Total Parenteral Nutrition (TPN) containing glucose + lipids vs glucose only. In the present study we measured substrate utilisation by Indirect Calorimetry (IDC) over 5 hr and from the excretion of $^{13}CO_2$ during a primed constant infusion of U-^{13}C-glucose. Glucose oxidation (GO) was measured from the $^{13}CO_2$ enrichtment in breath at plateau and fat oxidation (FO) as the difference of non-protein metabolic rate and glucose oxidation. We compared metabolic rate (MR) and substrate utilisation in two groups of AGA infants on an isocaloric intake. Group I received glucose/aminoacids only, Group II received a lipid emulsion as well, fat intake 2.0±0.1 g/kg/d. Birthweight (2.5±0.2 vs 2.7±0.2 kg), gest. age (35.9±1.0 vs 36.9±1.0 wk), study weight (2.5±0.2 vs 2.7±0.2 kg) and postnatal age (18.7±2.4 vs 13.2±2.0 d) was not different between groups.

		Energy int.	MR	Gluc Int	GO	FO
	n	kcal/kg/d		g/kg/d		
Gr. I	13	82.7±2.7	51.9±1.6	18.2±0.6	9.6±0.6	0.96±0.1
Gr. II	15	86.3±1.6	46.0±1.0	13.8±0.3	6.4±0.3	1.77±0.1
		n.s.	p<.005	p<.001	p<.001	p<.001

Conclusions: 1. The replacement of glucose by fat results in a lower metabolic rate, presumably as a result of a decreased conversion of glucose into fat. 2. Fat intake is almost equal to fat oxidation at an intake of 2 g/kg/d. 3. AGA newborn infants oxidize fat efficiently. 4. TPN with lipids has advantages above glucose/amino acids only.

32 CATCH UP GROWTH AND CHILD ABUSE

J M King, L S Taitz (Introduced by R D G Milner)
Department of Paediatrics, University of Sheffield, The Children's Hospital, Sheffield, England

Poor growth in association with child abuse is well recognised but eventual outcome in terms of growth parameters has not been clearly defined. The changes in standard deviation scores (SDS) for height and weight were calculated in 95 children who had suffered child abuse. Catch up growth was defined as an increase in SDS >1.0. Ages at presentation ranged from 6 weeks to 12 years and length of follow up from 9 months to 8 years. The mean SDS for height and weight increased significantly between presentation and most recent clinic visit ($p < 0.001$ and $p < 0.01$ respectively). The 64 children who remained at home (group 1) showed a significant increase in height SDS only ($p < 0.01$). The 20 children taken into long term fostercare or adopted (group 2) showed significant increases in both height and weight SDS ($p < 0.001$ and $p < 0.01$ respectively). The remaining 11 children who were fostered for short periods only (group 3) showed little change in either index. Catch up growth in height occurred in 12% of group 1 compared to 55% group 2 ($p < 0.001$). Catch up growth in weight occurred in 22% group 1 compared to 50% group 2 ($p < 0.05$). Children suffering child abuse show greater catch up growth when taken into long term care.

33 DERANGED ENERGY METABOLISM IN THE BRAIN OF NEWBORN INFANTS WITH INCREASED CEREBRAL ECHODENSITIES.

PA Hamilton, PL Hope, EB Cady, ACM Chu, D Delpy, JS Wyatt, EOR Reynolds
Dept. of Paediatrics, University College London, London WC1E 6JJ, England.

Increased cerebral echodensities detected by ultrasound scanning of the brain in newborn infants often resolve, but sometimes progress to cystic periventricular leukoencephalopathy or other loss of brain tissue. The purpose of this investigation was to explore whether increased echodensities were associated with evidence of deranged cerebral energy metabolism as determined by phosphorus nuclear magnetic resonance spectroscopy (NMRS), and whether measurement of the energy status of cerebral tissue identified those infants who died or subsequently developed cysts or microcephaly.

15 normal infants born between 28 and 40 weeks of gestation were studied as controls at ages between 1 day and 14 weeks, using methods that have previously been described[1]. The phosphocreatine (PCr)/inorganic orthophosphate (P_i) ratio, which is an index of the energy state of the tissue, increased from 0.77 ± 0.14 (95% confidence limits) at 30 weeks of gestational age plus postnatal age to 1.12 ± 0.14 at 40 weeks.

20 infants, born at 29-41 weeks of gestation, with increased echodensities (not apparently due to haemorrhage, and associated with birth asphyxia in 9 infants) were studied by NMRS aged 1day-4weeks. PCr/P_i was below the normal range in all 5 infants who died. Sequential ultrasound scanning in the 15 survivors showed no evidence of loss of brain substance in the 5 infants whose PCr/P_i ratios were within the normal range, whereas cysts or microcephaly developed in 8 of the 10 infants whose ratios were abnormally low (Fisher's exact test, $p<0.01$).

We conclude that (1) cerebral energy status was abnormal in some infants with increased cerebral echodensities and (2), the infants with abnormal energy status were much more likely to die or show subsequent loss of brain substance.

1. Hope PL et al. Lancet 1984; ii: 336.

34 DECREASE OF PERIVENTRICULAR HEMORRHAGE WITHOUT PLANNED INTERVENTION.

A.G.S. Philip, L.R. Sawyer, W.C. Allan, Dept. of Pediatrics, Maine Medical Center, Portland, Maine.

Intraventricular/periventricular hemorrhage (IV-PVH) continues to be a major problem for preterm infants. Since the advent of ultrasound scanning, detection of IV-PVH has been made with some precision. We have routinely used an ATL sector scanner to detect IV-PVH in infants with gestational age ≤ 34 weeks since 1979. All scans have been interpreted by a single individual (WCA), providing a high degree of consistency. During the 5 year period Sept. 1979-Aug. 1984, we studied 655 infants ≤ 34 weeks gestation who survived for more than 24 hours. A progressive decline in the overall incidence of IV-PVH occurred, despite an increase in the proportion of infants ≤ 26 weeks gestation. Only 2% (6% of all IV-PVH) had evidence of parenchymal hemorrhage in 1981-84.

Year	Total ≤ 34w	IV-PVH ≤ 34w	IV-PVH <1500g	Gest. ≤ 26w	grade of IV-PVH (n) SEH*	IVH	IPH
1980	124	40%	46%	10%	10	34 (24%)	9
1981	149	32%	41%	6%	16	29 (19%)	2
1982	119	29%	33%	13%	11	22 (18%)	2
1983	135	24%	32%	13%	11	20 (15%)	2
1984	128	26%+	27%+	20%	14	16 (13%)+	3

*SE=sub-ependymal; IP=intraparenchymal; +=$p < 0.01$.

Using these data we have generated a grid to determine the risk of IV-PVH according to birth-weight-gestational age categories.

Reasons for the decline in IV-PVH are uncertain, but include a decrease in outborn admissions and changing attitudes of obstetricians towards extreme prematurity. The decline of IV-PVH without resorting to pharmacologic agents emphasizes the importance of controlled trials in evaluating intervention. *(See also Pediatr Res 19:1482-357a, 1985)*

35 PERIVENTRICULAR LEUKOMALACIA OF THE PRETERM INFANT AND NEURODEVELOPMENTAL OUTCOME AT 18 MONTHS.

C.Fawer, A.Calame, A.Anderegg, C.Guinard. (Introduced by J.Micheli) Dept.Paed.CHUV, Lausanne, Switzerland

Periventricular leukomalacia (PVL) can be accurately identified in the neonatal period by means of ultrasonography and the use of high-frequency transducer. In order to establish its pronostic value, a prospective neurological and developmental assessment (Griffith's development quotient [DQ]) at 18 months corrected for prematurity was performed on 82 infants of 34 weeks'gestation or less, allocated in 4 groups : 41 with normal scans (group I), 13 with isolated haemorrhage (group II), 4 with post-haemorrhagic ventricular dilatation (group III) and 24 with PVL associated or not with haemorrhage or dilatation (group IV). The outcome was good and similar in group I, II and III.

	GROUP I	GROUP II	GROUP III	GROUP IV
DQ (mean ± 1SD)	99.3 ± 4.8	101.8 ± 3.7	97.5 ± 5.6	87.7 ± 20.5
Major handicap	0/41	0/13	0/4	8/24

By contrast, the outcome of group IV was worse and seemed to depend on the extent and site of PVL: 1) Frontal areas (n=13): all babies developed normally. 2) Frontal-parietal areas (n=6): 3 had major handicap. 3) Frontal-parietal-Occipital areas (n=5): all had major handicap. Our study indicate that major sequelae are more closely related to the extent and localisation of PVL than to haemorrhage or ventricular dilatation as previously reported.

36 OUTCOME OF PRETERM INFANTS WITH NEONATAL APNOEIC ATTACKS.

Gillian Levitt, Alan Mushin, Susan Bellman and David Harvey. Queen Charlotte's Maternity Hospital, London United Kingdom.

The incidence of major disability associated with neonatal apnoeic attacks was studied. The infants had been monitored for apnoea by routine nursing observations and intermittent 24 hr pneumocardiograms. Thirty infants of less than 31 weeks gestation at birth were seen between 2 and 4 years of age. Full physical and ophthalmological examinations were carried out as well as audiometry and Griffiths developmental assessment. The infants were divided into three groups according to the severity of the apnoea: Group I, no apnoea (15 infants); Group II, moderate apnoea (attacks of 20-59 seconds duration) (8 infants); Group III, severe apnoea (10 episodes in 24 hrs, an attack longer than 60 seconds or those requiring ventilation) (17 infants). The results of physical examinations were all normal. No overt defect of visual acuity was found but further fundal examination is being carried out. There was one case of sensorineural deafness in Group I. Developmental quotients (DQ) of the infants in the three groups showed there were no statistically significant differences: Group I, median DQ96; Group II, median DQ 102; Group III, median DQ 91. The result of this study suggests that recurrent or severe apnoeic attacks do not imply a poor prognosis in infants with no other serious neonatal problems.