83 METABOLISM OF GLUCOSE IN LOW BIRTH WEIGHT NEWBORN INFANTS IN THE FIRST HOURS OF LIFE.

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Glucose metabolism was studied in low birth weight (LBW) infants before any oral feeding was given. We have measured glucose utilization using a stable isotope dilution technique and calculated glucose oxidation from oxygen consumption and carbondioxide production measured with open circuit indirect calorimetry. Birth weight of the infants ranged from 1.29-2.26 kg, gestational age from 32-39 weeks. Results are shown in the table.

Infant	age (hrs)	serum glucose (mmol/l)	glucose oxidation (mg/kg/min)	glucose utilization (mg/kg/min)
1	5	2.5	5.8	5.3
2	3	3.8	5.8	5.1
3	5	2.9	5.6	5.5
4	14	3.4	3.9	5.9
5	5	4.6	3.4	5.8
6	8	4.6	5.7	8.1
7	6	5.1	7.5	8.1

The discrepancies between glucose utilization and glucose oxidation suggest that in some LBW infants substrates other than glucose are oxidized, and in some infants not all utilized glucose is oxidized.

84 ENDOGENOUS GLUCOSE PRODUCTION IN SMALL FOR DATES NEWBORN INFANTS IN THE FIRST HOURS OF LIFE.

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Most studies on endogenous glucose production (GPR) in newborn infants, reported so far, have been carried out beyond the first day of life. Moreover, most infants had been fed orally. This study was performed to measure GPR in small for dates newborn infants in the first hours of life, using the prime dose-constant rate infusion technique with 6.6-dideuteroglucose. During the study the glucose levels remained within the normal range. Results:

Birthweight (gm)	Gest.Age (wk)	Postnatal Age(hours)	Glucose i.v. (mg/kg per min)	GPR
1780	36	5	3.3	2.0
2255	38	3	3.0	2.1
2170	39	5	3.1	2.4
1870	36.3	14	2.7	3.2
1835	37.6	5	3.2	2.7

In conclusion: Near term and term small for dates newborn infants do not seem to have a limited endogenous GPR.

85 ERYTHROBLASTOSIS AND FETAL GROWTH RETARDATION (IUGR):

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Little has been published about circulating nucleated red blood cells (NRBC) in very low birth weight (VLBW) infants. We saw marked erythroblastosis (increased NRBC) in several infants with severe IUGR who were also VLBW. Hence, we evaluated the NRBC of all VLBW infants admitted to our intensive care nursery in 1983-86. There were 361 infants with birthweight <1500 g admitted in the first 24 hours after birth, but 31 died before studies were obtained. Of the remainder 278 were appropriate (AGA) and 52 small for gestational age (SGA). NRBC were significantly* increased in SGA infants compared to AGA infants.

	NRBC per 100 WBC		Absolute NRBC ($\times 10^3/\ell$)		
	> 40	> 100	> 4.0	> 10.0	
AGA (n=278)	71(26%)	20(7%)	55(20%)	17(6%)	*$p < 0.001$
SGA (n=52)	35(67%)	20(38%)	25(48%)	15(29%)	

In AGA infants, marked increase in NRBC was associated with extreme prematurity and bacterial infection. Findings in SGA infants are consistent with recent observations demonstrating erythroblastosis in blood from fetuses with IUGR (using "cordocentesis"), in whom the severity of hypoxia was correlated with the increase in NRBC. Abnormally increased NRBC at birth in VLBW infants with IUGR may indicate chronic intra-uterine hypoxemia and predict infants at risk of neonatal complications (e.g. NEC).

86 THE EFFECT OF SEVERE HYPOGLYCAEMIA AND DIABETES ON THE EEG IN DIABETIC CHILDREN

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Insulin hypoglycaemia (H) is the commonest form of H in childhood and severe and recurrent H can cause brain damage. Serial EEG recordings were made in 70 diabetic children (DC) (age 11,2+0,5 yr, duration of diabetes (DD) 5,1+0,4 yr, mean+SE) and EEG findings were related to age (at EEG and at diagnosis), DD, daily insulin dose, long-term metabolic control assessed by HbA_1 and severe H episodes. EEG abnormalities (EEG-A) were found in 26 p.c. of DC. There was no relationship between EEG-A and DD, daily insulin dose or HbA_1. DC with EEG-A were younger (9,6+0,7 vs. 12,8+0,7 yr, $p < 0,01$), had an earlier onset of diabetes (4,8+0,6 vs. 7,6+0,6 yr) and 78 p.c. of them had severe antecedent H, whereas EEG-A were found in only 22 p.c. of DC with no H ($p < 0,001$). All DC with H and convulsions had permanent EEG-A. The degree of metabolic control has no effect on the EEG during the early years of D, but severe antecedent H, young age and early onset are important risk factors.

87 FATTY ACID OXIDATION IN ARTIFICIALLY FED INFANTS DURING CONTINUOUS INFUSION OF ^{13}C-TRIOCTANOIN AND ^{13}C-TRIOLEIN.

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Previous investigations of MCT in infants showed a high energetic utilization of the given substrate after single injection of ^{13}C-MCT. To what extent these patients can oxidize MCT during continuous application, especially in comparison to LCT, is still uncertain. Therefore we measured with the $^{13}CO_2$-breath test the oxidation of MCT and LCT in infants during continuous infusion of ^{13}C-triglycerides. 9 infants, fed with supplementary parenteral nutrition, were investigated. The carbohydrate intake was 7.4-19.7 g/kg·24h. For investigation the parenterally applied fat emulsion was substituted by an isocaloric amount of the testemulsion either enriched with ^{13}C-trioctanoin or ^{13}C-triolein. The ^{13}C-content, measured by mass-spectrometry, represents fatty acid oxidation. We found a higher MCT-oxidation compared with LCT: $\bar{x}$=35.6±15.0% vs. $\bar{x}$=18.6±8.3%. Both oxidation rates revealed a negative correlation to concomitant carbohydrate supply. The results show, that all patients can utilize parenterally applied MCT to a greater amount than LCT. MCT oxidation is also reduced by concomitant carbohydrate intake like LCT. On account of their high energetic utilization MCT in general are an adequate substrate for parenteral nutrition in infancy.

88 DETERMINANTS OF THE GROWTH OF BREAST-FED INFANTS IN NORTHERN THAILAND.

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Predictors of growth were examined cross-sectionally in a random sample of 65 infants under 1 year of age in a rural Thai community. Infants were studied for a continuous 48hr period in their own home; variables measured included: nutrient intake from breast milk (b.m.) and supplementary food (s.f.); morbidity (over the previous month); and total bacterial intake from s.f. and water.

Overall age, sex & birthweight accounted for most variance (87%) in attained weight. Total protein intake (g/24hr, summed for b.m. & s.f.) predicted current weight, accounting for a further 1.3% of the variance in weight ($p=.02$), while total calorie intake (kcal/24hr) showed no association ($p=.10$). Morbidity did not predict attained weight at any stage of analysis ($p=.61$), but total bacterial intake was inversely related to weight ($p=.01$).

These findings differ prior to 6 mos of age when either protein or calories from b.m. predict infant weight ($p=.004$). After 6 mos total calorie intake no longer predicts weight, and the association with protein is now predominantly due to s.f. intake. This change is related to the observed increase in s.f. at around 6 mos of age, with a concomitant reduction in the number of breast feeds.