265

DIETARY HABITS AND POLYUNSATURATED FATTY ACID (PUFA) STATUS IN SEVERELY OBESE VS NORMAL-WEIGHING CHILDREN

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Background: Pathologic obesity (classified according to the International Obesity Task Force, IOTF) has been associated with an early development of the metabolic syndrome. Within this context, an altered status of PUFA and their longer chain derivatives may play a role and has already been reported.

Methods: Fifteen obese (BMI 25.1–38.2, mean 30.6, SD 3.4) children (9 males, 6 females, mean age 10.9, SD 2.6, yrs) have been compared to 67 normal-weighting (BMI 12.4–17.5, mean 15.2, SD 1.2) children (39 males, 28 females, mean age 8.4, SD 0.9, yrs) as far as dietary habits (by means of a validated Food Frequency Questionnaire), macronutrient intakes (expressed as total energy %) and plasma fatty acid (FA) composition. The analysis of FA (expressed as FA%) was performed by means of capillary gas-chromatography after lipid extraction according to Folch. Statistics: parametric and non parametric tests, when appropriate.

Results: Compared to normal-weighting, obese children showed higher energy and lipid intakes, with lower carbohydrate intake ($0.03 > p < 0.004$). Within ingested lipids, obese showed higher levels of all the main FA families, including PUFA (5.4, SD 1.8, vs 4.3, SD 1.0, $p = 0.02$). Among plasma FA, obese children displayed lower levels of C22:6n-3 (1.1, SD 0.3, vs 1.7, SD 0.9, $p = 0.01$) and total n-3 long-chain PUFA (1.8, SD 0.5, vs 2.5, SD 1.4, $p = 0.04$) in spite of higher levels of the precursor C18:3n-3 (0.3, SD 0.1, vs 0.2, SD 0.1, $p = 0.001$). The C22:6n-3/C20:4n-6 ratio was lower in obese vs normal-weighting children ($p=0.001$). At a bivariate analysis inclusive of all the study subjects, C22:6n-3 was inversely related to BMI.

Conclusion: In this report on pathologic obesity in childhood, excess of BMI is related to lower levels of C22:6–3 in spite of higher levels of the circulating precursor, C18:3 n-3. This observation, coupled with the higher levels of PUFA intake in obese children, may suggest a metabolic derangement in the synthetic pathway of the n-3 series.

266

ACADEMIC ACHIEVEMENT IN 19 YEAR OLD ADOLESCENTS, BORN AS PRETERM INFANTS (GA<32 WEEKS AND/OR BW<1500 GRAM)

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Background: In view of the increasing survival rate of very preterm and very low birth weight infants, long term outcome of these infants born in 1980's is of interest not only in terms of handicaps and disabilities but also of educational achievement. Objective: The purpose of this study was to determine the highest level of completed or current education at age 19 in adolescents born as very preterm or very low birth weight.

Methods: In 1983 94% of the infants born alive in the Netherlands with a gestational age less than 32 weeks or a birth weight less than 1500 grams were included in the POPS-study (Project on Preterm and Small for gestational age infants). All 959 surviving adolescents (72% of original cohort) were invited for a full examination (including physical, mental, behavioral and educational development and social participation) in a hospital at the age of 19 years. If the adolescents refused to take part in the full assessment, they were invited to complete postal questionnaires only. Education was assessed using the same categories as the Dutch Central Bureau of Statistics (CBS) in the total population at the same age (reference category).

Results: There were 596 (62%) full-responders, 109 (11%) postal responders and 254 (26%) non-responders. In non-responders we found (based on previous assessments) significantly more adolescents of non-Dutch origin and of low educated mothers, and more severe handicaps. Of 705 responders, data on education were available in 688. Of POPS-adolescents, 17% had completed special education or lower vocational training (CBS: 6%) and 9% primary school only (CBS: 6%). Secondary education had been completed or was being attended by 60% (CBS: 81%) of which 21% (CBS: 29%) was currently at university.

Conclusions: Academic achievement in adolescents born very preterm or very low birth weight is significantly lower than that in the general population of the same age in the Netherlands. In view of probable positive selection bias in responders, the level of completed education in non-responders will be even lower. Social participation, future employment and independency may be difficult to achieve for many of these survivors of neonatal intensive care.

267

THE CLINICAL IMPORTANCE OF NEUTROPENIA IN HOSPITALIZED PEDIATRIC PATIENTS

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Background: Neutropenia is not a common finding in hospitalized pediatric patients. However, it causes a great concern to the treating physicians. The aim of this study was to evaluate the clinical characteristics of the neutropenic patients admitted in general pediatric ward and to identify the relationship of neutropenia with the hematologic parameters and the degree of inflammation.

Methods: The admitted patients in general pediatric ward during a period of twelve months and having at least one blood count from the same coulter analyzer were included in the study. The patients with chronic illnesses were not included. The clinical characteristics, the complete blood count and the sedimentation rate (SR) were recorded.

Results: 193/1547 (12.47%) patients were neutropenic. 16.5% of the neutropenic patients were leukopenic and 2.5% were thrombocytopenic. Studying the hematologic parameters of neutropenic and non neutropenic patients, the following mean values $\pm$ standard deviation, p value were reported: WBC 6.66 ± 3 vs. 10.8 ± 2.9 cells/mm³, $p < 0.001$, Absolute number of monocytes 480 ± 0.5 vs. 614 ± 0.5 cells/mm³, $p < 0.001$, Hemoglobin 10.9 ± 1.7 vs. 11.5 ± 1.6 gr/dl, $p < 0.001$, Platelet count $383 \pm 151 \times 10^3$ vs. $417 \pm 154 \times 10^3$ cells/mm³, $p = 0.005$. The neutropenic patients had statistical significant higher mean SR (33.2 ± 29 vs. 27.2 ± 23 mmHg, $p = 0.001$). Only 14.5% of the neutropenic patients and 18.6% of the non neutropenic ones had SR50 mmHg. The length of hospitalization was of statistical significant different between the two groups: neutropenic patients 6.7 ± 4 days vs. non neutropenic patients 6.3 ± 4.3 days, $p = 0.12$. The most common final diagnoses of the neutropenic patients were: 35.2% viral infection, 16.5% occult bacteremia, 10.3% urinary tract infection, 6.2% pneumonia, 6% non infectious etiology. None of the patients was diagnosed with malignancy.

Conclusion: 1/10 hospitalized patients are neutropenic with a small percentage of them being leukopenic or thrombocytopenic as well. Although the number of neutrophils has some relationship with the degree of inflammation, this isn't clinical significant, since only a low percentage of the neutropenic patients has SR50. The other hematologic parameters are lower in the neutropenic patients, but without any clinical significant effect. Neutropenia doesn't affect the course of the underlying illness since doesn't prolong the length of hospitalization. Malignancy is not a common cause of neutropenia. 3/4 of the neutropenic patients are diagnosed with infection.

268

DOES CUP FEEDING HELP IMPROVE BREAST FEEDING RATES IN PRETERM BABIES?

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Background: Many neonatal units in the United Kingdom strongly advocate cup feeding rather than bottle feeding of maternal expressed breast milk to preterm babies whose mothers wish to breast feed but who cannot be present for all 'breast feeds'. Cup feeding is believed to increase the chances of successful breast feeding. The lapping and sucking movements babies use at the cup are thought to be more similar to the tongue movements used for breast feeding than are the sucking motions used in bottle feeding. However there is no clear evidence base for this practice. We undertook a pilot randomised controlled trial to study the benefits, if any, of cup feeding preterm babies in this way.

Methods: A randomised controlled trial of cup feeding v bottle feeding as an adjunct to tube feeding of preterm babies < 35 weeks gestation whose mothers had declared a wish to breast feed. Primary outcome was breast feeding at discharge from the neonatal unit.

Results: The table shows that there were no significant differences between the groups for birth weight and gestation. Breast feeding rates were similar in the two groups, as was the age at which the nasogastric tube was withdrawn. These figures count 'breast feeding' as any feed at the breast on each day. There was an insignificant trend towards exclusive breast feeding in the cup fed group compared to the bottle fed group at discharge (10/27 v 4/27). Difficulties in recruitment prevented us reaching the group size (n=94) needed for more statistically valid results.

	Cup fed babies	Bottle fed babies
N	27	27
Median (range) birth weight	1560(580–2870)	1750(944–2980)
Median (range) gestation (wk)	31 (25–34)	32 (26–34)
Breast feeding at discharge	14	12
Breast feeding at term	11	12
Breast feeding at term plus 6 weeks	5	6
Median (range) post conceptual age at withdrawal of nasogastric tube (days)	250 (243–257)	
251 9241–2590		

Conclusion: This pilot study showed no benefit for cup feeding in either breast feeding rates at discharge or later, or in the post-conceptual age at which the naso-gastric tube was withdrawn.

269

SECONDARY ENERGY FAILURE IN A MODEL OF HYPOXIC ISCHAEMIC BRAIN INJURY ASSESSED BY SERIAL PHOSPHOROUS MAGNETIC RESONANCE SPECTROSCOPY, WATER APPARENT DIFFUSION AND ELECTROPHYSIOLOGY: A PILOT STUDY

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Background: The electroencephalogram (EEG) provides a sensitive means of predicting outcome early after perinatal hypoxia-ischaemia (HI). The amplitude integrated EEG (aEEG), a compressed, rectified and filtered form of 1-channel EEG, is increasingly used clinically to define infants who may benefit from neuroprotective intervention following perinatal HI. The precise relationship between electrophysiology and brain energetics remains unclear however. Phosphorous-31 magnetic resonance spectroscopy (31P MRS) and magnetic resonance imaging measurements of the apparent diffusion coefficient of water (ADC) provide useful information about local tissue energetics following HI. **Objective:** (i) To compare electro-cortical activity to cerebral energetics and ADC during the evolution of secondary energy failure (SEF); (ii) To assess the correlation between aEEG and standard EEG during HI and SEF.

Methods: A newborn piglet was studied under general anaesthesia (isoflurane & fentanyl) before, during and after acute HI (carotid occlusion and reduction of FiO₂ to 12% for 45min). The standard EEG (8 surface electrodes, 10–20 system) and aEEG were recorded for 30min before, immediately following and approximately every 4hrs after HI. Whole-brain 31P MRS spectra and ADC maps from the central axial slice were also obtained.

Results: Changes in the EEG are summarised in Table 1. The corresponding aEEG recordings are shown in Fig. 1. Abnormal burst (4hrs post-HI) and seizure activity (11hrs post-HI) were transformed into similar appearing traces on aEEG. The density of high voltage activity was highest during seizures. Energetic status assessed by phosphocreatine to inorganic phosphate ratio ([PCr]/[Pi]) and ATP to total exchangeable phosphate pool ([ATP]/[EPP]) is shown in Table 1. ADC was reduced as early as 4hrs after the insult, initially in the deep grey matter and watershed deep white matter, extending throughout the brain by 15hrs (Table 1 & Fig. 1).

Conclusion: Changes in electro-cortical activity, brain energy metabolism and water diffusion were observed during the evolution of SEF. The aEEG trace was sufficiently sensitive to capture globally abnormal cortical activity in this pilot study. We recorded abnormal burst activity many hours before PCr/Pi decreased. Future work aims to fully characterise the relationship between electrophysiology and brain energetics during SEF. Such information will facilitate the selection of infants who may benefit from neuroprotective strategies.

Time period	EEG Pattern	background activity (μV)	High voltage activity (μV)	[PCr] / [Pi]	[ATP] / [EPP]	ADC (% baseline)	
						Mean White Matter	Mean Grey Matter
Baseline	Discontinuous pattern	20–50	50–70	1.1	0.6	100	100
During HI (at nadir)	Severe suppression	<2.5	none	0.0	0.3	-	-
4 Hrs	Dysmorphic burst and suppression	10–17	80–150	1.0	0.6	80	92
11 Hrs	Seizure and suppression	<2.5	20–40	0.1	0.2	49	73
15 Hrs	Flat	<2.5	none	0.0	0.1	48	54

Table 1: Serial EEG, ³¹P MRS and ADC data collected at baseline, during and following resuscitation from acute HI.

Figure 1: Serial aEEG traces and ADC parameter maps collected at baseline and following resuscitation from acute HI.