1 G.PUTET*,G.VERELLEN*,T.HEIM & P. SWYER*.Dept. Pediat. Univ.Toronto and Res.Inst. The Hosp.for Sick Children, Toronto,Ont.Canada. Effect of calorie intake on the utilization of carbohydrate(C) protein(P) and fat(F) as energy substrate in the ill newborn infant on total parenteral nutrition(TPN). Energy (Kcal)input for infants may need to be restricted due to glucose(GL) or Intralipid(IL)intolerance caused by a variety of diseases. Using indirect calorimetry(IC) and urinary nitrogen output (UN) at thermoneutrality,energy needs from endogenous and exogenous sources were determined by studying 34 infants at TPN intakes(INT.) from 20-110 Kcal/Kg/day by GL alone,by GL plus amino acid(AA) or GL+AA+IL infusions. Resting metabolic rate(RMR)correlated positively with Kcal INT.(r=0.58 p<0.001).With GL+AA infusion the contribution of endogenous F to energy metabolism was inversely proportional to Kcal INT.(r=0.91 p<0.001)until an INT.of 70 Kcal/Kg/d.was provided from exogenous sources.Over an INT.of 70 Kcal/Kg/d. a large proportion of infused IL was oxidised(OXID.:60-70%)and the balance deposited.

INT.Kcal/Kg/d.	20-30	31-50	51-70	71-90	71-90	>90
No.of cases	4	4	8	7	8	3
P INT(g/Kd/d.	0	2.3±0.6	2.5±0.2	3.0±0.2	2.3±0.4	2.5±0.4
P OXID. "	0.4±0.2	0.7±0.5	0.8±0.3	0.9±0.5	0.5±0.3	0.6±0.2
C INT. "	6.4±1.6	8.5±1.5	12.7±1.2	16.7±1.2	13.1±1.5	14.5±0.7
C OXID. "	3.1±1.9	6.6±2.3	8.6±2.4	13.1±1.6	8.9±1.8	11.8±3.4
F INT. "	0	0	0	0	2.6±0.5	3.2±0.4
F OXID. "	2.9±1.1	1.6±0.7	1.3±0.3	0.1±0.2	1.8±0.6	1.8±0.7

Thus(a)neonates show metabolic flexibility to diverse substrates(b) the proportion of protein OXID.is independent of calorie INT. (c) fat emulsion is used preferentially as an energy substrate with less than 30-40% assimilation.(data are expressed as M±SD).

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NITROGEN BALANCE AND HORMONAL RESPONSE IN PREMATURE INFANTS UNDER PARENTERAL AMINO ACID/CARBOHYDRATE NUTRITION.

Negative nitrogen balance impairs prognosis of severe neonatal diseases especially RDS, in premature infants. We compared the effect of different 1 - amino - acid (AA)- carbohydrate mixtures on nitrogen balance, blood glucose (BG), plasma insulin (IRI), glucagon and human growth hormone (HGH). Three AA/carbohydrate mixtures were supplied each to 10 premature infants: I) AA 5 g/ sorbitol 5 g/ xylitol 5 g/100 ml, II) AA 5 g/100 ml, III) AA 5 g/ glucose 10 g/100 ml.

RESULTS: BG showes a mild elevation in group I, no change in II and a sharp rise up to 180 mg% after 60 min. in III. IRI corresponded well with continous levels between 10 and 20 uU/ml in I and II and a sharp rise up to 110 uU/ml in III 75 min. after starting infusion. Within 150 min. IRI returned to a basal level of 8 uU/ml. Glucagon was not affected in I and III. In II there was a peak of 273 pg/ml after 60 min. Plasma HGH was well stimulated in all groups with an average maximum of 43 ng/ml after 60 min., returning to evelated basal levels of 19 ng/ml after 150 min. Infants on 24 h-infusion with I showed a positive nitrogen balance within the first day.

CONCLUSION: AA-carbohydrate-infusions caused high endogenous HGH-levels in premature infants leading to positive nitrogen balance within the first 24 hours. Amino acids did not affect carbohydrate induced BG, insulin and glucagon reaction.

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Impaired fatty acid oxidation and increased gluconeogenic plasma substrates in SGA newborns with hypoglycemia - Improvement after injection of lipids.

The hypoglycemia frequently found in newborn small-for-gestational age (SGA) infants has been attributed to small glycogen depots, but recently also to impaired gluconeogenesis. SGA newborns given fat emulsion by injection show reduced capacity for lipolysis and slow elimination of FFA from plasma. We have studied substrates of lipid and carbohydrate metabolism in plasma before and after injection of fat emulsion (0.5 g of fat/kg b.w.). Initially, at 4 h of age, infants with hypoglycemia (glucose < 1.65 mmol/l) had sign. higher lactate and alanine than infants with normoglycemia. Positive correlations were found between the initial FFA and glucose concentrations and between FFA and betahydroxybutyrate concentrations, indicating low FFA oxidation in babies with low glucose. After fat injection, betahydroxybutyrate increased as a sign of fat oxidation. At the same time glucose increased and lactate and alanine fell in cases with hypoglycemia indicating improved gluconeogenesis. In babies given an equicaloric amount of breast-milk, glucose rose, if a certain level of betahydroxybutyrate was present in plasma as indicator of endogenous fat oxidation. The rate of gluconeogenesis seemd to be dependent on oxidation of fatty acids and on availability of substrates. These results are supported by recent studies on starved newborn rats (Ferré et al. Biochem Soc Trans 5:982,1977). Parental consent and Human Investigation Committee approval obtained.

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Bacterial Quality Control in Human Milk Banking.

We have analysed the bacteriological quality of milk donated to the Oxford milk bank: we have studied the effects on bacteriology of simple additional antisepsis during the collection procedure and of Holder pasteurisation in a purpose built human milk pasteuriser. 12 pools of human drip breast milk (each comprising 75-120 24 hour milk samples) were studied: 6 pools were collected into vessels washed in detergent (Group I): in the other 6 a hypochlorite sterilising agent was used (Group II). Potentially pathogenic organisms were grown in untreated milk from both group I & II pools and included E.coli, S.aureus and Group B B-haemolytic streptococci. 7 species of non-pathogenic organisms were identified from both group I & II pools. Pasteurisation eliminated all potential pathogens from both groups, but did not reliably remove any of the non-pathogenic species. The collection of milk in hypochlorite sterilised vessels (GroupII), resulted in a significantly lower bacterial count of both pathogenic and non-pathogenic species before pasteurisation; and significantly increased the chance of pasteurisation giving a sterile product. The potential effects of bacterial contamination on milk quality, and the production of toxic bacterial products in milk are discussed. We recommend post-pasteurisation monitoring for specified pathogens and immuno-diffusion testing for staphylococcal exterotoxin. We conclude that for pooled human milk, attention to the sterility of collecting vessels, together with precise Holder pasteurisation, results in a bacteriologically safe product which retains many of the protective properties of raw milk.

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Platelet function and morphology in homocystinuria.

Platelet-function tests and morphological investigations by electron microscopy were performed in 8 patients with homocystinuria due to cystathionine synthase deficiency. In 3 patients, all pyridoxine-responders, the collagen-, ADP- and adrenalin-induced platelet aggregation was decreased before treatment; it returned to normal during treatment when homocystine disappeared from plasma. The platelet factor 3 availability, decreased before treatment, also became normal when pyridoxine was administered. The open canalicular system occupied 7.2 per cent of the total area of the platelets before treatment and decreased to 4.5 per cent 3 months later; the α-granules, on the other hand, increased from 7.2 to 11.1 per cent. Functional and morphological studies in the 5 patients already under treatment showed normal results. We conclude that the platelet alterations in untreated patients result from their refractory stage after a release reaction has taken place; the latter may be caused by the homocystine-induced endothelial lesions. In adequately treated patients, our data suggest that no additional administration of inhibitors of platelet aggregation is necessary.

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Effect of Propranolol on the Erythrocyte Membrane and Mechanism of its Action on the Oxygen Affinity.

Propranolol, a β-adrenergic blocker, is known to decrease the hemoglobin oxygen affinity in concentrations 5 to 10 fold the therapeutic level. Since both intraerythrocytic pH and potassium concentration decrease upon propranolol treatment, the change in oxygen affinity could be based on the Bohr-effect or alternatively due to an increased binding of 2,3-diphosphoglycerate to hemoglobin, secondary to a decrease in intracellular ion-concentration. Using a new method to monitor rapid and small changes in oxygen affinity of intact red blood cells, it is shown that almost the whole effect of propranolol in decreasing the oxygen affinity is attributable to a decrease in intracellular pH. Further, propranolol, a membrane active drug modifies the disc shape of erythrocytes (cupping). This morphological change is not associated with the propranolol induced potassium loss and therefore not a result of the concomittantly occurring loss of cell volume. Depletion of the red cells ATP by incubation with iodoacetate inhibits the effects of propranolol, indicating its possible interference with membrane phosphorylation.