The Working Group on Mineral Metabolism
Abstracts for Oral Presentations

78 IMMUNOREACTIVE HYPERCALCITONINEMIA IN FULMINANT MENINGOCOCCAEMIA IN CHILDREN. E. MALLET, A. MEURANT▲, A.M. DEVAUX▲, J.P. BASUYAU▲, P. ENSEL▲, Ph. BRUNELLE▲. Department of Pediatrics, Hôpital Charles Nicolle, 76031 ROUEN, FRANCE.

In an attempt to elucidate the pathophysiology of the hypocalcemia which occurs in fulminant meningococcaemia we found inapropriate immunoreactive hypercalcitoninemia (iCT) (Lancet 1983 ; 1 : 294). It appeared nescessary to determined if CT detected corresponds to CT 1-32 and what causes this hyper CT. Some pathologic situations were investigated. iCT was determined by radioimmunoassay (Mallinckrodt).

Laboratory data (mean ± sem)		iCT ng/ml < 0.4	Calcium mmol/l 2.25-2.60	Urea Nitrogen mmol/l 2-7
-Fulminant meningococcaemia	n=10	1.84±0.24	2.04±0.09	7.64±0.07
-Bacterial meningitis	n=18	0.78±0.11	2.34±0.04	4.71±0.46
-Viral meninfitis	n= 7	0.05±0.01	2.44±0.05	4.9 ±0.51
-Severe deshydratation	n=11	0.67±0.3	2.34±0.10	16.13±2.48
-Septicemia	n= 5	1.59±0.2	2.07±0.10	7.5 ±0.7

Therefore to appreciate the iCT detected, radioimmunoassay curves were performed. Results with infants sera dilutions were not similar to those obtained with human synthetic CT used as standard or medullary carcinoma sera. These data lead to speculate : that iCT detected probably is not CT 1-32. This hyper CT "like" is correlated with shock syndrom especially from septic origin and despite the correlation with hypocalcemia is probably only one of shock syndrom biological signs.

79 Renal responses to acetazolamide and parathyroid hormone in carbonic anhydrase II deficiency - M. Vainsel. F. Vertongen. W.S. Sly. Department of Pediatrics - Université Libre de Bruxelles and Washington University School of Medicine. St.Louis.

Carbonic anhydrase II (CA II) deficiency was identified in a patient presenting with osteopetrosis, and cerebral calcifications. The defect was demonstrated in erythrocytes by starch gel electrophoresis, immunodiffusion with specific anti-serum and reverse-phase HPLC for the quantitation of the CA II and CA I isozymes levels. The patient appeared to have a mixed type RTA including both a proximal and a distal defect. Renal responses to acetazolamide and parathyroid hormone were studied on different days. Acetazolamide injection given at a dose of 100 mg during acid loading, induced an abrupt rise of both urinary pH and bicarbonate. Renal response to IV parathyroid hormone (bPTH 1-34 Beckman, 200 MRC units) was blunted for both urinary cAMP and phosphate excretions. In the serum PTH was normal, calcium slightly decreased (8,6-8,8 mg/dl) and phosphorus elevated for age (6,0 to 6,2 mg/dl), from infancy to adolescence. The enhancement of HCO_3^- excretion due to acetazolamide suggests the persistence of a functionnaly normal membrane bound CA in the kidneys of CA II deficient patients while the blunted cAMP response associated with hyperphosphatemia supports the hypothesis that CA II might be required to achieve the normal intracellular response to PTH.

80 Effect of phosphorus (P) supplementation in very low birthweight (VLBW) infants fed human breast milk (HBM) on calcium (Ca) and P metabolism. L. Sann, B. Loras, F. Durr, L. David, Y. Lasne, Hôpital Debrousse & Hôpital E. Herriot, Lyon, France.

VLBW fed banked HBM are at risk from P depletion with hypercalciuria and high plasma 1.25 OHD concentration. The aim of this study was to deter mine the dose of P required to reduce urinary Ca (UCa) excretion. In addition, its effects on serum immunoreactive parathyroid hormon (iPTH) was also controlled. Forty two infants (GA = 27 - 34 W ; BW = 820 - 1350 g) were fed with HBM (up to 220 ml/kg/24 h) and given daily 1000 IU of vit-D2. 20 were randomly selected in control groups. P as K2 HPO4 was supplemented daily from the 28th day for 4 weeks : 0.48 mmol/kg in 11 infants (group I) and 0.8 mmol/kg in 11 infants (group II). In group I, there was no significant (sig.) change of serum Ca, P, iPTH and UCa (median & range) : 205 umol/kg/24 h (105 - 520) vs 286 (205 - 679) in controls. In group II a sig. increase in serum P and UP was observed with a sig. decrease in plasma 1.25 OHD 186 umol (106 - 271) vs 261 (213 - 293) and UCa 47 umol/kg/24 h (23 - 163) vs 332 (158 - 827) Serum Ca and plasma 25 OHD showed no sig. change. Serum iPTH increased slightly 55 ulEq/ml (<25 - 80) vs 40 (<25 - 50) but remained within the normal range (<100).

These date suggest that the largest dose of P was requiered to reduce UCa excretion. This effect was observed without secondary hyperparathyroidism and with a limited decrease of plasma 1.25 OHD.