67 CYCLIC AMP IN URINE OF HYPOCALCAEMIC NEWBORN INFANTS

L. Aksnes[+], O. Sövik and P. Finne Dept. of Pediatrics, University of Bergen.

Transient hypoparathyroidism has been proposed as an explanation of neonatal hypocalcaemia. We have studied this possibility by examining the urinary excretion of 3',5'-adenosine monophosphate /cAMP/, which is increased by parathyroid hormone. Urinary cAMP was measured on the first, second and third days of life in premature infants with one or more serum calcium values in the range of 6,9 to 8,3 mg per loo ml. The values were compared with those obtained in normal newborn infants. Comparing cAMP excretion on Day 1 and Day 3 the normal newborn infants exhibited an increase from 3,84 ± o,8o /S.E., n=12/ nmoles per mg creatinine to 6,5o ± 1,o6 /n=4/. In the patients with low serum calcium there was a more pronounced increase, from 4,41 ± 1,o6 /n=7/ nmoles per mg creatinine on Day 1 to 1o,4o ± 1,72 /n=5/ on Day 3. The sharp rise of cAMP excretion in the hypocalcaemic patients points to a compensatory hyperparathyroidism rather than hypoparathyroidism.

68 EFFECTS OF COPPER DEFICIT ON MYELINATION

J.-M. Matthieu, A.W. Zimmerman[+], R.H. Quarles[+] and R.O. Brady[+] Service de Pédiatrie, Hôpital Cantonal Universitaire, Lausanne, Switzerland, The Johns Hopkins Hospital, Baltimore, Md. and The National Institutes of Health, Bethesda, Md. U.S.A.

Three generations of rats were raised with a low copper diet. Copper deficiency was associated with significant reductions in the yield of myelin, but brain weight was less affected. The chemical composition of myelin was not different from controls in the content of its proteins, lipids or G_{M1} ganglioside. The main difference was a shift of the major myelin glycoprotein toward higher apparent molecular weight in the copper deficient animals. Postnatal copper replacement failed to reverse the deficiency of brain and body growth or the neurological symptoms. Copper replacement in a copperdeficient mother's diet prior to conception corrected all abnormalities in a subsequent litter when compared to her previous litters. The results suggest that copper is essential for myelin formation and general growth during critical periods in development.

69 EFFECTS OF QUANTITY AND QUALITY OF MILK PROTEIN ON AMINO ACID AND PROTEIN METABOLISM OF PREMATURE INFANTS

K. Heinonen[+], N. Räihä, D. Rassing[+], G. Gaull[+] Dept. Ped. Univ. Helsinki and Dept. Ped. Res. N.Y. State Inst. Res. Ment. Retard.

lo5 prematures, divided into 3 gestational age groups, received either human breast milk /BM/ or one of four isocaloric formulas which provided the following protein intakes /g/kg/day/: F_1=2.2; F_2=4.4 /casein: whey prot=4o:6o/ F_3=2.2; F_4=4.4 /casein: whey prot= 82:18/ BUN was elevated in infants on the high protein diets. Total plasma proteins and A/G ratio reflected the amount of protein in the diet. The essential amino acids in plasma and urine; phe, met, thr, leu, ileu, val, and in addition tyr, correlated with both quantity and quality of ingested protein. Plasma phe and tyr were lo-loo times higher in small infants on F_4 when compared to infants on BM or F_1. Urinary cystathionine was elevated in infants fed diet F_4. There were no significant differences in other plasma and urine amino acids. Physical growth was similar both in formula-fed and in BM-fed infants. The plasma amino acid imbalance produced by high protein/high casein diets, without accelerated growth, makes the use of these diets in pre-term infant feeding questionable.

70 EFFECT OF BLOCKADE OF ANGIOTENSIN II BY SAR 1-ALA 8- ANGIOTENSIN II IN BARTTER'S SYNDROME

L. David[+], L. Sann[+], J. Sassard[+], C. Bizollon[+] /Intr. by R. Francois/ Service de Pédiatrie. Pav.5bis. Hopital Edouard Herriot. Lyon.

Angiotensin II was blocked by its inhibitor Sarl-Ala8- Angiotensin II /Eaton Lab. Norwick NY/ in 2 Bratter's syndromes: case I without and case II with the salt loosing form. Infusion of the inhibitor was performed during 6h at progressive rate /1 to looug/kg/mn/ in case I and during 4h at constant rate /loo/ug/kg/mn/ in case II. Blood pressure /BP/, plasma renin activity /PRA/, Aldosterone /A/, ACTH, Cortisol /F/, Na and K levels, urinary Na and K excretion /as related to creatinine clearence /were studied hourly. In both cases PRA increased sharply but changes were different for the other parameters. In case I, BP, ACTH and F remained stable; A decreased; in urine Na increased while K decreased with opposite changes in plasma. In case II there was a transient decrease in BP; ACTH, F and A raised sharply during the lrst h followed by a progressive decrease; no change occured in Na and K excretion; plasma Na and K showed a small decrease. Conclusion: In case I blockade of Angiotensin induced a basal hyperreninemia and hyperaldosteronemia was an efficient compensatory mechanism for a primary urinary Na leakage. In case II this compensatory mechanism was inefficient.

71 NEW METHODS IN MEASURING TUBERCULIN SENSITIVITY

K. Kouvalainen and Marjatta Lanning Department of Pediatrics, University of Oulu, and The State Public Health Laboratory, Oulu, Finland.

Intracutaneous test with "purified protein derivate" /PPD/ of the tbc bacillus are generally as satisfying for most clinical purposes. In many occasions, however, more accurate methods, are needed. This is especially obvious in clinical research. We have made a comparative study using three different methods in measuring tuberculin hypersensitivity: 1. The classic Mantoux test with PPD, 2. Mantoux test+thermographic measurement of the skin reaction, 3. In vitro test in lymphocyte cultures using PPD as immunogen. The number of PPD sensitive cells was compared with that of PHA reactive cells /total number of T lymphocytes./ Following conclusions was drawn from the results: 1. The two first mentioned methods are semiquantitative. 2. The thermograph is a useful tool in the clinical characterization of the cellmediated immune reaction in the skin. 3. The in vitro test is by far the most accurate method in measuring tuberculin sensitivity. We emphasize, that the in vitro test should be carried in all occasions when the patient is expected to show a weak reaction to tuberculin or when quantitative data are needed for clinical or scientific purposes.

72 EFFECT OF ARGININE LOADING ON RENAL TUBULAR REABSORPTION OF CYSTINE AND LYSINE

A. Sass-Kortsak[+], D.E.C. Cole[+], S. Graham[+] /Intr. by D. Boda/. Res. Inst., Hosp. for Sick Children, Toronto, Canada.

Five children with growth failure undergoing routine investigation by the arginine loading test, a normal adult, a petient with cystinuria /Type II/II/and a heterozygote of cystinuria/Type II/served as subjects. Serial blood samples and quantitative timed urine collections were obtained before and after i.v. infusion of o.5mg/kg body weight L-arginine in buffered saline. Plasma and urinary amino acids were measured by automated column chromatography. The clearences of amino acids and, on the basis of creatinine clearances, the filtered loads and percentage reabsorptions were calculated. Arginine infusion led to vast increases in the filtered load of arginine, minor increase in the filtered load of ornithine, minimal increases in that of lysine, but the filtered load of cystine was not changed. Following the arginine infusion the tubular reabsorption of cystine, lysine, ornithine and arginine was markedly reduced. In the patient with cystinuria marked tubular secretion of cystine was observed. We conclude that under physiological and certain pathological conditions cystine transport is competitively inhibited in vivo by the dibasic amino acid arginine.