

MINERAL METABOLISM

STUDIES OF IMMUNOREACTIVE PARATHYROID HORMONE (IPTH) AND CALCITONIN (ICT) IN INFANTS AND CHILDREN. L. David, C. Anast, Univ. of Missouri, Pediatrics, Columbia Mo., USA.

Serum IPTH levels have been determined in 114 normal children 2 mos. to 15 yrs. old and in 28 children with various disorders of Ca and bone metabolism. Serum IPTH levels in normal children were similar to those of normal adults ranging from non-detectable to 10 μ l Eq/ml with 75% detectable levels. Normal levels were found in 2 cases of osteogenesis imperfecta, in 3 Turner's syndrome, and in 3 children with renal stones. High levels of serum IPTH (35 to 250 μ l Eq/ml) were found in children with chronic renal disease and in children with hypocalcemia associated with a variety of syndromes, including congenital obstructive jaundice, hemolytic uremic syndrome, glomerulonephritis, nephrotic syndrome, hemorrhage and massive transfusions, salicylate intoxication, and acute leukemia. The majority of normal newborns as well as newborns with hypocalcemia had non-detectable to low levels of IPTH during the first 48 to 72 hrs. of life. In another study, serum ICT levels were non-detectable (<50 pg/ml) in 18 normal children and in 2 children with hypercalcemia secondary to vitamin D toxicity. Non-detectable to low levels of ICT were found in normal newborns and in 3 newborns with hypocalcemic tetany. Elevated serum ICT with normal serum gastrin was found in a hypocalcemic 5-week old infant following a massive intestinal resection. The significance of these results will be discussed.

PTH SECRETION IN EXCHANGE TRANSFUSION.

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The citrate load in exchange transfusion (ET) performed with donor blood preserved with acid-citrate and dextrose causes a sharp fall in plasma ionized calcium (I.C. Radde et al. Ped. Res. 6, 43 (1972)). PTH secretion during ET was studied by measuring plasma PTH, Ca, PO₄, citrate and albumen in the donor blood and in the infant during the procedure. Donor plasma PTH was 164 \pm 46 pg/ml, significantly lower than that in the baby, 728 \pm 134 pg/ml (n=18). During the ET the infant's plasma PTH remained steady or rose slightly indicating increased secretion of PTH. Infants studied more than once had increasing PTH secretion with postnatal age or a high steady PTH secretion. ET caused a large net positive balance of citrate and significant negative balances of PTH, Ca and PO₄. The results show that the newborn erythroblastotic infant is able to respond to a fall in plasma ionized calcium with increased PTH secretion and that, in some infants, this response develops in early postnatal life.

IMPORTANCE OF THE PARATHYROID IN THE BONE DEFENSE AGAINST ACIDIC DIET. P. Cuisinier-Gleizes, D. Benest, A. George, H. Mathieu. INSERM U-120, Chateau de Longchamp, Bois de Boulogne 75016 Paris-France.

We studied the effects of acidic feeding on thyroparathyroidectomized (TPTX) rats supplemented with thyroxine & sham-operated pair-fed (Sh.o) rats. The control groups had normal diet. Results. After 7 weeks, Mg & Na in urine & serum, serum inorganic P & alkaline phosphatases, net intestinal Ca absorption, were unchanged in TPTX & Sh-op animals. In both groups, in the urine, pH was similarly decreased, TA, NH₄, Ca & inorganic P were similarly increased. The others parameters showed differences in the rats fed acidic diet depending on whether or not they were Sh-op or TPTX. Sh-op rats showed no change in serum Ca or Cl nor in urinary hydroxyproline or blood pH. In TPTX rats serum Ca & urinary hydroxyproline were increased; blood pH was decreased. Histologic tibial studies evidence osteoporosis in both experimental groups, more marked in the TPTX group. IT resulted from bone formation decreased & bone resorption increased. But there was no increase in the osteoclasts number, rather a small decrease. These data suggest that the osteoporosis resulting from acidic diet is not parathyroid-mediated, & that parathyroid is involved in the defense against acidosis of/by skeleton. (granted by CNRS & CEA).

STUDY OF PARATHYROID ACTIVITY IN VITAMIN D DEFICIENCY RICKETS.

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35 patients with vitamin D deficiency rickets were investigated. PTH levels were determined by radioimmunoassay.

Serum phosphate is decreased. Serum calcium is related to age. Past 15 months of life, hypocalcemia is never observed. PTH has a tendency to decrease with age. No significant correlation could be found between calcium and PTH levels, nor between phosphate and PTH levels. The correlation between PTH levels and aminoaciduria is poor. The subjects were divided into three groups according to calcemia and age: group I-hypocalcemic children younger than 15 months; group II-normocalcemic children younger than 15 months; group III-normocalcemic children older than 15 months. PTH levels are always high in group I and variable in groups II, III. The parathyroid function and parathyroid responsiveness to serum calcium, in vitamin D deficiency state are discussed according to these results.

SERUM-PTH LEVELS DURING RECOVERY FROM VITAMIN D-DEFICIENT RICKETS.

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Several years ago the author showed that administration of exogenous parathyroid hormone (PTH) in a child with healing rickets did not result in hypercalcemia before the bone lesions were no longer visible on X-ray examination (Acta Paed. 53:105, 1964).

A recent study in an infant with severe vitamin D-deficient rickets revealed that the plasma-levels of endogenous PTH fell slowly during administration of vitamin D. Serum-Ca became normal in 4 weeks from the beginning of treatment. At that time PTH was still elevated and the bone lesions had not yet completely disappeared on X-ray examination. It is suggested that the presence of rachitic bone lesions require an elevated PTH-level to maintain serum-Ca within the normal range, as has also been found in dogs (Jowsey, J., J. Clin. Invest. 51:9, 1972).

LATENT PSEUDOHYPOPARATHYROIDISM. H. Peter Kind, David K. Parkinson, Sang Whay Kooh, Donald Fraser. Dept. of Paediatrics, University of Toronto, and Research Institute, Hospital for Sick Children, Toronto, Can.

Testing urinary excretion of cyclic adenosine 3', 5'-monophosphate (Uc-AMP) in response to infusion of parathyroid extract (PTE) is an accurate index for establishing the diagnosis of pseudohypoparathyroidism (PHP). Uc-AMP after one single dose of i.v. PTE (8 U/kg) was measured in five children of two families in both of which one other member is affected with PHP. All five had normal serum calcium and phosphorus levels at the time of testing and none of them had physical signs of PHP. PTE caused a marked increase in Uc-AMP in three children similar to that of controls. From two girls, tested at the age of 5 days and 13 months respectively, one did not respond to the hormone; the other showed only minimal increase in Uc-AMP, identical to the amount observed in patients with PHP. Thus the inherited metabolic defect in the adenyl cyclase-c-AMP system, present in PHP, can already be demonstrated in the presymptomatic state. We conclude that these two children suffer from latent Pseudohypoparathyroidism and advocate that they will develop hypocalcemia later in life.