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Effect of PTH and TCT on renal reabsorption of bicarbonate in pseudohypoparathyroidism.

Four children in the same family, each presenting a pseudohypoparathyroidism were studied. The diagnosis of the condition is based on the clinical features and the absence of an elevation of the urinary cAMP, after the injection of PTH.

Three of the children showed no response with regard to the renal reabsorption of bicarbonate after injection of 200 U.USP of PTH, while the fourth child showed a weak response (8% of the filtered bicarbonate was not reabsorbed). The injection of TCT induced a loss of bicarbonate (bicarbonate wasting) of 10%, 12%, 10% and 13% for each of the four children. This type of response is comparable to that which might be observed in normal subjects or subjects with hypoparathyroidism.

The observations demonstrate: first, that pseudohypoparathyroid subjects show an insensitivity of the tubule to all of the effects of PTH, including the effect on the reabsorption of bicarbonate; secondly, that the sensitivity of the tubule to TCT is normal; thirdly, that the action of PTH and TCT on the reabsorption of bicarbonate probably is brought about through the use of different receptors.

186 B. Peitersen, H. Andersen and W. Hjarde. Treatment of congenital osteogenesis imperfecta with Salmon-Calcitonin and HGH. Children's Hospital, Fuglebakken, Copenhagen, Denmark.

In attempt to improve bone-mineralization and prevent the occurrence of fractures, 2 infants with congenital osteogenesis imperfecta were treated, within the same period of time, with Salmon-Calcitonin 12-15 µU/kg/wk. Treatment was given in two 6 months periods with a period of 6 months in between without treatment. Furthermore at the end of the last period HGH was added to the course (2mg HGH daily/10 days). The daily retention of calcium and phosphate was measured while the patients were on a fixed diet. Furthermore the excretion of hydroxyproline in urine was determined. In the first period of treatment there was a marked increase in calcium and phosphate retention, mostly in the first. A significant decrease in hydroxyproline excretion took place within the first month. In the period without treatment the calcium and phosphate retention decreased initially to pre-treatment values while at the end of this period an increase in retention was observed, which reached the same levels as seen under treatment. In the second period of treatment no further change in calcium or phosphate retention was seen. When HGH was added a marked decrease in retention was noted. The hydroxyproline excretion did not change significantly in the last two periods. **Conclusion:** Treatment with calcitonin results in an increased phosphate and calcium retention and affects collagen turnover as demonstrated in the first period. The observations in the last period may be explained by formation of calcitonin antibodies, compensatory mechanisms in mineral-metabolism or changes in absorption. No change in the occurrence of fractures has hitherto been noticed.

187 A. CONSTANTOPOULOS, V. DIAOURTA*, D. ZOUMBOULAKIS* and N. MATSANIOTIS. Athens Univ., 1st Dept. of Pediatrics, Athens, Greece. Cerebrospinal Fluid (C.S.F.) and Serum Lysozyme Activity in Bacterial and Viral Meningitis.

C.S.F. and serum lysozyme concentrations were determined in 28 normal infants and children, in 19 patients with bacterial meningitis and in 7 with viral meningitis. Serum lysozyme values from patients with bacterial and viral meningitis were found within normal range (9.9±1.24 and 9.5±0.75 µg/ml respectively). High levels C.S.F. lysozyme activity was found in bacterial meningitis (4-12 µg/ml) but significantly lower levels were observed in patients with viral meningitis (2.5-4 µg/ml). A decrease in the lysozyme activity coincided with clinical improvement (1.8-4.3 µg/ml). A positive correlation there was present between lysozyme activity and total number of leucocytes and percentage of polymorphonuclear cells of C.S.F.

In conclusion the lysozyme activity in C.S.F. could be significant value in detecting an inflammatory disease of the central nervous system.