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Induction of cystathionase in human fetal liver explants.

Previous studies have shown that cystathionase activity is virtually absent from human fetal liver and develops some time after birth. In the present study we describe the in vitro effects of dibutyryl cyclic AMP(dBcAMP), glucagon and dexamethasone on the activity of cystathionase in human fetal liver. Human fetal liver explants (gestational age 10 to 20 weeks) incubated in organ culture for 1-3 days contained only trace amounts of cystathionase activity. Incubation of liver explants for 24 hours with dBcAMP (0.2mM) in the presence of theophylline (0.5mM) increased the activity of cystathionase to adult human levels (104-198 nmoles cysteine/mg protein/h). Incubations in the presence of glucagon (150ug/ml) or dexamethasone (20ug/ml) also increased the cystathionase activity of the liver explants to the similar levels. The increase in cystathionase activity was inhibited by simultaneous incubation with cycloheximide or actinomycin D. These experiments show that cystathionase activity can be induced in human liver in vitro.

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The effect of thyroxine on renal phosphorus handling

Elevated serum-P-levels in thyrotoxicosis have been ascribed to increased renal P-reabsorption as a result of suppressed parathyroid activity. In acute experiements, Beisel found a decrease of TRP under T3 in PTX dogs. In our experiments the longterm effect of experimental hypo- and hyperthyreoidism on renal P-handling in PTX rats was measured. Hypothyroidism (TX): thyroidectomy 3 w. prior to the experiment; hyperthyroidism (Ht): daily i.p. inj. 0,05 mg T4 over 6 days. Results: Serum-Ca: TXPTX 3,42±0,06 mEq/l; euthyroid PTX 3,23±0,06; HtPTX 3,13±0,08. Serum-P: EuPTX 14,4±0,276 mg%; TXPTX 12,7±0,325 mg%; HtPTX 15,5 ±0,255 mg% ($p < 0,01$). No significant difference of TRP in any group. Correlation between filtered load and TRP showed that, - at any given GFR x serum-P, - TRP was higher in thyrotoxicosis.

Conclusion: Hyperphosphatemia was induced by T4 even in PTX rats. Therefore elevated serum-P-levels in thyrotoxicosis are not due to parathyroid suppression but due to parathyroid suppression but due to a renal effect of T4 (increased tubular reabsorption).

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Study on the site of renal salt loss in 3 patients with Bartter's syndrome.

A sodium loss syndrome has been shown in some patients with Bartter's syndrome. According with Chaimovitz's experience, we tried to localise the site of renal salt loss in this syndrome. We have studied 3 children with Bartter's syndrome submitting them to an intravenous 0,45% hypotonic saline infusion, until a total dose of 2.000 ml/m/1.73 m^2, adjusting the rythm of perfusion to exceed urinary flow by 5 ml/m/1.73 m^2 and having begun the perfusion at a rate of 5 ml/m/1.73m^2. The results obtained in a group of 6 healthy children, gave to us the following figures(all them referred to 100 ml of GFR): V/m: 15.9 cc/m; Cl_{H2O}: 11.6 cc/m; Cl_{Na}: 1.8cc/m $Cl_{H2O}+Cl_{Na}$: 13.4 cc/m; $U_{Na}V$: 262 Eq/m; U_KV: 51 Eq/m; being $Cl_{H2O}/Cl_{H2O}+Cl_{Na}x100$: 86% and Cl_{in} 111.6 cc/m/1.73 m^2. In two of our patients, V/m and Na delivery were found normal; Cl_{Na}, $U_{Na}V$ and U_KV were increased, and Cl_{H2O} and $Cl_{H2O}/Cl_{H2O}+Cl_{Na}x100$ were reduced. The third patient showed an increase of $U_{Na}V$, U_KV and Cl_{Na}, but Na delivery, as well as Cl_{H2O} were significantly increased while $Cl_{H2O}/Cl_{H2O}+Cl_{Na}x100$ remained low. These results suggest that the site of renal salt loss in the two first patients is the ascendent limb of Henle. On the other hand, the third patient seems to demonstrate a defective proximal tubular Na reabsorption with a good compensatory mechanism in the ascendent limb of Henle.

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Modifications in fluid compartments, electrolytes and intracellular proteins of muscle in uremic children.

Water, electrolytes (Na,K,Cl,P) and intracellular proteins were determined on muscle sampled by needle biopsies in 50 chronic uremic children. Their creatinine clearance was below 30 ml/mn/1.73 m2 and seven patients were already in chronic haemodialysis. Total water was increased in all cases. The increase was due to an excess of ECW and ICW in moderate chronic renal failure, while at the terminal stage,only ECW was augmented. BP were positively correlated with ECW. Muscle sodium (Na_m) content increased with the renal failure. ECW and Na_m were in direct relation, however the Na_m was increased less than the expansion of ECW. Muscle chloride, normal in many cases, was very high (>2 Cl^-) in 9 terminal cases.Intracellular K^+ was increased in half of the patients especially dialysed and terminal uremic patients. Total phosphorus was slightly increased only in terminal renal failure. These modifications in muscle electrolytes cannot be assumed by their plasma values. Total intracellular proteins were below the normal range in more than half the cases. These results suggest that there is a correlation between alteration of renal functions and changes in muscle milieu. However these disturbances appear obvious only when renal function is really reduced (cl. creat. < 5 ml/mn/1.73 m2).