**23** E.RITZ*, G.GILLI*, O.MEHLS, A.FAHRIG* Medizinische- und Kinderklinik der Universität Heidelberg,FRG. Effect of vitamin $D_3$ and 1.25 $(OH)_2D_3$ on growth in experimental uremia(EU).

Growth retardation is a major unsolved problem in children with chronic renal failure. A stimulatory effect of $D_3$ on growth rate in EU has been demonstrated(Mehls et al. Am.J.Clin.Nutr.,1978),but clinical results are conflicting. Chesney et al.(New Eng.J.Med.,1978)reported increased growth velocity in uremic children treated by 1.25 $(OH)_2D_3$ who had failed to respond to high dosage $D_3$.The present study was designed to compare the efficacy of $D_3$ and 1.25$(OH)_2D_3$(application i.p.;3-point dose response curve) on growth in rats with EU. Methods: 90g Sprague-Dawley rats with stable uremia(2-stage subtotal nephrectomy) of 2 weeks' duration($C_{CR}$ 18% of normal). All animals fed ad lib. Five groups: I: NX solvent; II and III: NX+$D_3$ (0.5 and 1.0 µg/70g/day); IV and V: NX+1,25$(OH)_2D_3$( 0.625 and 1.25 ng/70g/day). Results: Serum Ca increased ($p<0.01$) to the same extend in NX+$D_3$ and NX+1.25$(OH)_2D_3$. Compared to NX solvent animals(5.9±0.2cm/14 days;21±5g/14 days,n=14), the growth rate was significantly better in NX+$D_3$ (6.4±0.3cm/14 days;31±6g/14 days;n=12) and in NX+1.25$(OH)_2D_3$ (6.6±0.3cm/14 days; 32±6 g/14 days;n=12). The difference between NX+$D_3$ and NX+1.25$(OH)_2D_3$ was not significant. Conclusion: The present study can not confirm that 1.25 $(OH)_2D_3$has a more beneficial effect on growth in uremia than $D_3$.

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Familial hypophosphatemic rickets and idiopathic Fanconi syndrome: Effect of 1,25(OH)2 cholecalciferol *(Rocaltrol®)* (1,25(OH)2CC) on metabolism of inorganic phosphate (Pi).

2 patients with familial hypophosphatemic rickets (FHR) and 1 patient with idiopathic Fanconi syndrome under oral phosphate treatment were given 1,25(OH)2CC (0,75-1,5 µg/day) and the effects on the intestinal absorption and renal tubular handling of Pi were studied. Intestinal absorption of Pi: During a 3-hour oral loading test (300 mg Pi) the serum Pi levels were significantly higher after 1,25(OH)2CC treatment, and ionized serum calcium levels did not change. Thus, in the 3 patients Pi intestinal absorption was increased by 1,25 (OH)2CC.

Renal handling of Pi: In the 2 patients with FHR, 1,25 (OH)2CC increased Pi filtered load and absolute tubular reabsorption and decreased fractional clearance (before treatment: 34 and 39%, after treatment: 8 and 12% respectively). In the patient with idiopathic Fanconi syndrome, 1,25(OH)2CC increased Pi filtered load and absolute tubular reabsorption but the fractional clearance did not change. This strongly suggests 2 different pathophysiological mechanisms responsible for the renal tubular loss of Pi. In the 3 patients, 1,25(OH)2CC markedly improved the bone lesions.

**25** E. MALLET, M. GARABEDIAN, J.P. BASUYAUX Dept de Pédiatrie, Hôp. Charles Nicolle, ROUEN and INSERM U 30, Hôp. des Enfants Malades, PARIS (FRANCE). EVALUATION OF THE INHIBITORY EFFECT OF VITAMIN D METABOLITES ON CIRCULATING IMMUNOREACTIVE PARATHYROID HORMONE (IPTH) IN INFANTILE NUTRITIONAL RICKETS : STUDY OF VARIATIONS OF SERUM IPTH AND 24,25 and 1,25 DIHYDROXYCHOLECALCIFEROL LEVELS AFTER 25 HYDROXYCHOLECALCIFEROL ADMINISTRATION (25 HCC). Recent works have demonstrated an essentially inhibitory effect of vitamin D on IPTH secretion. We present an in vivo study, carried out in ricket patients, in an attempt to confirm previous observations and also contribute to a better physiopathological understanding of this affection. 13 and 23 months old advanced nutritional rickets patients who presented hypocalcemia and hypophosphoremia were initially treated exclusively with 25 HCC (DEDROGYL®) at a dose of 20 µg per day. The serum calcium and IPTH couple was assayed daily (IPTH determined by BW 211/41 antiserum). Serum hydroxylated vitamin D metabolites (25;24,25;1,25 HCC) were concurrently determined. Prior to administration, there were high IPTH levels corresponding to a low serum calcium titer. Following 25 HCC administration, a decrease in IPTH levels was observed in spite of the persistance of identical low serum calcium. This decrease attained a maximum value of 36 % at the 5th day. Furthermore, there was a decrease in alkaline phosphatases and urine cAMP excretion. During this initial IPTH fall no change in serum 24,25 HCC level was observed while 1,25 HCC increased and there was a negative correlation between IPTH and 1,25 HCC levels. Considering the fact that hypocalcemia is the only recognized stimulator of IPTH secretion, these findings are in agreement with the hypothesis that vitamin D metabolites and mainly 1,25 HCC have an inhibitory effect on parathyroïd hormone secretion in nutritional rickets

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The Effect of Phototherapy on Neonatal Vitamin D Metabolism.

Phototherapy for the treatment of neonatal jaundice has now been widely accepted. Its effect on Vitamin D metabolism is however unknown. In this study 10 full-term jaundiced babies receiving phototherapy were investigated. Serum 25OHD, 24,25$(OH)_2D$, Ca and P were measured. Samples were taken before the onset of phototherapy, 48 hours after the onset and 24 hours after the finish of phototherapy. The mean weight of the 10 babies was 3.4 kg, mean age of onset of phototherapy 64 hours of age,mean bilirubin level at onset 17.1mg%. The mean duration of phototherapy was 83 hours. Causes of jaundice were $G_6PD$ deficiency (1), rhesus incompatibility (1), blood group incompatibility (3), and unknown (5). Infants were nursed naked under the phototherapy light which was provided by seven daylight fluorescent lamps 60 cm above the baby. Babies were fed a cow's milk formula not containing Vit. D. The mean serum 25OHD and serum 24,25$(OH)_2D$ before phototherapy were 8.49±6.91 ng/ml and 1.29±2.64 respectively, 48 hours after the onset were 8.72±6.10 and 0.84±1.53 and 24 hours after the finish were 7.51±5.34 and 0.46±0.74 respectively. Mean calcium values before and 24 hours after the finish of phototherapy were 9.36±1.10 mg% and 9.83±0.93 mg%. Likewise mean phosphate values were 6.19±1.58 and 7.35± 0.97 mg%. The results of this preliminary study thus suggest that the neonatal jaundiced skin does not convert provitamin D to active Vitamin D in response to phototherapy light (420λ). Neither was hypo or hypercalcaemia observed after phototherapy as has been previously reported.

**27** L. SANN, R. ROUSSON, A. FREDERICH, L. DAVID, P. GUIBAUD

**SEVERE RICKETS IN A 41 DAY OLD TURKISH BOY DUE TO MATERNAL VITAMIN D DEFICIENCY.**

**A turkish boy was referred at age 41 days for convulsions. Clinical and radiologic examination showed marked features of rickets. Serum calcium (Ca) and phosphorus (P) concentrations were 1.74 and 1.66 mmol/l respectively and alkaline phosphatase 2388 IU/l. Serum immunoreactive PTH concentration was > 400 µlEq/ml (N < 100) and serum i Calcitonin concentration was normal ( < 150 pg/ml). Plasma 25 hydroxycholecalciferol (25 OH CC) concentration was < 10 nmol/l (N : 25-80). Hypocalcemia and rickets were cured by Ca supplementation, 1300 µg of 25 OH CC and 200 000 IU of vitamin $D_3$. His mother has low serum Ca : 1.60 mmol/l and non detectable plasma 25 OH CC concentrations.**

**These data suggest that this early rickets was from maternal origin.**

**28** W.KREUSSER,* O.MEHLS, R.WEINKAUF,* E.RITZ.* Medizinische- und Kinderklinik der Universität Heidelberg,F.R.G. Effect of somatotropin(STH), parathyroid hormone(PTH) and calcitonin(CT) on cAMP-content of growth cartilage in uremia.

Retardation of growth is a well known feature in uremic children.There is considerable evidence that several hormone systems,which play an important role in normal growth,e.g.STH,PTH are altered in uremia.Adenylate cyclase mediates the action of most of these hormones on cell proliferation in the chondrocytes.The effects of STH,PTH and CT on cAMP content of cartilage was investigated. Methods:Male Wistar rats(60gBW)were bilaterally nephretomized(NX)or sham-operated(SO)24h before sacrifice.Bovine STH(6h),PTH and CT(15min) was given i.v. cAMP in cartilage was measured by RIA. Results: Basal cAMP content in cartilage was not different between NX and SO. Stimulation with 40IU i.v.PTH caused a significantly($p$ 0.01)lower cAMP increment in NX(11.4±1.0;SEM; n=20)than in SO(24.0±2.6).CT(2.5U)administration resulted in higher cAMP in NX(23.8±1.8;n=19)vs.SO(16.6±1.6). STH(0.1-5.14)was without effect on cAMP in NX or SO,but enhanced $^3$Thymidine incorporation. Discussion: Target organ resistance to PTH seems to be reflected by diminished cAMP response in uremic growth cartilage.CT has the opposite effect on NX.In the above model,with the dose of STH used,no effect on cAMP could be seen.Our present results provide evidence that in acute uremia the cAMP system may be involved in the growth abnormality of RF.