ROLE OF THE KIDNEY IN THE PARATHYROID CONTROL OF SERUM CALCIUM. H. Mathieu, P. Cuisinier-Gleizes, INSERM. U-120, Chateau de Longchamp, Bois de Boulogne, 75016 Paris, France

The mechanism by which the kidney is involved in the parathyroid control of serum Ca is questionable. To approach it we measured the serum Ca (mg/100ml) 6 hours after total nephrectomy (NX) or bilateral ureteral ligation (UX) or sham-operation (Sh.o) in rats undergoing at the same time either parathyroidectomy (PTX) or sham-operation (Sh.o). Results. NX-Sh.o: 16.6 ± 0.45; NX-PTX: 10.3 ± 0.28; UX-Sh.o: 8.0 ± 0.30; UX-PTX: 5.3 ± 0.17; Sh.o-PTX: 4.8 ± 0.10; Sh.o-Sh.o: 8.6 ± 0.35. These data show 1) after total nephrectomy the SCa rises but not after bilateral ureteral ligation; 2) total nephrectomy prevents the fall in SCa after PTX; in contrast ureteral ligation does not. Inasmuch as bilateral ureteral ligation fully suppressed the urinary excretory function, we may conclude that 1) this one is not or poorly involved in the parathyroid control of SCa; more probably this control is related to endocrine or metabolic function of the kidney; 2) if the prolonged half-life of PTH can explain why SCa stays normal in NX-PTX rats, the rise of SCa in NX-Sh.o rats means that total nephrectomy results in an unresponsiveness of PTH secretion control. (granted by FRMF)

CORRELATION BETWEEN SERUM PTH LEVELS AND BONE MORPHOLOGY IN CHILDREN WITH RENAL OSTEODYSTROPHY. Mehls O., Krempien B., Ritz E. Scharer K., Schuler.

In 106 uremic children, aged 4-17 years, a study was performed to evaluate clinical signs of metabolic bone disease, serum biochemistry, serum PTH levels, roentgenological signs and iliac crest histology. A correlation was found between serum PTH levels and intensity of subperiosteal resorptions and of acroosteolysis on X-rays. Higher serum PTH levels and higher alkaline serum phosphatase levels were found in children with severe metaphyseal changes in the long tubular bones (particularly in those with epiphyseolysis) as compared with children without metaphyseal abnormalities. Serum PTH levels could also be correlated with quantitative measurements of bone histology.

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FOLLOW-UP EXAMINATIONS OF THE MINERAL SALT CONTENT IN THE SKELETON WITH VARIOUS VITAMIN D RESISTANT FORMS OF RICKETS OF RENAL ORIGIN. W. Schuster, Erlangen, Germany.

With the aid of experimental facilities specially developed for paediatric requirements, 10 patients affected by chronic phosphatic diabetes, 2 patients with the Debré-de Toni-Fanconi syndrome, 2 children with distal tubular acidosis of the Albright type and 8 children with uraemic osteodystrophy were examined during the last 4 years to determine the mineral salt content in peripheral parts of the skeleton and, in some, also in the spine. The results of the longterm follow-up reveal the different response to the therapeutic measures so far possible, in the case of the various vitamin D resistant forms of rickets of renal origin. In chronic phosphatic diabetes, even long-term treatment fails to replenish the calcium depots in bones to any noticeable extent. With the Debré-de Toni-Fanconi syndrome and Albright's disease, therapy brings about a rapid increase of the mineral salt concentration in the skeleton. Also with uraemic osteodystrophy the treatment usually leads to a marked increase of the mineral salt content and occasionally even to raised values.

THERAPEUTIC AND COLATERAL EFFECTS OF 25-HYDROXYCHOLECALCIFEROL IN VITAMIN D DEFICIENCY. M. Moya, J. Colomer, and J. Beltran. Dept. Pediatrics, University of Valencia, Spain. Vitamin D polar metabolite, 25-HCC has been used in 2 infant groups whose ages range from 0-18 months. In the 1st group of 15 infants, all of them suffered from rickets, clinically, biochemistry and radiologically and no prophylactic vitamin D administration could be demonstrated. The control group (15 infants too) was admitted with similar diseases but no one suffered from rickets. Routine determinations for metabolic assessment of Ca phosphoric metabolism were performed in plasma and in 24h. urine collections, prior and after 25-HCC administration. The rickets group received 20 mcg/day, orally for 20 days and the control 10mcg/day in the same way. The biochemical study demonstrated that both groups were significantly different at the beginning because of plasmatic phosphate and alkaline phosphatase, and in urine because of calciuria and pH (p. 0.05). And neither plasma Ca, nor titrable acidity or ammoniuria were significant. After treatment the rickets group showed a conspicuous normalization of biochemical parameters consistent with the clinical findings. Nevertheless the most important feature was the sharp rise in calcemia and calciuria (p 0.01) in both groups. In the control urine Ca was over 6mg/kg/day average; with treatment, urine pH differences disappeared. There is as well a therapeutic trial with 8 cases of metabolic rickets.

AN UNUSUAL TYPE OF IDIOPATHIC OSTEOPOROSIS. A. Fanconi (Winterthur) and G. Fanconi (Zurich)

A 17 year old boy and a 11-year-old girl, both with negative family histories, have been observed since the age of 6 years (case 1), and of 3 months (case 2) Generalized osteoporosis without rickets was discovered at the age of 9 years (case 1) and 3 years (case 2), and persists without any sign of improvement. Two pathological fractures occurred in the boy, 3 in the girl. They healed with hardly any callus formation. The serum phosphorus is slightly decreased in both patients, while serum calcium, alkaline phosphatase, and renal functions are normal. The boy shows a marked hypercalciuria (6 to 13mg/kg/24h.), which responds to treatment with oral sodium phosphate, but not to hydrochlorothiazide. In addition, there is a severe mental and a moderate growth retardation in both children. The boy is microcephalic. No other chronic disease could be found which would cause the bone disease. This osteoporosis does not fit into the picture of osteogenesis imperfecta nor of idiopathic juvenile osteoporosis, and might therefore be another type of demineralizing bone disease.

FETAL DEVELOPMENT IN EXPERIMENTAL UREMIA Bommer J., Ritz E., Krempien B., Mehls O. Medical School, Heidelberg, Germany.

Since under maintenance hemodialysis several pregnancies have been carried out to term, the effects of maternal uremia on fetal development are of considerable interest. Experimental uremia was induced in inbred rats on the 15th day of the 23 days' pregnancy by 5/6 nephrectomy. Feti were removed surgically on the 21st day. Findings: While longitudinal growth was not impaired, feti of uremic mother rats weighed less than control feti. Skeletal development: Mineralisation both in desmal and enchondral ossification centers was entirely normal (in contrast to feti of rats with nutritional vitamin D deficiency), suggesting synthesis of active vitamin D metabolites in fetal kidneys. No evidence of parathyroid overactivity was detectable. Comparison of the effects of administration of PTH to the mother versus injection of PTH into fetal livers suggests a relative impermeability of the placenta to PTH. Erythropoiesis: Neither the number nor the composition of hemotopoietic foci in organs with extramedullary hemotopoiesis was abnormal.