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Growth and bone histology in experimental uremia

Material and Methods: Male Sprague-Dawley rats were subjected to subtotal nephrectomy (2 stage procedure with irradiation of rest parenchyma) reducing GFR(C_{CR}) 4 weeks p.o. to 19.8% of shamop, paired controls. Serum (creatinine, urea, electrolytes) and urine (electrolytes, protein, cAMP, aminoacids) chemistry, fecal fat and nitrogen, body weight, body length, tibia geometry, tibia histology (v.Kossa stain) and total body fat were measured. **Results and conclusions:** Longitudinal growth in uremic rats was reduced with respect to ad lib. fed control rats, but identical with respect to paired control rats. The reduction of longitudinal growth is entirely accounted for by reduced calorie and protein intake in uremia (U), although rickets (histology) and 20 hyperparathyroidism (histology, urinary cAMP) are present in the U skeleton. In spite of their identical length, weight was less in U rats than in control rats. This difference is not accounted for by difference of intracellular water (measured as difference organ dry weight - organ wet weight) or extracellular water (measured as 82 Br-space). It may point to a disturbance of protein synthesis in U.

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Congenital rickets a premature infant : evolution of serum parathyroid hormone (iPTH) and plasma 25 hydroxycholecalciferol (25 OH CC) (Intr. by R. FRANCOIS)

A small-for-date premature infant (gest. age = 34 weeks, birthweight 1100g) had typical X-Rays aspect of rickets at birth. At the age of 4 days, low serum Ca = 8,2 mg/100 ml, low serum P = 8,4/100 ml and high serum alkaline phosphatase (A.P.) 323 IU/ml (< 200) were found. Very high level of serum iPTH was found at 17 days of age : 295 pEq/ml (N < 50). Evidence of maternal vitamin D deficiency was shown by low plasma 25 OH CC : 1,1 ng/ml (N 13 ± 4). Ca infusion (15 mg/kg/3h) resulted in marked decrease in iPTH (280 to 84 pEq/ml). At the age of 30 days, administration of vitamin D₂ (2400 u/day) for 10 days induced some healing of X-Ray aspect ; A.P. remained high : 400 IU/ml ; plasma 25 OH CC was normal 10,2 ng/ml and serum iPTH decreased to 115 pEq/ml. At the age of 40 days, oral administration of 25 OH CC (15 µg/24h) was given for one week : it resulted in a rise of 25 OH CC to 64,5 ng/ml with minor change of serum iPTH (94 pEq/ml). This data show that :

- 1) a secondary hyperparathyroidism is present in congenital rickets
- 2) it is normally depressed by calcium infusion
- 3) in view of the recent demonstration that 1-25 OH CC may be an important factor of regulation of PTH secretion (B. S. CHERTON et al. S.C.I. 56, 688, 1975), the absence of iPTH normalization in our case, in spite of high plasma 25 OH CC suggests a reduced activation of 25 OH CC.

6 N. MATSANIOTIS, T. KARPATIOS*, F. MAOUNIS*, P. NICOLAIDOU*, C. THEODORIDIS*. 1st Department of Pediatrics of Athens University, Athens 608, Greece. Hyperparathyroidism in children with endemic fluorosis.

Dental fluorosis was found in nearly 80% of 520 children aged 12 to 18 years who lived permanently in two Greek villages Kolchikon and Lavrion where the water supply contains 3,6 and 2,7 ppm of fluoride. Detailed radiological, ophthalmological and biochemical studies in 52 children with marked dental fluorosis revealed that 29 had bone changes in the mandible consisting of erosion or loss of the lamina dura, cystic rarefaction and sclerosis; opacities of the lens were seen in 57% of the patients. Serum Ca was found to be in the normal range in the majority of patients. Serum P, Mg and creatinine as well as blood urea and urinalysis were normal. Serum alkaline phosphatase was generally high in the presence of bone changes. Serum parathyroid hormone was elevated in 10 children (685, 980, 1200, 1280, 2450, 2600, 3000, 3100, 6200, 7200 pg/ml) with increased serum alkaline phosphatase and with skeletal changes resembling hyperparathyroidism and normal in four children (185, 280, 420, 630 pg/ml) who had normal serum alkaline phosphatase and no bone changes. It is suggested that fluorosis can cause secondary hyperparathyroidism.

8 D. BRACHER* (Intr. by F. VASSELLA). Department of Pediatrics, University of Berne, Berne, Switzerland. Disturbed vasomotion of the central retinal arteries in neonatal postasphyxia.

Owing to the simplification brought about by the use of a special contact lens, the fundus oculi of neonates was repeatedly photographed even during intensive care with a hand held fundus camera. The true dimensions of vessel diameters are obtained when measurements on the film negative are converted according to the refractive power of a theoretical eye model and corrected for individual ametropia. The main branches of the central retinal artery have a diameter of 80 - 150 microns. In uneventfully born term births there is a small decrease if any between the diameters measured at 2h and at 24 or 48 h of life. Neonates with congenital cardiac malformation show changes according to what was expected: dilatation in hypoxia, hypercapnia and hypotonia, constriction in hypertonia. Asphyxiated neonates may have in their first hours of life dilated arteries, even in the presence of hyperoxia (320 mm Hg, extreme measurement). Later on the arteries may appear narrow even in the presence of hypoxia and hypotonia. The diminution can go up to 40%. We assume that these results are of interest both for research in retrolental fibroplasia and in cerebral perfusion (many common factors in vasomotion of both systems). Study approved by the ethical committee, Department of Pediatrics, Berne.

9 JOHNSON, P., HARDING, R. H., McCLELLAND, M. E., & WILKINSON, A. H. Nuffield Institute for Medical Research, University of Oxford, England.

Cardiovascular changes associated with laryngeal stimulation and feeding in newborn lambs.

Apnoea initiated by stimulation of chemoreceptors at the entrance to the larynx with certain fluids has been described previously. The cardiovascular changes associated with this form of apnoea are instantaneous & not associated with asphyxia although they may be enhanced by concurrent asphyxia. In 18 unanaesthetized lambs the changes in cardiac output (C.O.) and its distribution, carotid arterial and abdominal aortic flow, H.R., arterial pressure (AP) and blood gases were measured during water stimulation of the larynx (8 breathing air and 5 100% oxygen) and in 5 during spontaneous feeding. Mean AP rose (+26%), HR fell (37%) and CO fell (26%) during apnoea. While blood flow increased to brain (+204%), the head extracerebral tissues (+86%) myocardium (+80%) and lungs (bronchial arterial plus L-R shunting) (+35%) it fell elsewhere, especially to small gut (-68%) and kidney (-32%). Similar changes still occurred in 5 lambs breathing 100% O₂. During feeding significant increases in flow to the head (+160%) and brain (+86%) occurred while pO₂, fell and pCO₂ rose significantly. It is suggested that similar reflex cardiovascular events may have considerable importance in the neonatal period particularly in the premature infant.

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Insensible water loss of low birthweight newborns in a warmed wall incubator.

Low birthweight newborns are treated in an incubator in which the temperature of both the wall and the air are controlled. The air entering the incubator is very low. In 30 newborns (0.88-2.88 kg), all in neutral temperature, insensible water loss is estimated by means of a continuous weighing device. Results are presented for the first and the second week of life. The insensible water loss of newborns >1.75 kg is 19.3-26.2 g/kg/day (mean), however in very low birthweight newborns (<1.25 kg) the insensible water loss is much larger, 54.2 ± 12.9 g/kg/day. (first week) and 42.2 ± 13.4 g/kg/day (second week) (mean and s.d.). The results are compared with reported insensible water loss of newborns in humidified conventional incubators and radiant heaters. It is concluded that insensible water loss of newborns in this type of, non humidified, incubator is lower than in conventional humidified incubators and under radiant heaters. It is suggested that this is due to the low air velocity (<0.01 m/sec) in this incubator.