A. DONATH, P. INDERMUHLE*, D. NUSSLE* and R. BAUD*, Center for the Study of Bone Diseases, Hôpital Cantonal, University of Geneva, Geneva, Switzerland.

In vivo determination of bone mass in children
Bone mass has been determined at the distal third of the radius shaft by measuring the attenuation of a beam from a ^{125}I source when it crosses bone. This technique has a precision of 2 % and the radiation dose to the forearm is 2 mrad (natural dose in Geneva : 2 mrad/week). Three groups of healthy children aged 6 to 19 years have been measured : 967 live in the city of Geneva, 333 in a country village (Vollèges) and 164 in the next village (Sembrancher), where water is naturally fluoridised. Bone mass increases with age, and after puberty values of boys are higher than the ones of girls. The differences between the three groups are not significant, but the values obtained in the United-States are at all ages higher than ours. In another group of 96 hospitalised children bone mass has been compared to metacarpal cortical thickness and the correlation is good ($r=0.89$). A group of about fifty children suffering from various disorders of phosphocalcium metabolism present more or less severely reduced values. This simple, precise and easily reproducible examination is the best one to measure a diminution of bone mass quantitatively, to follow the natural evolution of a disease or the effect of a therapy.

R. STEENDIJK, Departments of Paediatrics and Histology, University of Amsterdam. Plasma-PTH, serum-Ca and bone structure in hypophosphataemic (vitamin D-resistant) rickets.

In 1972 Jowsey showed in dogs that bone became unresponsive to PTH in the presence of much osteoid (J. Clin. Invest. 51: 9, 1972). This might also explain the elevated PTH-levels in vitamin D-deficient rickets in man. But if rachitic bone structure is the cause of the raised plasma-PTH values, this should be independent of vitamin D and therefore plasma-PTH levels in other types of rickets should also be above normal. This was examined in 2 patients with hypophosphataemic rickets (a 16-year-old boy and a 7-year-old girl). Serum-Ca and plasma-PTH were determined simultaneously. A bone biopsy, taken at the same time, was embedded in methyl-methacrylate without decalcification, and 100 μ thick sections were made. These were examined microscopically: the length of the bone surface, covered with osteoid, was measured and expressed as a fraction of the total length of the surface. Serum-Ca and plasma-PTH were normal, whilst more than 75 % of the bone surface was covered with osteoid. These findings do not support the hypothesis that in man the effect of PTH on the homeostasis of calcium is inhibited by severe rachitic changes of bone tissue.

C. RICOUR, A.M. DARTOIS* and S. BALSAN. Laboratoire des Tissus Calcifiés (CNRS ER 126 et INSERM U.30). Phosphorus depletion in children on long term total parenteral nutrition.

During the past 4 years we have had on long term parenteral nutrition 70 infants. In 7 of these patients an important hypophosphatemia (Pi below 20 mg/L), associated to neurological symptoms in one case, was observed without any significant change of serum Ca concentration. The purpose of the present study was : 1) To analyze the factor(s) responsible of phosphorus depletion during long-term parenteral nutrition. 2) To determine the amounts of I.V. Calcium, phosphorus and nitrogen necessary for the prevention of such accidents. This investigation was done by stable balance technique under a constant caloric intake (100 Cal/Kg/24 h) ; calcium, phosphorus and nitrogen intakes were varied simultaneously or alternatively : Ca from 25 to 60 mg/Kg/24 h, P 10 to 60 mg/Kg/24 h, and N 320 to 640 mg/Kg/24 h. The subjects studied included 9 infants. Results are as follows : 1) Phosphorus balance is highly correlated to calcium, nitrogen and caloric intakes ; 2) When excessive amounts of protein and/or calcium are given intravenously, a state of phosphorus depletion, most probably secondary to cellular anabolism, may occur ; and 3) A caloric intake of 100 Cal/Kg/24 h, a calcium intake of 35 mg/Kg/24 h, a nitrogen intake of 400 mg/kg/24 h and a phosphorus intake of 40 mg/Kg/24 h will prevent this accident and promote a satisfactory development of the child.

H.E. FRANZ⁺, K. HELMKE⁺, K. FEDERLIN⁺, P. PIAZOLO⁺, E. LINK⁺, J. STRÖDER⁺ and R. JESCHKE⁺ (intr. by E. Gautier). Department of Medicine, University of Ulm, Childrens Hospital, University of Würzburg, Federal Republic of Germany^X. Determination of calcium binding protein in duodenum of rachitic babies before and after treatment with 25-hydroxycholecalciferol.

Antiserum directed against calcium binding protein (CaBP) isolated from human kidneys was used for the immunofluorescent localization of CaBP in human intestine. Frozen sections of intestine obtained by biopsy from normal persons were tested by the indirect fluorescent antibody technique. Specific fluorescence indicating the presence of CaBP was observed at both the basal and apical poles of the intestinal absorptive cells while the goblet cells appeared to fluoresce non-specifically. Treatment of rachitic children with 25-hydroxycholecalciferol generally restored the pattern of fluorescence seen in intestinal tissue from normal persons. Concentration of CaBP as determined by the LAUREL-technique increases after treatment.

^X Supported by Deutsche Forschungsgemeinschaft (Contract FR 200/13)