G. MAYOR\*, A. TORRADO\*, N. GENTON\* and J.P. GUIGNARD. Departments of Paediatric Surgery and Paediatrics, Hôpital Cantonal Universitaire, Lausanne, Switzerland.
A follow-up study of renal function in post-

A follow-up study of renal function in postobstructive nephropathy. Renal function was studied in children with

congenital obstructive uropathy, and followed for 3 to 13 years after reconstructive surgery. Glomerular filtration rate (GFR) was assessed by the clearance of endogenous creatinine or inulin. Effective renal plasma flow was assessed by the clearance of PAH. In 14 out of 28 patients, reconstructive surgery was performed during the first year of life. Urines were subsequently kept sterile. Three different patterns of evolution could be observed after repair of obstructive uropathy: 1) GFR remained normal and stable in all patients with a normal renal function at the time of surgery. 2) Long-term prognosis was good in those children with a moderate decrease in GFR (less than 40%), when surgical treatment was performed during the first year of life. 3) A progressive deterioration of renal function was observed in patients with a marked decrease in GFR (more than 40%) at the time of surgery, and/or when surgical treatment was performed after 12 months of life.

L.A.H. MONNENS, P.M.V. VAN WIERINGEN\* and M.C.J. DE JONG\*. Pediatric Department, University of Nijmegen, The Netherlands. The haemolytic-uraemic syndrome (H.U.S.): follow up of the renal function.

Follow up studies will be presented of the renal function in 46 children with H.U.S. treated with streptokinase. Bloodpressure and renal function (urinary deposit, proteinuria, concentration test, hydrogen ion loading test and clearance of creatinine) were measured 6 - 9 months (46 pat.), 2 years (27 pat.) and 5 years (11 pat.) after the acute phase. Some data are also collected 4 - 6 weeks after the onset of the acute period. After 2 years 2 children had a clearance of creatinine \$\leq 80 \text{ ml/min/1.73 S.A., 3 children had a mild elevation of arterial bloodpressure.}

N.McINTOSH\*, J.C.L.Shaw & A.Taghizadeh. Paediatric Dept., University College Hospital, London.

Direct evidence for calcium and trace mineral deficits in the skeleton of preterm infants

To document quantitatively the way bone growth of preterm infants differs from intrauterine growth, the right femurs of fresh stillbirths and neonatal and late postnatal deaths have been analysed for calcium, magnesium, copper and zinc by atomic absorption spectrophotometry. These analyses show that the intrauterine accumulation of these elements between 22 and 40 weeks gestation can be described by a simple exponential equation. Analysis of bones from infants dying within the first 7 days are not materially different from those of infants dying within 24 hours of birth but later neonatal deaths show significant deficits in each substance when compared with infants dying in the first week. This trend confirms the findings of earlier balance studies showing inadequate absorption of calcium and also raises the possibility of magnesium, copper and zinc deficiency in preterm infants.

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B.Krempien, O.Mehls and E.Ritz (Intr. by G.Gilli). Department of Pathology, Pediatrics and Internal Medicine, University of Heidelberg, GFR. Micromorphometric and microradiographic studies of the growth apparatus of normal and diseased children.

Epiphyseal plates (upper and distal femur, tibia) of loo children from 0-15 years were studied in undecalcified sections. The children came to autopsy after sudden death without immobilization or after immobilization due to disease of various duration. Micromorphometric data were obtained by quantitative measurements of the epiphyseal cartilage and the metaphyseal spongiosa (parameters after SCHENK: volumetric density, surface density, specific surface, surface fraction of osteoid, osteoblasts and osteoclasts). Mineralization of cartilage and of spongiosal trabeculae was revealed by microradiography. Our data document that duration of disease as well as kind of disease (renal failure, cardiac failure, tumor, leukemia, liver disease, malabsorption, infectious disease) have profound effects on longitudinal growth (growth arrest), on bone density, cellular activity and on the vascularization of the growth apparatus.