gated. 8 had proven extrahepatic biliary atresia and the others had intrahepatic obstructive liver disease of undetermined aetiology since early infancy.

A five-day fat balance showed that 11 patients had steatorrhoea. The mean fat excretion was $35 \pm 10\%$ of dietary intake. Hence about two-thirds of the ingested fat was absorbed. All of these had obstructive jaundice and 8 were on or below the 10th centile for length and weight. By contrast, 3 of the 4 patients who absorbed fat normally were above the 50th centile. 2 of these had normal bilirubin concentrations.

Proximal intestinal contents were aspirated after ingestion of a standard test meal which contained polyethylene glycol (PEG) as unabsorbable marker. Luminal concentrations of bile salts, lipid, and PEG were measured and, after ultracentrifugation, also the proportion of the lipid present in the aqueous phase of the aspirate.

The mean bile salt concentration was significantly less in those patients with steatorrhoea ($P < 0.005$). Indeed bile salt concentrations above the critical micellar concentration were found in only 3 of the 4 children whose fat absorption was normal. No significant difference was shown in mean PEG or lipid concentration between patients with steatorrhoea and those with normal fat absorption. Patients with steatorrhoea, however, solubilized significantly less fat during each of the four 30-minute collection periods than those who absorbed fat normally ($P < 0.005$). A positive correlation exists between the concentration of luminal bile salts and the proportion of dietary lipid solubilized ($r = +0.8$).

These studies support earlier investigations in adult patients and highlight the close relation between the intestinal bile salt concentration and fat absorption. It is pointed out, however, that a proportion of dietary fat is absorbed even when bile salts are lacking.

One hour blood D-xylose as a screening test for malabsorption in infants and young children.

C. J. Rolles and M. J. Kendall (*introduced by P. N. Rayner*). (*Institute of Child Health, Francis Road, Birmingham 16.*) A single blood xylose estimation one hour after an oral dose of 5 g has proved to be a good guide to upper gastrointestinal absorption. 40 control subjects with no evidence of gastrointestinal disorder had a blood xylose level of over 25 mg/100 ml, whereas 9 untreated coeliac patients matched for age had levels below 16 mg/100 ml.

When repeated daily or weekly under standard conditions, the results were consistent in any given patient.

Untreated coeliac patients put on a gluten-free diet all showed a rise in xylose absorption within a few days. A treated coeliac given gluten for only one day showed a marked drop in xylose absorption—this reverted to normal when continuing the gluten-free diet. The use of this test called the 'gluten provocation test' had also proved to be of value in making a retrospective diagnosis of coeliac disease in a child put on a gluten-free diet in the past without a definitive biopsy.

Absorption of calcium by premature infants using a stable isotope. D. Barltrop and A. Sutton. (*St. Mary's Hospital Medical School, London W.2.*) Tracer investigations can be done in human infants or children without exposure of the subject to ionizing radiation. This paper reports the first application of stable ^{46}Ca as a marker for the measurement of calcium absorbed in bottle-fed newborn premature infants. A trace amount of calcium enriched in ^{46}Ca is administered to the infant as a solution of the chloride mixed with a normal feed. After the feed, urine and faeces are separately collected and specimens of blood obtained. The ^{46}Ca content of the samples is estimated by means of neutron activation analysis. The results obtained with this technique have been compared with conventional metabolic balance studies.

Effect of diet on water intake and urinary solute concentrations in infants. L. S. Taitz. (*Department of Child Health, Children's Hospital, Western Bank, Sheffield S10 2TH.*)

Circadian variation in plasma 17-hydroxyprogesterone in patients with congenital adrenal hyperplasia. Shelia M. Atherden, N. D. Barnes, and D. B. Grant (*introduced by June K. Lloyd*). (*Division of Infant Development, Clinical Research Centre, Watford Road, Harrow, Middx.*) (Page 602 of this issue.)

Defective aldosterone synthesis: 18-hydroxylase defect. Anne E. McCandless and William Hamilton. (*Royal Hospital for Sick Children, Yorkhill, Glasgow.*)

Studies on hydronephrosis. M. H. Winterborn (*introduced by R. H. White*). (*Children's Hospital, Birmingham.*) Papillary necrosis is rarely diagnosed in human hydronephrosis and then only in association with acute infection. On the other hand, animal experiments, particularly those of Hodson and his colleagues with the pig, have suggested that this complication may commonly cause the anatomical changes of hydronephrosis.

In the course of a retrospective study of hydronephrosis in children's kidneys at the Queen Elizabeth Hospital for Children, Hackney, an attempt was made to discover the frequency of papillary necrosis. The methods used were naked eye inspection, microdissection and counting of the number of ducts opening into each minor calyx using the dissecting microscope. Papillary necrosis was thought to have occurred in 3 out of 63 kidneys but was apparent to the naked eye in only one. There was good evidence that all three kidneys had been infected. With increasingly severe hydronephrosis there is a tendency for the duct count to rise and for the openings to become scattered over the surfaces of the papillae. This is interpreted as evidence of distortion of the kidney and it is suggested that 'back pressure distortion' rather than obstructive atrophy would be a more accurate, if less euphonious descriptive term for the radiological changes of hydronephrosis.