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 ± 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 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. Bartrup 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 46Ca as a marker for the measurement of calcium absorbed in bottle-fed newborn premature infants. A trace amount of calcium enriched in 46Ca 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 46Ca 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.
Neonatal bacteriuria and 'Uriglox'. S. Dosa and I. B. Houston. *(Department of Child Health, St. Mary's Hospital, Manchester.)* 'Uriglox' is a paper strip technique intended to detect bacteriuria by showing the consumption of glucose normally found in urine (by the metabolically active bacteria). Problems were foreseen in its application to the diagnosis of bacteriuria in the newborn and a trial was designed to test its reliability.

423 newborn babies 3 to 10 days old were studied; 65 (15.1%) were found to have >10^5 bacteria/ml urine at the initial culture but further repetition, culminating in suprapubic bladder aspiration showed that none had a true bacteriuria. In 63 of these 65 contaminated specimens, the Uriglox test was normal but overall 4 out of 423 tests were abnormal, a false positive rate of 0.9%.

For comparison, urine specimens were obtained by suprapubic aspiration from 27 infants suspected of having bacteriuria (on the basis of earlier urine cultures using per-urethral collections). 19 specimens were sterile, 8 specimens contained >10^3 bacteria/ml and 5 of these also had an abnormal Uriglox test. This gave a false negative rate of 3 in 8.

More than 100 leucocytes/µl urine were found in 3.3% of the normal urine specimens and in 5 of the 8 bacteriuric samples; one urine specimen with true bacteriuria contained less than 10 leucocytes/µl.

We conclude that, in the circumstances of this study, the proportion of false negative results is too high to justify the use of Uriglox for screening babies for bacteriuria. The low incidence of bacteriuria in the newborn group studied is also worthy of further comment.

Response to glucagon in small-for-dates hypoglycaemic newborn infants. Marthe A. Le Dune *(introduced by G. Arnett). (Department of Child Health, Royal Hospital for Sick Children, Glasgow.)* To be published in full in the *Archives.*

Study of immunoreactive pancreatic glucagon in the newborn period. D. I. Johnston and S. R. Bloom *(introduced by Alexander Mowat). (Department of Child Health, King's College Hospital, London.)* Pancreatic glucagon (PG) stimulates hepatic glycogenolysis, lipolysis, and may induce and activate rate-limiting steps in gluconeogenesis. These functions suggest that this hormone is relevant to the homeostasis of the newborn. Until now, methods for PG measurement have been too crude to evaluate its role in this age group.

A sensitive immunoassay for PG has been developed. The design of the assay follows the principles suggested by Albano and Ekins (1970). Using 100 µl plasma it can detect changes of 25 pg/ml within 95% confidence limits. Cross-reaction with glucagon of gut origin is avoided by the use of a specific antiserum.

PG was measured in maternal and cord blood in over 80 deliveries. Labour caused a rise in maternal PG. In 50 normal deliveries the mean difference between maternal and cord values was not significant. In 20 deliveries with evident fetal distress (scalp pH < 7.20) the mean cord value was significantly greater than the maternal level.

At 2 hours after delivery the peripheral venous PG of premature and small-for-dates infants showed a significant rise over the cord value. The rise in normal term infants was less significant. All infants had higher levels at 2 hours than their mothers.

This study indicates that the infant is capable of autonomous PG production at delivery. There is no evidence of impaired secretion in very premature infants or in SFD infants. PG appears to be produced in response to the metabolic demands of fetal distress.

Reference


Clinical pharmacology of gentamycin in the newborn. R. D. G. Milner, Julia Ross, and D. J. R. Proud. *(Department of Child Health, University of Manchester, and Roussel Laboratories Ltd., Swindon.)*

Use of the 'Gregory box' (CPAP) in treatment of RDS of the newborn: preliminary report. P. M. Dunn, M. J. Thearle, A. C. Parsons, and J. L. Watts. *(University of Bristol, Department of Child Health, Southmead Hospital, Bristol.)* Between October 1971 and January 1972, continuous positive airway pressure (CPAP) with the aid of a Gregory box (Gregory et al., 1971) was used by us in the treatment of severe respiratory distress syndrome of the newborn (RDS) on 6 occasions. The apparatus we used to administer CPAP (Dunn et al., 1971) and to monitor and control the pressure is briefly described.

Our early clinical experience may be summarized as follows. 4 infants treated with CPAP improved dramatically. Their mean gestational age was 31 weeks and birth weight 1930 g. Two of the mothers had had abruptio placenta. All developed uncomplicated RDS. Reporting mean values only, treatment was begun at 5 hours when the arterial blood pH was 7.07 and oxygen tension 53 mmHg in 37% oxygen. Starting CPAP with 6 mmHg, while maintaining the ambient oxygen unchanged led to an 89% rise in arterial oxygen tension to 100 mmHg. Treatment was maintained on average for 64 hours (range 46 to 93). All 4 survived.

The remaining 2 infants, both of 34 weeks’ gestation, were also born to mothers with abruptio placenta. Both developed RDS complicated by polycythemia and repeated apnoeic attacks. Though both responded to dilution exchange transfusion with plasma and to CPAP, apnoeic attacks continued. One infant required artificial ventilation after 4 hours of CPAP and survived. A second infant, with an initial pH of 6.92, failed to recover from an apnoeic attack after 5 hours of CPAP and necropsy revealed a large intraventricular