Comparisons of phenobarbitone, magnesium sulphate, and calcium gluconate in treatment of neonatal hypocalcaemic convulsions. T. Turner introduced by F. Cockburn. Paediatric Department, Simpson Memorial Maternity Pavilion, Royal Infirmary, Edinburgh.

Randomized treatment with calcium gluconate (Ca) 10 ml 10% solution with each feed (22 infants), phenobarbitone (Phe) 7·5 mg 6-hourly orally or intramuscularly (19 infants), or magnesium sulphate (Mg) 50% solution 0·2 ml/kg intramuscularly on two or three occasions at 12-hourly intervals (14 infants) was given to 55 artificially-fed term infants having convulsions associated with hypocalcaemia on the 4th to 7th day of life. Treatment was discontinued after 48 hours in each of the three groups.

Pretreatment plasma calcium concentrations were −2 SD below the mean for breast-fed infants in all groups but were significantly greater in the Mg-treated group when compared with the Ca-treated group ($P = 0·005$). There were no other significant biochemical differences found before treatment, and the number of pretreatment convulsions did not differ between the groups.

There were no significant differences between the mean numbers of convulsions in the first 48 hours after the start of treatment ($4·85 \pm 5·9$ SD in the Mg-treated group, $9·36 \pm 9·3$ SD in the Ca-treated group, and $9·36 \pm 10·9$ SD in the Phe-treated group). The number of infants still convulsing 48 hours after the start of Mg therapy was significantly lower than the number convulsing after Ca therapy (Mg:Ca $P < 0·05$; Mg:Phe $P > 0·1$; Ca:Phe $P > 0·3$).

After 48 hours' treatment the following biochemical differences were noted. Post-treatment plasma calcium concentrations were significantly greater in the Mg-treated group (Mg:Ca $P < 0·005$; Mg:Phe $P < 0·01$). Post-treatment plasma phosphate values were significantly lower in the Phe-treated group (Phe:Mg $P < 0·01$; Phe:Ca $P < 0·025$). Post-treatment plasma magnesium concentrations were significantly greater in the Mg-treated group (Mg:Ca $P < 0·001$; Mg:Phe $P < 0·001$).

Although prevention of convulsions associated with neonatal hypocalcaemia is better than cure, treatment of the established case with intramuscular magnesium sulphate is more effective than treatment with oral calcium gluconate, and probably more effective than treatment with phenobarbitone.

Secretin cells in childhood coeliac disease. Mary A. Rossiter and Susan Van Noorden introduced by B. A. Wharton. Queen Elizabeth Hospital for Children, London E.2, and the Royal Postgraduate Medical School, London W.12.

Although there has been considerable investigation of the absorptive and immunological functions of the small intestine in coeliac disease, less attention has been paid to the activity of the hormone-secreting 'Apud' cells.

Jejunal biopsy specimens from 17 children with untreated coeliac disease and 22 children with normal intestinal mucosa were examined for the presence of 'S' cells. These cells are thought to produce secretin, and can be identified by immunofluorescence and by electron microscopy, if they contain hormone (Polak *et al.*, 1971). The results are shown in the Table. Most of the jejunal biopsies from children with coeliac disease contained 'S' cells, whereas no cells were seen in the majority of the 'normals'.

TABLE

Occurrence of 'S' cells (by immunofluorescence) in jejunal biopsies from children with coeliac disease

Jejunal morphology	No. of jejunal biopsies containing 'S' cells		
	Large no. of cells	Few cells	No cells
Subtotal villous atrophy due to coeliac disease	10	2	5
'Normal'	1	5	16

$P < 0·001$, χ^2 test.

The increase in hormone-containing cells in cases of coeliac disease was also shown by ultrastructural and cytochemical techniques, which appeared to show actual hyperplasia of the 'S' cells.

It seems that secretin (and possibly other intestinal hormones, too) is not released normally from the Apud cells in childhood coeliac disease. This would affect intraluminal digestion and the metabolic rate of food substances after absorption.

REFERENCE

Polak, J. M., Bloom, S., Coulling, I., and Pearse, A. G. E. (1971). Immunofluorescent localization of secretin in the canine duodenum. *Gut*, **12**, 605.

Limitations of xylose tolerance test as screening procedure for coeliac disease. S. P. Lamabadusuriya, S. Packer, and J. T. Harries. The Hospital for Sick Children, Great Ormond Street, and Institute of Child Health, London W.C.1.

The xylose tolerance test is widely used as a screening test for coeliac disease, and a recent communication (C. J. Rolles, B.P.A. Annual Meeting, 1973) reported that a modified version of the test (blood xylose concentration 1 hour after the oral load) was extremely useful in predicting patients who were subsequently proven to have coeliac disease.

We have compared urinary excretion (5- and 24-hour collections) and blood concentrations (1- and 3-hour samples) of xylose after an oral load (0·4 g/kg of D-xylose) in 47 children who underwent jejunal biopsy for suspected coeliac disease. The children fell into one of 3 groups. (A) Normal biopsy (27 patients); (B) abnormal biopsy due to coeliac disease (19 patients); (C) abnormal biopsy due to other causes (5 patients).

5- and 24-hour urinary xylose excretion, and 3-hour blood xylose levels were of no value in discriminating between the 3 groups.

The mean and (range) of 1-hour blood xylose levels (mg/100 ml) were 37·3 (22·5–54·5) group A, 21·5