3 Khoury MJ, Erickson JD, Adams MJ Jr. Trends in postneonatal mortality in the United States. 1962 through 1978. *JAMA* 1984;**252**:367–72.
4 Ford RPK. Postneonatal mortality in Christchurch. *NZ Med J* 1986;**99**:939–41.
5 Taylor EM, Emery JL. Trends in unexpected infant deaths in Sheffield. *Lancet* 1988;**ii**:1121–3.
6 Irgens LM, Skjaeven R, Lie RT. Secular trends of sudden infant death syndrome and other causes of post perinatal mortality in Norwegian birth cohorts. *Acta Paediatr Scand* 1989;**78**:228–32.
7 Erickson JD, Bjerkedal T. Fetal and infant mortality in Norway and the United States. *JAMA* 1982;**247**:987–91.
8 Wallace HM, Goldstein H, Ericson A. Comparison of infant mortality in the United States and Sweden. *Clin Pediatr (Phila)* 1982;**21**:156–62.
9 Nelson EAS. *Sudden infant death syndrome*. Otago: University of Otago, 1989. (MD thesis.)

Forty years ago

Coeliac disease

Forty years ago the relationship between coeliac disease and gluten had not yet been established. In a lecture given at the University of Birmingham in May 1948 Alan Brown, Professor of Paediatrics in Toronto, discussed the then current theories of the aetiology of coeliac disease.[1] He had found a higher incidence of coeliac disease (and of diabetes mellitus) in the first degree relatives of patients with the disease than in the population as a whole. Two thirds of the children with coeliac disease had never been breast fed; in those who had been breast fed the onset of symptoms was on average six months later. Brown also referred to evidence that fat was absorbed as either fatty acids, which reached the liver via the portal vein, or small particles passed through the thoracic duct to the blood. The cause of the impairment of fat absorption in coeliac disease remained unknown but the roles of infection, gastrointestinal allergy and vitamin B complex deficiency were all considered worthy of further investigation.

Sheldon postulated that intolerance of starch might have a primary aetiological role in the disease with failure of fat absorption as a secondary effect.[2] He therefore planned an investigation to compare the degree of fat absorption in children with coeliac disease on two consecutive diets, one containing starch, the other starch free. In the absence of information from jejunal biopsies the diagnosis of coeliac disease was established in children with a typical history of steatorrhoea by a negative tuberculin test, the presence of normal amounts of trypsin in the duodenal juice, the absence of parasites in the stools, and a flat oral glucose tolerance curve.

Fifteen children aged from 1 to 6 years were studied. In nine the diet containing starch was given for varying periods, with a minimum of six days, followed by periods of at least seven days on a starch free diet. In the other six children the order of the diets was reversed. Both diets contained normal amounts of fat. The starch free diet was made palatable by including, in place of wheat and other cereals, biscuits made with soya bean flour that contains no starch, the carbohydrate being in the form of dextrins. At the end of each period on a test diet 12 day fat balances were carried out in all but four of the children, in whom the balances were restricted to eight days.

There was an average rise of 15% in fat absorption after the withdrawal of starch from the diet and this was highly significant. In patients with steatorrhoea isolated faecal fat estimations were found to be unreliable indicators of fat absorption. Eight day fat balances were more reliable than four day balances and no less accurate than 12 day balances.

In a further study of children with coeliac disease the finely emulsified fat particles (chylomicrons) in the serum were counted at half hourly intervals for three to four hours after a meal containing fat to produce a chylomicron curve as a measure of particulate fat absorption.[3] In children with coeliac disease the chylomicron curves tended to be flat but returned towards normal as the children improved on a starch free diet. This finding led Sheldon to suggest that defective particulate fat absorption might explain the steatorrhoea in coeliac disease.

In an attempt to determine how dietary starch interfered with fat absorption Sheldon also studied the effect of raising the content of dextrin (described as the first product of starch digestion) to between 20 and 50% of the total carbohydrate in the starch free diets.[3] In three children with coeliac disease the high dextrin diets caused as much impairment of fat absorption as reintroducing starch. Sheldon did not, however, draw any conclusions from this finding.

In four children with steatorrhoea due to cystic fibrosis starch free diets did not improve fat absorption.

1 Brown A. Some etiological factors in the coeliac syndrome. *Arch Dis Child* 1949;**24**:99–106.
2 Sheldon W. Coeliac disease: a relation between dietary starch and fat absorption. *Arch Dis Child* 1949;**24**:81–7.
3 Sheldon W. Studies in coeliac disease: fat absorption. *Arch Dis Child* 1949;**24**:245–9.

A D M JACKSON