months. Physical examination revealed an underweight infant (7000 g) with somewhat distended abdomen. Bone age was 3 months $\pm$ 5 months. His stools were gross and frequent but he had no diarrhoea. Antibodies to gluten but not to milk or reticulins were detected. Milk antibodies appeared later in serum. IgG and IgA were normal for age, but IgM was zero and no IgM was detected on immunoelectrophoresis. Chest x-ray showed expansion of the anterior ends of the ribs. Steatorrhoea was heavy (89.2 mmol fat/24 hours). No parasites were found in faeces or duodenal juice. Small bowel biopsy revealed normal mucosa. Sweat electrolytes were repeatedly normal. The diagnosis of SDS was confirmed by the absence of trypsin activity in faeces, and by estimation of trypsin in duodenal juice, where it was almost absent even after stimulation. Bone x-rays were performed at age 15 months and dysostoses were noted in proximal femoral metaphyses. Liver enzymes showed elevated levels of aspartate aminotransferase (236 U/l) and alanine aminotransferase (429 U/l). He had no history of jaundice and no liver disease could be detected.

Now, at age 17 months the boy is slowly gaining weight (9130 g). He still has elevated liver enzymes (ASAT 190 U/l and ALAT 236 U/l); immunoglobulin IgG 6.8 g/l, IgA 0.3 g/l, and IgM 0.1 g/l. Neutrophil count was 1.3×10^9 /l, the lowest value observed.

The findings in this patient show the broad spectrum of SDS. The hepatic involvement caused some confusion until we read the report of Brueton *et al.* (1977). Although liver biopsy was not done in our patient, we believe that hepatic dysfunction is part of this syndrome. The dys-gammaglobulinaemias previously described have often involved the reduction of IgA but less often low levels of IgG or IgM (Brueton *et al.*, 1977). Our patient has shown at various times normal levels of IgG (6.0–10.0 g/l) and IgA (0.3–1.1 g/l), but IgM seems to be constantly

reduced. So far he has suffered from prolonged respiratory infection and otitis media. Selective IgM deficiency has been reported in association with several diseases (Ross and Thompson, 1976), and SDS is joining the list.

M. MÄKI, AIRA SORTO,
O. HÄLLSTRÖM and J. K. VISAKORPI
Departments of Pediatrics and Central Laboratory,
Tampere Central Hospital, Tampere, and
Institute of Clinical Sciences, University of Tampere,
Tampere, Finland

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Dr Brueton comments:

We were interested to hear of a further child with liver dysfunction in association with this syndrome. We should like to draw attention to a review of 20 patients presented by Aggett *et al.* at the British Paediatric Association meeting in March 1977 (*Archives*, 1977, **52**, 815). Serum transaminase levels were increased in 17 of these children and liver biopsies in 6 showed nonspecific fatty changes and mild fibrosis.

M. J. BRUETON
The Children's Hospital,
Ladywood Middleway,
Ladywood, Birmingham B16 8ET