The sampling tube, an open-ended polyethylene feeding catheter sterilised by the manufacturer, is filled with sterile distilled water. A 2.0% suspension of agar in distilled water is prepared and enough cochineal to colour the solution is added. This is autoclaved at 44N/10 min to dissolve and sterilise the agar. When the solution has cooled to 60°C an 8 cm length of the coloured agar solution is aspirated into the distal end of the tube. The colouring reagent helps when filling the tube. The tube is left for an hour at room temperature for the agar to gel and it can then be passed into the duodenum where its position is confirmed radiographically. The agar plug is then expelled by flushing the tube through with sterile distilled water using hydraulic pressure to dislodge the plug. This technique has been successfully used by us on many occasions to obtain samples of small intestinal juice for microbiological study from patients with gastrointestinal disorders.

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Recurrent meningitis in a child with combined IgA deficiency and splenic hypoplasia

Sir,

Referring to the paper by Thong et al.¹ I describe a girl who presented in a similar way, also with compromised immunity.

A 12-year-old girl presented with two episodes of meningitis and one of septicaemia over a 2-month period, despite adequate treatment each time. Pneumococci were isolated on each occasion, and each time the serotype was different. Her medical history included referrals with localised cutaneous scleroderma (morphoa) and recurrent upper respiratory tract infections, but there was no history of recurrent infections in the family.

The following investigations were done: serum immunoglobulins IgG 9.45 g/l (normal 11.24 ± 2.35), IgM 0.7 g/l (normal 0.79 ± 0.33), IgA none detected. CHS50 normal, C3 + C4 components of complement were slightly increased. Lymphocyte transformation by pokeweed mitogen and phytohaemagglutinin was suboptimal. Escherichia coli antibodies (haemagglutination) were detected at a dilution 1 in 2 (patient serum), 1 in 64 (normal serum). Nitroblue tetrazolium screening test was normal. No Howell-Jolly bodies could be seen in the blood but an ultrasound scan failed to detect the spleen. An isotope scan showed the presence of a very small spleen in the left hypochondrium. The liver appeared normal.

The patient described by Thong et al.¹ was unusual in having only one detectable abnormality in the immune system. Other reports have suggested that children who have more than one immunodeficiency—for example, a defect in both the classical and alternative pathways of complement²—that will be particularly vulnerable to recurrent meningitis or septicaemia.

In our patient it is tempting to speculate that the absence of IgA led to failure of immune exclusion with consequent severe bacteraemia. Clearance of circulating bacteria by macrophages in the hypoplastic spleen was likely to have been impaired. Activity of the alternate pathway of complement was probably reduced as occurs in the functional hyposplenism of sickle cell disease³ and after splenectomy⁴ where fulminant pneumococcal infection is also seen.

In cases of recurrent meningitis where a cerebrospinal fluid fistula cannot be demonstrated full immune investigation is required. Evidence of splenic hypoplasia should be sought in each case. With the use of ultrasound a normally sized and positioned spleen can be demonstrated in a non-invasive way, and confirmation obtained by isotope scan in cases where no convincing echoes are seen. It is stressed that the absence of Howell-Jolly bodies from the blood cannot be relied on to exclude a diagnosis of splenic hypoplasia.

I thank the Department of Immunology, East Birmingham Hospital, for help with this case.

References


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Intractable diarrhoea of infancy and latent otomastoiditis

Sir,

The article by Salazar de Sousa et al.⁷ prompts us to report our recent experience.

In 1980 13 infants under age 6 months were in hospital with prolonged and resistant diarrhoea of unknown origin. Each had already received some kind of antimicrobial
treatment, and this was followed by broad-spectrum antibiotics after admission. Otoscopy, stool, and blood cultures were repeatedly negative in each case. The infants were malnourished and required parenteral nutrition for between 6 and 23 days.

One baby died, and necropsy showed typhoid ulcers of the bowels. Investigation of the family showed *Salmonella typhimurium* in the stools of the symptomless mother. One other infant recovered and after treatment with antibiotics had been stopped *Salmonella enteritidis* was cultivated from the stools.

Three babies recovered after parenteral nutrition and a special diet, but the clinical condition of 8 babies became worse and bilateral antrotomy was performed despite normal otoscopies. In one baby unilateral mastoiditis was found; the other 7 had bilateral latent mastoiditis. Cultures from the ear cavities were negative in 6 cases but *Pseudomonas aeruginosa* and *Staphylococcus aureus* were identified in the others. After surgery the diarrhoea cleared in 2 to 5 days and there was rapid improvement and weight gain in all antrotomised infants.

In our cases the correlation between leucocyte counts and diarrhoea or clinical condition was not as unequivocal as was described by Salazar de Sousa *et al.*, and some of them did not fully correspond to the criteria of infantile intractable diarrhoea as laid down by Avery *et al.* However, our findings suggest that if other forms of treatment have failed antrotomy should be considered in the treatment of prolonged and resistant diarrhoea.

**References**


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**Editorial Committee**

Dr A J Barson, Professor H Bickel, Sir Cyril A Clarke, and Dr J A Walker-Smith recently completed their 5-year term as members of the Editorial Committee. We are grateful to them for their help. Professor A S McNeish, Dr M E Pembrey, and Dr George Rylance have joined the Editorial Committee.
Intractable diarrhoea of infancy and latent otomastoiditis.

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*Arch Dis Child* 1981 56: 486-487
doi: 10.1136/adc.56.6.486-a

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