readily established in most children by primary immunisation alone, and the prime benefit of reinforcement at school entry may be to prolong immunity through adolescence into adulthood. In the case of poliomyelitis, reinforcing immunisation was largely without effect, probably on account of exclusion by pre-existing antibody. Thus it might be argued that, except in the case of immunity to diphtheria, the benefits of reinforcing immunisation are chiefly the immunisation against tetanus and poliomyelitis of children who have inadvertently missed primary immunisation.

Extrapolation from the samples to the cohorts from which they were drawn provides estimates of the percentages of children who, by the criteria of this study, may be considered to have been adequately protected. Thus in the 1969 cohort, in which the best immunisation coverage was achieved (84% of the children were fully immunised and a further 14% had received primary immunisation, although not reinforcement), the estimate of those protected from tetanus was 98% and of those protected from diphtheria and all three types of poliomyelitis 85%. In view of the excellence of the protection from tetanus and the additional protection provided by the herd in the cases of the communicable infections, it seems unreasonable to suppose

that more rigorous implementation of an immunisation programme would provide anything other than marginal additional benefit.

We thank the children for the blood samples, and their parents for allowing them to participate; Mr L Youens and Mrs Vivienne Miller and staff of the child health service of the Avon AHA; the doctors of the Avon AHA for performing the venepunctures; Dr Suzanne Clarke, of the Bristol PHLS Laboratory, for separating the sera; and Miss Janet Bootman, Mrs Anne Johns, Miss Moira Melville, and Miss Johanna Watkins for skilled technical help.

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Campylobacter colitis

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Summary and conclusions

Eleven consecutive patients with diarrhoea from whose stools campylobacter were isolated were investigated by sigmoidoscopy and rectal biopsy. Eight had definite proctitis, and in seven biopsy specimens were abnormal with histological changes ranging from non-specific colitis to gross colitis with goblet-cell depletion and crypt-abscess formation. Nine of the patients passed blood in their stools, and in all but one abdominal pain was a feature of the illness.

Severe campylobacter colitis may be clinically, sigmoidoscopically, and histologically difficult to differentiate from ulcerative colitis and is a differential diagnosis in acute colitis.

Introduction

Campylobacter has only recently been recognised as a cause of diarrhoea. Skirrow¹ reported that this organism could be isolated by selective culture of the faeces of over 7% of unselected patients with diarrhoea but not from control samples from subjects without diarrhoea. Other studies²⁻⁸ have confirmed these findings. During 1977 1513 reports of the

isolation of campylobacter from the faeces of patients were received by the Communicable Disease Surveillance Centre (Public Health Laboratory Service), and out of 1336 cases analysed, 95% of the patients were suffering from diarrhoea.⁹ The clear correlation between symptomatic diarrhoea and the isolation of campylobacter leaves little doubt that it is potentially pathogenic, though "carrier states" probably exist.

The concept of colonic disease resulting from salmonella infection, which was previously regarded as causing "enteritis," has recently gained wide acceptance.^{10 11} In view of the close similarity between the clinical features of salmonella and campylobacter infections we carried out the present study to determine whether similar colonic changes occurred in patients with diarrhoea due to campylobacter.

Patients and methods

Eleven patients (three female, eight male) presented during a six-month period with a diarrhoeal illness and campylobacter in their faeces. They were aged 14-80 years (mean 46 years), and 10 warranted admission for inpatient management. Sigmoidoscopy was carried out in all these cases and rectal biopsy specimens taken; in eight cases this was done shortly after admission. Three patients who did not undergo sigmoidoscopy on admission were found to have campylobacter in their stools only after the diarrhoea had settled; sigmoidoscopy and biopsy were performed as soon as the diagnosis was made, so in these cases the results represent a resolving or resolved condition. A barium enema was thought to be justified in two patients. Filtrates of faecal suspensions were cultured at 43°C on Oxoid BA base No 2 with 7% lysed horse blood plus (final concentrations) vancomycin 10 µg/ml, polymixin B sulphate 2.5 IU/ml, and trimethoprim lactate 5 µg/ml, in an atmosphere of 5% oxygen, 10% carbon dioxide, and 85% hydrogen.

CLINICAL FEATURES

The patients presented with diarrhoea or abdominal pain, or both. In most cases the duration of diarrhoea before presentation was

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under seven days, but three patients had experienced recent diarrhoeal episodes, usually lasting three to seven days, which resolved only to recur. The longest total history of episodic diarrhoea was seven weeks. The stage of profuse diarrhoea lasted usually three to five days, then gradually subsided (seven cases), but in prolonged cases lasted 10-21 days. The diarrhoea was severe in several cases; over half of the patients passed fluid stools more than 10 times daily, and the two worst affected had bowel actions 20 times a day and were sometimes incontinent.

Blood was present in the stools in nine cases; in seven it had been noticed by the patient and in a further two was detected by the laboratory. Colicky abdominal pain was prominent in all but one patient and was considered by some to be their worst symptom. It sometimes preceded the onset of diarrhoea and in many cases was partly relieved by defecation. In three patients the pain persisted for two to three days after the diarrhoea had settled. Mild tenderness was present in either the left or right iliac fossa in five cases, and this resolved as the diarrhoea settled; its presence was unrelated to the severity of the symptoms. Only three patients had a temperature above 37.5°C, and only two had a white cell count higher than $10 \times 10^9/l$ ($10\,000/mm^3$). Eight of the patients experienced a comparatively short illness and were hospitalised for four to nine (mean six) days. The other three, aged 80, 14, and 72 years, required inpatient care for 13, 20, and 29 days respectively. The severity of their illness may be seen from the following case history.

Case report—A 14-year-old schoolboy presented with a two-day history of severe colicky abdominal pain, followed after 24 hours by the onset of profuse bloody diarrhoea with bowel actions 12 times daily. He was anorectic but had not vomited. On examination he was flushed and dehydrated with a pulse of 60 beats/min, blood pressure 130/80 mm Hg, and temperature 38.1°C. The abdomen was soft without tenderness. His haemoglobin concentration was 15.4 g/dl and white cell count $10.4 \times 10^9/l$ ($10\,400/mm^3$). Sigmoidoscopy showed the rectum to contain mucopus, and the mucosa was oedematous and granular and bled on contact. Rectal biopsy (fig 1) confirmed the inflammatory changes and was considered to be

compatible with a diagnosis of chronic ulcerative colitis. Four days after admission his symptoms were unchanged and a barium enema (fig 2) showed a pancolitis. He was treated with intravenous fluids and erythromycin 500 mg four times daily. He made a steady recovery and was symptom free 10 days later. Twenty days after admission sigmoidoscopic appearances were normal and he was discharged from hospital. A barium enema showed minimal abnormality at three months and no abnormality at six months.

FIG 2—Result of barium enema, showing pancolitis.

FIG 1—Rectal biopsy specimen, showing dense infiltrate of lymphocytes and plasma cells. Crypt abscess present near centre of specimen. (Haematoxylin and eosin. $\times 50$.)

Results

The table gives the sigmoidoscopic and biopsy findings. In three patients the histological changes were suggestive of ulcerative colitis (showing a predominance of chronic inflammatory cellular infiltrate and pronounced goblet-cell depletion). In one patient changes were typical of infective colitis,¹² whereas in three others the histological changes were not sufficiently characteristic for the diagnosis of either irritable bowel disease or infective colitis.

Discussion

In previous reports of campylobacter infections¹⁻⁹ the outstanding clinical features have been profuse watery diarrhoea and occasionally peritoneal irritation severe enough to warrant laparotomy. These features have suggested that in most cases the small bowel has been affected, and this was supported by the observation of ileitis in occasional patients who underwent

Clinical details of patients, and results of sigmoidoscopy and rectal biopsy

Sex and age	Abdominal pain	No of bowel actions/day	Blood in stools	Sigmoidoscopic appearance	Rectal biopsy
M14	+	12	+	Exudation, granularity, contact bleeding	Chronic ulcerative colitis, active phase
M22	+	10	+	Erythema, oedema	Non-specific colitis
M80	+	5	+	Oedema	Non-specific colitis
M30	+	20	+	Exudation, granularity, spontaneous bleeding	Chronic ulcerative colitis in active phase
M55	+	3	—	Exudation, granularity, contact bleeding	Infective colitis
F22	+	12	+	Exudation, granularity, oedema	Chronic ulcerative colitis, resolving phase
M72	+	8	+	Granularity, oedema	(Inadequate material)
F74	+	5	—	Normal*	Non-specific colitis*
M25	+	12	+	Normal*	Normal*
F52	+	5	+	Normal*	Normal*
M58	—	20	+	Granularity, oedema	Normal

*Carried out after diarrhoea resolved.

laparotomy¹ or were examined post mortem.^{13 14} An appreciable proportion of patients, however—14% in the Public Health Laboratory Service series⁹—had fresh blood, pus, or mucus in their stools, suggesting colorectal inflammation. The presence of proctitis in eight of the 11 patients in the present series suggested that colonic inflammation occurred commonly in those patients whose symptoms were severe enough to require hospital admission, and may have played an important part in producing the diarrhoea.

It is accepted that infection with salmonella, shigella, or amoeba should be included in the differential diagnosis of irritable bowel disease, and our findings indicate that campylobacter should be considered. The changes seen on sigmoidoscopy and with a barium enema have little value in differentiating campylobacter colitis from irritable bowel disease, and, moreover, the histology of rectal biopsy specimens may show changes of this disease, thus compounding the problem. The importance of comprehensive bacteriological studies in all patients presenting with bloody diarrhoea cannot be overemphasised. Patients who in the past were diagnosed as having irritable bowel disease on clinical, sigmoidoscopic,

and histological grounds may well have been suffering from unrecognised campylobacter colitis.

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SHORT REPORTS

Mercury battery ingestion

When a child presents in the casualty department having swallowed a foreign body, it is standard practice to leave this to pass naturally if it has reached the stomach.^{1 2} This report describes a case where such action would have been hazardous.

Case report

The patient, a 2-year-old boy, presented one hour after ingestion of a small camera battery. An x-ray film showed it to be in the fundus of the stomach. The parents were advised to observe him at home and sift the faeces for the battery. Twenty-four hours later he was brought back to the casualty department, having passed a black stool. He was physically well apart from an upper respiratory tract infection. Rectal examination showed a dark stool, typical of iron discoloration rather than melaena. A further x-ray film showed that the battery was in its previous position.

At this stage advice from the makers of the battery in question (an Ever Ready PX 625 dry cell) was sought. The contents were given as roughly 2 g of mercuric oxide interleaved with zinc amalgam in a stainless steel case. Their view was that this was unlikely to corrode swiftly, but we thought it advisable to make a test of the battery's solubility in view of the danger to the child if its contents should be released, since the estimated lethal dose of ionised mercuric salts is about 0.5-1 g.³ The parents had brought a similar battery to the hospital so this was tested by placing it in an N/20 solution of hydrochloric acid prepared by the pharmacy, with a pH of about 1.5. Some five hours later it was inspected and found to be discoloured and bubbling vigorously.

Operative removal was then undertaken, the battery being extracted through a gastrotomy. It was observed to be heavily corroded and, in fact,

fell in two during removal (figure). The question was then raised whether toxic ingestion of either iron or mercury had occurred. Estimation of the serum iron concentration showed that this was normal at 12 $\mu\text{mol/l}$. Blood was sent to Guy's Hospital Poisons Unit for estimation of its mercury concentration; the result, available the next day, was 10 $\mu\text{g/l}$ (normal < 15 $\mu\text{g/l}$). Meanwhile the child showed no sign of toxicity and his urine contained no mercury on testing with potassium iodide. He was allowed home on the sixth postoperative day.

Comment

Mercury batteries are probably an easily swallowed source of potentially fatal poisoning for children, especially since the small button-sized battery used in some cameras might be mistaken for a sweet. On the basis of this experience early operative, rather than conservative management, is indicated for this particular foreign body.

I thank Guy's Hospital Poisons Unit for their help and Mr D F L Watkin for permission to report the case under his care.

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Battery after five hours in N/20 hydrochloric acid. Right: similar battery on removal after 30 hours in the stomach.

Defective colour vision in diabetes: a hazard to management

Benedict's test for glycosuria and its modification Clinitest require adequate colour vision for correctly interpreting results. Defective colour vision in a diabetic patient was reportedly responsible for mistaken Clinitest readings with consequently impaired control.¹ We have therefore surveyed a diabetic clinic population to discover the extent of such colour vision deficiency and determine its possible effects on diabetic control.