Correspondence

Eosinophilic colitis

SIR,—When we read the report by Moore *et al* on eosinophilic gastroenteritis with isolated colonic involvement (*Gut* 1986; 27: 1219–22) we were reminded of a similar patient we have been following for five years.

In November 1981 a 12 year old girl was seen with an eight week history of crampy lower abdominal pain, diarrhoea, and intermittent rectal bleeding. She averaged four bowel movements per day and occasionally a bowel movement at night. The stool was watery, brown in colour, and malodorous. The bleeding occurred with bowel movements, was bright red in colour and was mixed in with the stool. It varied in quantity. She denied fever, chills, anorexia, nausea or vomiting. Past health was unremarkable and functional inquiry was normal. Physical examination, upper gastrointestinal series and follow through, and barium enema were normal. Colonoscopy to the mid-transverse colon revealed numerous haemorrhagic blebs 1–2 mm in diameter. Some were intact and looked like 'blood blisters', while others had ruptured. The intervening mucosa was endoscopically normal and the rectum was spared. Multiple biopsies were obtained and reported as showing moderately severe inflammation with a high proportion of inflammatory cells being eosinophils. The inflammation did not extend past the muscularis mucosa. Crypt abscesses were not present, but sporadic cryptitis was seen. An unusual feature was that there was separation of the superficial mucosal epithelium from the underlying lamina propria.

Stools were negative for ova and parasites and no enteric pathogens were identified. Scotch tape test for pinworm was negative. Mild iron deficiency anaemia was present. The white blood cell count was normal without eosinophilia. Serum immunoglobulins including IgE were normal. Skin tests using the prick technique against milk, eggs, and wheat were negative. Complement studies were normal. A mild product elimination diet was tried for one month without benefit. Hydrocortisone enemas (100 mg) were given nightly for one week with resolution of the bleeding and diarrhoea. Nine months later symptoms recurred. Investigation revealed the same colonoscopic and histologic features. Symptoms rapidly resolved with a one week course of oral prednisone 15 mg daily. The prednisone was tapered. In the subsequent four years she had intermittent rectal bleeding with bowel movements. The bleeding could be minimised by keeping the stools soft with a high fibre diet.

In September 1986 the patient underwent flexible sigmoidoscopy and biopsy. Again, 1–2 mm superficial vascular blebs were seen. The intervening mucosa was normal. Biopsies were obtained from both normal and abnormal areas. The endoscopically normal areas were also histologically normal, whereas the endoscopically abnormal areas showed a marked degree of inflammation with infiltration of eosinophils, plasma cells and lymphocytes.

Like Moore's patient, ours had gastrointestinal blood loss, iron deficiency anaemia, and an eosinophilic colitis without evidence of more extensive gut involvement. There are a number of differences, however, our patient had no history of atopy and her peripheral eosinophil count was normal. The colonoscopic appearances were distinctly different. Whereas Moore's patient had 'erythematous granular and friable mucosa', our patient had discrete tiny 'blood blisters' with normal intervening mucosa and sparing of the rectum. Two short courses of corticosteroids led to prompt clinical improvement. The cause of this peculiar and presumably rare condition remains obscure.

S SULLIVAN AND M TROSTER

*Departments of Medicine and Pathology,
Victoria Hospital,
London, Canada, N6A 4G5*

Bile acid secretion in cystic fibrosis

SIR,—It is pleasing to note that other workers have studied bile acid secretion and pancreatic function in cystic fibrosis; however, we must take exception to the implication by Weizman *et al*¹ that these studies have not been attempted before. Our previous work published a year ago in this journal² and as an abstract in *Gastroenterology* in 1984³ addressed the same area of research.

The main difference in the two studies is that our patients were considerably younger (mean age 6.6 years v 21.7 years). Consequently, our finding of normal bile acid secretion rates in response to cholecystokinin/secretin differs from the lowered secretion rates from the latest study in older children. Our paper clearly shows a subgroup of older children (mean age 9.2 years) who also had lowered bile acid secretion rates and serum bile acid evidence of liver disease.

It is pleasing to see that bile acid secretion rates in controls and older children obtained in our study by a high performance thin layer chromatography method are almost identical to the rates found by Weizman *et al* using an enzymatic bile acid method. The conclusion from the studies is that total bile acid secretion rates are normal in young cystic fibrosis patients and deteriorate with age, probably because