

stones or more than one stone. The paper fails to make any comment on symptom relief, however, and the surprising result of our lithotripsy trial<sup>1</sup> was that the symptoms were relieved, just as much as after cholecystectomy, within the first month of treatment, well before any stones had disappeared; and the pain relief for the whole year and the relief of many other symptoms did not depend on stone clearance: so we have to distinguish between success in terms of stone clearance and success in terms of symptom relief. Lithotripsy, for whatever reason seems a very cost effective way of doing the second, at least in the short term. We are at present following up our patients for longer periods to see if this symptom improvement is maintained.

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1 Nicholl JP, Brazier JE, Milner PC, Westlake L, Kohler B, Williams BT, *et al.* Randomised controlled trial of cost-effectiveness of lithotripsy and open cholecystectomy as treatments for gall bladder stones. *Lancet* 1992; **340**: 801–7.

#### Reply

EDITOR,—We fully agree with Johnson and Ross that during a lithotripsy trial most patients feel extremely comfortable. This is not because of the lithotripsy itself, however, but a result of the well known effect of bile acids on gall bladder contraction.<sup>1</sup> Although, the effect on gall bladder motility is still somewhat controversial,<sup>2,3</sup> most authors agree that when ursodeoxycholic acid is given, gall bladder fasting volumes are larger<sup>4,5</sup> and gall bladder emptying is reduced.<sup>3,5</sup> The proposed mechanism is the decreased release of cholecystokinin through negative feedback control by an increased amount of intra-duodenal bile acid.<sup>4</sup> The same effect was seen with chenodeoxycholic acid,<sup>3</sup> and even with taurocholate a decreased cholecystokinin release and gall bladder contraction was seen.<sup>6</sup> On the other hand, bile acid depletion by the concurrent administration of cholestyramine increased cholecystokinin release and gall bladder contraction.<sup>6,7</sup>

This is the reason why, also in our experience, most patients with symptomatic gall stone disease remain free of symptoms when oral chemolytic treatment is started.

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- 1 Maton PN, Murphy GM, Dowling RH. Ursodeoxycholic acid treatment of gallstones. Dose response study and possible mechanism of action. *Lancet* 1977; **i**: 1297–301.
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- 7 Koop I, Fellgiebel A, Koop H, Schafmayer A, Arnold R. Effect of cholestyramine on plasma cholecystokinin and pancreatic polypeptide levels and exocrine pancreatic secretion. *Eur J Clin Invest* 1988; **18**: 517–23.

#### Barrett's oesophagus and development of dysplasia and adenocarcinoma

EDITOR,—Iftikhar *et al* (*Gut* 1992; **33**: 1155–8) present results from a 15 year prospective study of endoscopic surveillance of 102 patients with columnar lined epithelium (for example, Barrett's oesophagus). The aim of the study was to identify any significant risk factors for the subsequent development of adenocarcinoma. Data are presented suggesting that the length of columnar lined oesophagus was considerably longer in patients with dysplasia. None of the patients with dysplasia had a columnar lined oesophagus of less than 8 cm. The authors conclude that the length of Barrett's oesophagus is a significant risk factor in the development of dysplasia and subsequent carcinoma and recommend intensive follow up of patients with Barrett's oesophagus greater than 8 cm in length.

The results and conclusions of the study are inappropriate given the exclusion of patients with less than 5 cm of circumferential Barrett's oesophagus. Adenocarcinoma has been reported in tongues or short segments of Barrett's oesophagus.<sup>1</sup> At least 32 per cent of a series of 28 resected specimens with adenocarcinomas centred in the oesophagus had a length of Barrett's less than 5 cm.<sup>2</sup> Additionally, adenocarcinomas occurring near the gastro-oesophageal junction may arise from small areas of specialised epithelium, which may be obliterated by the carcinoma or not discovered.

It would be inappropriate to ignore patients with the potential for dysplastic change when short segment Barrett's oesophagus is found at endoscopy. Systematic biopsies should be taken and subsequent follow up should not differ from those patients with longer segment Barrett's oesophagus unless appropriately conducted studies show a lesser risk of cancer.

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- 1 Schnell TG, Sontag SJ, Chejfec G. Adenocarcinoma arising in tongues or short segments of Barrett's esophagus. *Dig Dis Sci* 1992; **37**: 137–43.
- 2 Hamilton SK, Smith RL, Cameron JL. Prevalence and characteristics of Barrett's esophagus in patients with adenocarcinoma of the esophagus or esophagogastric junction. *Human Pathol* 1988; **19**: 942–8.

#### Reply

EDITOR,—Thank you for giving us the opportunity to reply to the comments by Drs Faris and Sampliner. They are concerned that we excluded patients with less than 5 cms of circumferential columnar lined oesophagus and point to a study of resection specimens, which suggests that a high proportion of

oesophageal adenocarcinoma arise in association with short segments of Barrett's oesophagus. We should like to make the following points.

Firstly, we adhered to the recognised criterion of at least 5 cms of columnar lined oesophagus as it is difficult to make a confident diagnosis of Barrett's oesophagus with shorter lengths especially in the presence of a hiatus hernia. Secondly, we do not argue that patients with short segment Barrett's oesophagus should not be kept under endoscopic surveillance; we have merely shown that those with long segment disease are at greater risk of dysplasia and warrant more intensive follow up. Thirdly, in the paper by Hamilton *et al*, the length of Barrett's oesophagus was measured on fixed resection specimens. This is extremely unreliable as it will certainly underestimate length as measured in vivo by the endoscopist.

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#### Screening and management of familial adenomatous polyposis

EDITOR,—Tait *et al* advocate annual colonoscopy as the ideal screening method for first degree relatives who carry the gene for familial adenomatous polyposis (FAP). (Correspondence on Letter to the Editor and reply *Gut* 1993; **34**: 576.) Bradburn and Rhodes on the other hand make the case for the selective use of colonoscopy, recommending it only for those at high risk for FAP but without obvious polyps (or microadenomas) by their late teens, and in those in whom prophylactic colectomy has been delayed. This selective approach is suggested to minimise the morbidity and mortality associated with colonoscopy.

We report on two cases of colonoscopic morbidity, which, though anecdotal, add 'meat to the bones' of the present discussion.

*Case 1:* A 12 year old son of a patient with FAP was found to have polyps at sigmoidoscopy. Histological examination showed these to be adenomas. At colonoscopy at 16 years, the bowel was perforated. A laparotomy was performed for peritonitis and a defunctioning colostomy was created; this was closed six months after laparotomy. He was referred to St Mark's Hospital after his father died from an upper gastrointestinal malignancy, but refused to attend for further hospital appointments or to have surgery, and is now under psychiatric counselling.

*Case 2:* A 15 year old son of a patient with FAP was confirmed to have FAP at sigmoidoscopy. Annual colonoscopies were then performed, and at the age of 19 years, 20 polyps (size 2 mm) were removed. After this procedure the patient became unwell. A laparotomy was performed and a pelvic abscess was drained. He was referred six months later to St Mark's Hospital for definitive surgery. At laparotomy a large mesenteric desmoid tumour (not apparent at the first operation) was found. Neither the colon nor the desmoid tumour was able to be removed.

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