LETTERS TO THE EDITOR

Mucus synthesis by the human gall bladder

EDITOR,—I was interested to read the papers by Rhodes and colleagues concerning mucus synthesis by the human gall bladder, its apparent inhibition by aspirin, and other non-steroidal anti-inflammatory drugs (NSAIDs), and the suggestion that aspirin might prevent cholesterol gall stone formation by this action (*Gut* 1992; **33**: 1109–12 and 1113–7). As gall bladder mucus glycoprotein (mucin) is a secretory product, however, estimates of mucin synthesis must take account of secreted mucin as well as tissue mucin concentrations. These papers have reported tissue concentrations only. This is curious, especially as it is the secreted component that is thought to play a part in cholesterol gall stone formation.[1] It cannot be assumed that secreted mucin necessarily reflects tissue concentrations (it ranges from 30–50% of total mucin synthesis in the prairie dog model).[1,2]

Although aspirin and other NSAIDs reduced mucin concentrations in gall bladder explants in vitro their effects on mucin secretion in vitro were not presented. In the in vivo trial performed by the authors, however, mucin concentrations in the gall bladder bile of patients treated with aspirin were no lower than those in controls. These papers have not shown an effect of aspirin or other NSAIDs on gall bladder mucin synthesis or secretion. The case remains unproved.

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1 Lee SP, LaMont JT, Carey MC. Role of gallbladder mucus hypersecretion in the evolution of cholesterol gallstones. Studies in the prairie dog. *J Clin Invest* 1981; **67**: 1712–23.
2 O'Leary DP, LaMorte WW, Scott TE, Booker ML, Stevenson J. Inhibition of prostaglandin synthesis fails to prevent gallbladder mucin hypersecretion in the cholesterol-fed prairie dog. *Gastroenterology* 1991; **101**: 812–20.

Reply

EDITOR,—We thank Mr O'Leary for his interest in our papers but find his comments a little difficult to follow. He is quite correct when he states that we did not measure secreted mucin in the supernatant of our culture plates and this would provide additional information about the actions of aspirin in vitro. His conclusion that these papers 'have not shown any effect of aspirin on gall bladder mucin synthesis' is, however, incorrect. Both in vitro and in vivo we were able to show consistent inhibition of mucin synthesis.

Aspirin and NSAIDs undoubtedly have significant actions on the human gall bladder. This is confirmed by epidemiological findings,[1] biochemical studies such as these and other work in humans,[2] and motility work.[3] Whether these actions are clinically or therapeutically significant does indeed remain open to question but no doubt will be answered through the pages of this and other journals over the next five years.

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1 Hood K, Gleeson D, Ruppin DC, Dowling RH. Prevention of gallstone recurrence by non-steroidal anti-inflammatory drugs. *Lancet* 1988; ii: 1223–5.
2 Broomfield PH, Chopra R, Sheinbaum RC, *et al.* Effects of ursodeoxcholic acid and aspirin on the formation of lithogenic bile and gallstones during loss of weight. *N Engl J Med* 1988; **319**: 1567–72.
3 O'Donnell LJD, Wilson P, Guest P, Catnach SM, McLean A, Wickham JEA, *et al.* Indomethacin and postprandial gallbladder emptying. *Lancet* 1992; i: 269–71.

Treatment of achalasia

EDITOR,—Mr Spencer in his editorial review (*Gut* 1993; **34**: 148-9) has given a somewhat biased surgeon's view of the treatment of achalasia, favouring cardiomyotomy over balloon dilatation. We, as physicians, would regard balloon dilatation as being so good that surgery is almost never required.

As Mr Spencer rightly points out, although the debate over the choice of treatment would ideally be solved by carrying out prospective randomised trials, this is difficult, given the rarity of the disease. In support of cardiomyotomy, he quotes the solitary prospective randomised study that has been carried out to date – that of Csendes *et al.*[1] Although this study reported excellent results in 95% of patients after myotomy, but in only 65% of patients after one to two dilatations, the data need to be viewed with caution. The dilatations in the study were carried out for only 10–20 seconds, repeated twice. This is a considerably shorter dilatation time than in series reporting better success rates. Furthermore, the atropine premedication favoured by Csendes may have relaxed the lower oesophageal sphincter, thus rendering the dilatations less effective.[2] These factors may account for the poor success rate after dilatation in this study.

We carry out balloon dilatations for a three minute period, and in our published series of 66 patients,[3] 98% reported an immediate and appreciable improvement in symptoms after their initial dilatation. Only two patients developed a perforation (3%), both successfully managed conservatively, and three (4·5%) developed gastro-oesophageal reflux. Fifty eight patients were followed up for 1–12 years (median 55 months), and 91% of these remained dysphagia free after only one to two dilatations; a success rate comparable with that of Csendes' surgical series. Reflux rates are higher after cardiomyotomy in most series despite antireflux procedures, which may indeed sometimes recreate dysphagia. Moreover six days in hospital is required for cardiomyotomy, compared with only 24 hours for balloon dilatation. Lastly age, general frailty, and concomitant serious cardiorespiratory disease need not bar patients from selection for balloon dilatation.

We are now firmly of the opinion that surgery has little to offer in most cases of achalasia. Surgery for achalasia has not so much become unfashionable, as undesirable.

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1 Csendes A, Braghetto I, Henriquez A, Cortes C. Late results of a prospective randomised study comparing forceful dilatation and oesophagomyotomy in patients with achalasia. *Gut* 1989; **30**: 299–304.
2 Vantrappen G, Janssens J. To dilate or to operate? That is the question. *Gut* 1983; **24**: 1013–9.
3 Banerjee S, Pugh S, Smith PM. Pneumatic balloon dilatation in achalasia: A long term follow up. *Gullet* 1992; **2**: 28–32.

Reply

EDITOR,—I have read with interest the comments by Drs Banerjee, Pugh, and Smith on my editorial review. They agree with me that in so rare a disease it is difficult to obtain controlled data, but this seems a poor reason to reject out of hand the only controlled data available, and then accuse me of bias.

I had only two significant points to make. Firstly, that available data support rather than reject surgical treatment. The response to most trials that give results of which we disapprove is to say that those participating in the trial were doing it all wrong anyway. But is there controlled evidence that a longer dilatation gives better results than a shorter one? Or that anti-cholinergics decrease the effectiveness of dilatation? This in particular would be difficult to imagine, let alone prove. It is clear that excellent results may be obtained by dilatation, but with widely different complication rates, perforation occurring in 1–12%.[1,2]

My second point was that this scene is changing. In many centres laparoscopic (or thoracoscopic) cardiomyotomy is replacing open operation. It offers precise treatment. Radiological studies reported by Dr Smith and his colleagues, show that dilatation produces considerable tearing of the cardia; in an unpredictable few this tear leads to clinical perforation.[3] Precisely controlled myotomy can be performed at laparoscopy under excellent visualisation, and is tolerated by patients extremely well. Minimally invasive treatment no longer needs to be blind treatment.

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1 Vantrappen G, Janssens J. To dilate or operate? That is the question. *Gut* 1983; **24**: 1013–9.
2 Sauer L, Pellegrini CA, Way LW. The treatment of achalasia. A current perspective. *Arch Surg* 1989; **124**: 929–31.
3 Adams H, Roberts GM, Smith PM. Oesophageal tears during pneumatic balloon dilatation for the treatment of achalasia. *Clin Radiol* 1989; **40**: 53–7.

Injection sclerotherapy in portal hypertension

EDITOR,—We read with interest the article by Dr Heaton and Dr Howard (*Gut* 1993; **34**: 7-10) because we are in the early stages of a prospective study of percutaneous transjugular intrahepatic portosystemic shunt (TIPS) in recurrent variceal bleed. While we acknowledge that the article was mainly directed at injection sclerotherapy, the authors discuss the role of emergency surgical procedures in the management of acute variceal bleeding after failed injection sclerotherapy, but have failed to include in their discussion any reference to TIPS. We believe that early results suggest that TIPS should be considered before surgery,