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. 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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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, biochemical studies such as these and other work in humans, and motility work.1 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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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 cardiomytomy 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 cardiomytomy, he quotes the solitary prospective randomised study that has been carried out to date—of Coenees 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 Coenees may have reduced 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 during a 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 Coenees' surgical series. Reflux rates are higher after cardiomytomy in most series despite antireflux procedures, which may indeed sometimes work. More recently, we have used omeprazole. Moreover, six days in hospital is required for cardiomytomy, compared with only 24 hours for balloon dilatation. Lastly, age, general frailty, and concomitant serious cardiopulmonary disease need not be 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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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.