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 unproven.

DANIEL O'LEARY
Department of Surgery, Southmead Hospital, Weston-super-Dep, Bristol BS10 5NB


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 undoubtably have significant actions on the human gall bladder. This is confirmed by epidemiological findings,1 biochemical studies such as those by other work in humans,2 and motility work.2 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.

M RHODES
Academic Department of Surgery, University Hospital of Wales, Heath Park, Cardiff


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 and aspirin, and 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 Csenges et al. 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 Csenges may have reduced the lower oesophageal sphincter, thus rendering the dilatations less effective.1 These factors may account for the poor success rate after dilatation in this study.

We carry out balloon dilations for a three minute period, and in a published series of 66 patients,1 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 Csenges' surgical series. Reflux rates are higher after cardiomyotomy in most series despite antireflux procedures, which may indeed sometimes fail. We, and others, have found that patients who have undergone myotomy or balloon dilatation have a variety of symptoms, including post-prandial heartburn, nausea, and chest tightness. More recently we have found that patients may develop a post-prandial acid reflux.1,2 We do not currently have data on reflux rates after balloon dilatation. However, we have developed a questionnaire which asks patients whether they have symptoms of heartburn, nausea or post-prandial tightness. This may prove useful in the future. We would welcome information from other centres on this and other aspects of balloon dilatation. It is clear from the data presented by Mr Spencer that balloon dilatation is a highly effective procedure, which can be performed with minimal complications. We believe that balloon dilatation is a safe and effective procedure for the treatment of achalasia.

J SPENCER
Department of Surgery, Royal Postgraduate Medical School, Hammersmith Hospital, Du Canewe Road, London W12 0NN


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 varical 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 varical 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.

BANERJEE S
PUGH P
SMITH P M
Department of Gastroenterology, Llandough Hospital, Cardiff CF6 2XX

Treatment of achalasia.

S Banerjee, S Pugh and P M Smith

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