Correspondence

Endoscopic treatment of bile duct stones: (apples and oranges) – and lychees?

SIR,—We read Dr Peter Cotton's well balanced editorial on endoscopic sphincterotomy for common bile duct stones with interest.¹ While we agree that there is no place for any surgical complacency with the nationwide incidence of retained stones remaining in the region of 10%, the benefit of operative choledochoscopy is perhaps less than fairly treated. He quotes Rattner² with an incidence of 6% and Feliciano³ with an incidence of 8.9% retained stones. Do these two series represent some lychees among his apples and oranges?

Rattner reported eight retained stones in 144 patients – 5.6, not 6%. In two of these patients, however, the stones were visualised with the choledochoscope but were not removed; the failure rate of choledochoscopy was thus six of 144 patients – 4.2%. Feliciano found seven patients with retained stones in 79 patients – 8.9%. We have recently reviewed the published literature on choledochoscopy, with clarification of the number of stone positive bile duct explorations by correspondence with the authors where necessary. In a total of 37 reports on operative choledochoscopy retained stones occurred in 99 of 3159 patients – less than 3.1%. Feliciano's report was one of only five papers in which the incidence of retained stones exceeded 5%.⁴

Few surgeons resent the contribution that endoscopic sphincterotomy has made to the care of patients with common bile duct stones and the early experience of dealing with symptomatic common duct stones endoscopically in patients with gall bladders *in situ* is of great interest. Although these patients may now represent 50% of patients undergoing endoscopic sphincterotomy they do, of course, represent only a proportion of the 12% patients with gall stone disease who have stones in the bile duct as well as the gall bladder.

We entirely agree that endoscopic treatment should be used more widely. The weight of evidence that has amassed in favour of choledochoscopy compared with conventional bile duct exploration indicates the pressing need for routine endoscopy – at the time of common bile duct exploration – rather than afterwards.

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Oesophageal stricture after endoscopic sclerotherapy

SIR,—We are alarmed at the very high incidence of oesophageal stricture after endoscopic sclerotherapy as reported by Dr Sorensen and his colleagues (*Gut* 1984; **25**: 473–7) and we cannot agree with their conclusions that 'stricture and dysphagia constitute a continuing and prominent clinical problem among patients with oesophageal varices treated by sclerosing injections'.

Over a four year period we have treated 104 patients with sclerotherapy without a single case of stricture and only eight cases of transient dysphagia. If we exclude 19 patients who died within one month, 85 patients had a mean number of 3.8 treatments (range 1–14) during a mean follow up period of 14 months (range two months to four years).

We inject intravariceally, however, and stop the injection when any blanching of the mucosa occurs. It is not surprising that the paravascular technique produces strictures as it has its effect on the varices by producing surrounding fibrosis. Intravariceal injection produces thrombosis of the vessels and the mucosa is affected only when inadvertent extravascular injection occurs.

Intravariceal injections can be made without giving a high risk of variceal rebleeding (our risk being 0.065 bleeds per patient month of follow up). As the authors suggest, a controlled trial might be indicated, but this would not seem justified if aggressive paravascular injections give such a high incidence of complications.

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Reply

SIR,—We agree with McCormack and Johnson that the incidence of dysphagia and oesophageal stricture

in our series was surprisingly high. Conversely, their experience appears unusually good, even if compared with the prospective trial reported by McDougall *et al*¹ who used a similar sclerosing technique. The major question is which techniques provide the best protection against bleeding. We are willing to pay fairly heavily in terms of non-lethal, treatable complications such as dysphagia and stricture for a lesser risk of rebleeding. This risk is greatly variable among patients with oesophageal varices. It is very important to obtain balance in the pretreatment risk between groups of patients compared with the efficacy of various techniques of sclerotherapy. We believe that this balance cannot be established except by using the closed randomisation procedure.

We would like also to comment on the possibly misleading manner of reporting the risk of rebleeding presented by McCormack and Johnson and by others.¹ By dividing the total number of rebleeds in a series of patients by the total number of patient-months of follow up, an undue weight is given to long term survivors with few, or no episodes. To avoid this dilution of the denominator, we suggest the calculation of the incidence for each patient (number of rebleeds per months) and having this as the basis of reporting. The average, or median – for specified intervals of time after the first bleeding – of these individual risk estimates with appropriate measures of between patient variation would in our view give a more adequate picture of the rebleeding.

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More about refractory duodenal ulcers

SIR,—I was interested to read, in the July 1984 issue of *Gut*, the papers by Pounder,¹ Lam *et al*,² and Bardhan³ on the problem of refractory duodenal ulcers. I would like to discuss some personal experiences in this field, further to a previous report⁴ that Dr Bardhan was kind enough to quote in his paper.³

Since submission of our abstract to the BSG in

December 1982, the number of refractory subjects has increased. To date 32 duodenal ulcers resistant to an eight week treatment with cimetidine 1 g daily (19 patients) or ranitidine 300 mg daily (13 patients) have been shifted to sucral phosphate therapy for six weeks.

The healing rate was 81.2%, which is approximately the same observed with the first 20 cases.⁴ The remaining six patients showed neither healing nor reduction in ulcer size even after treatment was changed.

The absence of a control group of subjects continuing treatment with H₂-blockers makes it impossible to definitely conclude that ulcer healing was because of the change of therapy rather than to the mere prolongation of treatment. It seems unlikely, however, that more than 80% of our subjects were just 'slow responders'. Indeed ulcers the size of which remained unchanged after eight weeks of therapy with H₂-blockers can hardly be expected to completely heal during the following six weeks of treatment to such an extent. By and large our study, although unfortunately uncontrolled, compares well with the results by Dr Lam *et al*² with tripotassium dicitrato bismuthate (the mode of action of which partially resembles that of sucral phosphate). This seems to confirm the view that in refractory duodenal ulcers mucosal protection may be more effective than control of gastric acidity.

As for the profile of patients who failed to respond to H₂-receptor antagonists, we were unable to detect any particular features regarding sex, age, disease history, and gastric secretory capacity. Contrary to Lam's suggestion² and in agreement with Bardhan,³ smoking habits did not appear to influence the lack of response to H₂-blockers. On the other hand, refractory ulcers appear to represent a subgroup of duodenal ulcers with a different pathophysiology.²

Our incapacity to recognise the factors involved emphasised once again how little we know of the disease we are treating. At the present time we are unable to predict which subjects may benefit from therapy with antiseecretory drugs or with agents strengthening mucosal defences. As long as we cannot discriminate between different types of ulcers, we should expect the usual 20% of error (treatment failure) in the choice of the drug. Hopefully the dilemma might be solved in the future by using prostaglandins with both antiseecretory and cytoprotective activities.

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