

## Correspondence

### Forceful dilatation and oesophagomyotomy in patients with achalasia

SIR,—We were interested to read the further report by Csendes and his colleagues on the comparison of oesophagomyotomy and forceful dilatation for achalasia,<sup>1</sup> but believe it would be regrettable if their conclusions that 'surgical treatment offers better results than forceful dilatation with the Mosher bag' led others to believe that all dilatation treatment of achalasia is inferior to cardiomyotomy. We strongly believe that their comparison has been inappropriate because of their use of a dilatation technique which appears unsatisfactory. It was criticised<sup>2</sup> when described in their earlier report<sup>3</sup> but unfortunately there are still several reservations: (1) There is no mention of radiological evidence of full dilatation of the bag, so presumably this was not achieved in every case. (2) The pressure exerted '5.4 pounds per square inch' is rather low and unlikely to be effective; we use 20 pounds per square inch. (3) The duration of balloon inflation under pressure is probably too short to be beneficial. We find that full dilatation may take up to 20 seconds to be reached; our practice is then to keep the balloon fully inflated under pressure for 60 seconds. Other groups use comparable duration and pressure in their dilatations.<sup>4</sup> (4) The use of atropine may relax the sphincter, making the dilatation less efficacious. (5) It was distressing to read that patients experienced pain and discomfort during the procedure which had to be cut short. This probably did not allow full dilatation to take place making the comparison with the surgical group invalid. Our patients receive 10–20 mg Diazemuls and 50–100 pethidine intravenously which offer amnesia and alleviation of pain.

We have used several balloon dilators in the past, but we have recently been most satisfied with the 30 mm Rigiflex dilator which is designed in a similar way to the Grunzig angioplasty catheter.<sup>5</sup> Using this balloon in 23 patients in the last two years we have had no perforation and blood streaks were seen only in two cases without any significant bleeding; this is in contrast with 100% record of blood observed in the Santiago paper. It is premature to report on longterm results of our series but at annual follow up the success rate remains above 90%.

We think that forceful dilatation in experienced hands is still an effective and safe first line choice of treatment for all patients with achalasia, and would undoubtedly be the choice for the elderly patient.

Patients who fail to respond to forceful dilatation can still be offered cardiomyotomy.<sup>3,6,7</sup>

M DAKKAK AND JOHN R BENNETT

Hull Royal Infirmary,  
Anlaby Road,  
Hull, HU3 2JZ

### References

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### Reply

SIR,—I am deeply grateful to Drs M Dakkak and John Bennett for their interesting comments. My answers to their questions are the following: (1) All dilatations are done under fluoroscopic control. Therefore, we checked the correct placement of the back and the full dilatation of it. (2) There must be some printing errors because we use between 12 to 15 pounds per square inch, which is the measure of the Mosher back. (3) We have not been able to keep the balloon inflated for 60 seconds because patients very quickly experience pain and discomfort. We have not used diazemuls or pethidine intravenously because they could mask perforations in patients who do not feel pain. (4) The use of 0.5 mg atropine does not relax the sphincter in this patient as we have shown in an unpublished study. (5) Our results can only be applied to the Mosher back. Therefore, we know clearly that there are several types of balloons and probably some of them give better results. Only a late follow up of a prospective randomised study such as ours could solve the question concerning which is the best treatment in patients with achalasia. We should add that surgery in patients with failed dilatation is significantly more difficult and dangerous.

We are very happy that our study has provoked such controversy as only with cooperative and careful

investigation will help us in the future.

ATTILA CSENDES

Department of Surgery,  
University Hospital,  
JJ Aguirre,  
Santos Dumont 999,  
Santiago, Chile

### Relapse rates after duodenal ulcer healing – apples or pears?

SIR, – The one year maintenance study reported by Bardhan *et al* (*Gut* 1988; **29**: 1748–54) showed Maalox TC, given in a dose of 3 tablets (81 mmol acid neutralising capacity) twice daily, to be as effective as cimetidine 400 mg nocte in the prevention of duodenal ulcer relapse; both these agents were significantly better than placebo. The relapse rate of only 57% in their placebo treated group, however, contrasts rather strikingly with the 75 to 90% relapse rates reported in most other studies; the relapse rate in their cimetidine treated group was also somewhat low. Their low relapse rates are more comparable with those of Sontag *et al* who noted one year relapse rates of 50% on placebo and 28% on cimetidine maintenance therapy. Dare one speculate on the discrepancy between the relatively low relapse rates in these two studies compared with most others?

There is good evidence that six to 12 month relapse rates after initial duodenal ulcer healing with a colloidal bismuth agent, or sucralfate, tend to be lower than those after healing with an H<sub>2</sub>-receptor blocker.<sup>2,3</sup> One year relapse rates after colloidal bismuth healing, however, are usually in excess of 60%. In any event the duodenal ulcers in over 90% of the patients in Bardhan *et al*'s study were healed on an H<sub>2</sub>-receptor blocker and it is probable that the same applies to those patients drafted into the Sontag study.

Attention has recently been focussed on the speed of relapse in patients on placebo after treatment with an ulcer healing agent. Most maintenance studies allow for routine endoscopies at six and 12 months and it is common cause that the majority of relapses occur during the first six months. A few studies allow for routine endoscopies at four, eight, and 12 months and, in these, relapses within the first four months account for well over 60% of the total number of relapses at one year. This applies particularly to patients after initial healing with an H<sub>2</sub>-receptor blocker. Lee *et al*,<sup>4</sup> in a one year study in patients after healing with ranitidine (n=54) or a colloidal bismuth preparation (n=53), reported that no fewer than 40 (83%) of the 48 relapses in the ranitidine healed group occurred within the first four months. This compared with 22 (67%) of the 33 relapses in

patients treated initially with colloidal bismuth. It should be stressed that the four month relapse rates in this study were 74 and 41% respectively.

More recent studies have confirmed the rapidity of early relapse in patients after healing with an H<sub>2</sub>-receptor blocker. In the first, ulcer healing was documented after six weeks treatment with either ranitidine or sucralfate in 32 duodenal ulcer patients. Active treatment was discontinued, and a routine endoscopy carried out four weeks later. An ulcer relapse was noted in 10 of 15 ranitidine healed and in three of 17 sucralfate healed patients.<sup>5</sup> Boyd *et al*,<sup>6</sup> on the other hand, carried out monthly endoscopies in 34 patients admitted to a maintenance ranitidine study immediately after duodenal ulcer healing by ranitidine. The cumulative relapse rate at one year was 48% with more than half of the first recurrences occurring within the initial two months. The majority of endoscopic recurrences, it would seem, develop within the first few months after duodenal ulcer healing.

It is not known whether the duodenal ulcers which relapse within one or two months of endoscopic healing occur in patients with a more aggressive form of the disease. What is clear, however, is that commencing a maintenance study a month or more after documented healing automatically excludes a substantial proportion of early relapsers. Most maintenance studies do in fact commence within a few days of endoscopic healing of a previously active ulcer. Neither Bardhan *et al* nor Sontag *et al* had recent ulcer healing as a criterion for entry into their maintenance studies. Bardhan *et al* studied 'patients with previous symptomatic endoscopy proven DU which had been shown endoscopically to have healed within the previous one year, provided they were asymptomatic and ulcer free at endoscopy done less than seven days before commencing (maintenance) treatment'. The mean time interval between healing of the last ulcer and entry into the study was 51 days. In similar vein, Sontag *et al* required their patients to have 'a history of duodenal or channel ulcer diagnosed by endoscopy or unequivocal x-ray findings within the previous two years, with at least one episode of recurrent characteristic ulcer symptoms during the year preceding entry. Endoscopy was performed at entry, and only patients with a normal duodenal mucosa were included'. It follows that both protocols would have resulted in the exclusion of a large proportion of patients with a tendency to early relapse, and that this probably accounts for the seemingly lower relapse rates in these studies.

The above comments should not be construed as a criticism of either of these studies. Both Bardhan *et al* and Sontag *et al* presented their entry criteria in