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Balloon dilatation of benign oesophageal strictures

SIR,—We read with interest the recent paper in your journal by Cox *et al* (*Gut* 1988; **29**: 1741-7). The authors report that both methods are equally safe, but believe that bougie dilatation is better in reducing the incidence of redilatation, and recommend that balloon dilatation should be reserved for very select cases. Both methods used, involve radiology, and the patient staying in hospital overnight.

We are currently involved in the prospective evaluation of transendoscopic balloon dilatation as an outpatient procedure, for benign peptic strictures. We use a Rigiflex TTS transendoscopic balloon catheter (supplied by Keymed, Essex). Strictures are dilated under direct vision and do not require x-ray control. Over the past 18 months, we have performed 51 dilatations on 41 patients. The mean age group was 77.6 years. All procedures have been performed on an outpatient basis. No complications have been encountered. We have found the procedure an effective method of relieving symptoms, with only 10 patients requiring redilatation. We find this a safe and cost effective way of managing a common problem in an elderly population.

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Benign oesophageal stricture in Barrett's oesophagus

SIR,—I enjoyed the paper by Atkinson and Robertson¹ which showed that patients with benign oesophageal stricture associated with Barrett's oesophagus do well with conservative management. Their study showed that only 11 of 23 patients (48%) required more than one dilatation during follow up. In a much smaller series of such patients included in a paper by Barbezat and myself, seven patients with Barrett's oesophagus and benign oesophageal

stricture were followed up and only three (42%) needed more than an initial dilatation.² I was interested that Atkinson and Robertson¹ found that 41% of their 56 patients with Barrett's oesophagus had a benign oesophageal stricture. In contrast, Barbezat and I found only 10 patients with benign oesophageal stricture (19%) in a series of 52 patients with Barrett's oesophagus from Dunedin, New Zealand. In other series listed in Atkinson and Robertson's paper, benign oesophageal stricture was reported in 31% to 81% of patients with Barrett's oesophagus. I think the discrepancy between our figures and the others may be explained by the selection of cases in the series. Many of the reported series with much higher figures for the prevalence of benign oesophageal stricture are surgical series; surgeons are likely to see patients with troublesome symptoms and/or complications of Barrett's oesophagus. Patients with Barrett's oesophagus but few or no symptoms and no complications, are not going to be referred for surgery. This type of bias can be seen in a previous series from Dunedin, which included all patients with Barrett's oesophagus referred to the thoracic surgical unit between 1952 and 1973. All 45 patients in the series had evidence of benign oesophageal stricture on barium swallow and 44 of the 45 patients complained of dysphagia.³ Similar bias may be present in series such as those of Atkinson and Robertson,¹ which report cases from centres with a high reputation in the management of oesophageal disease and who are likely to acquire patients with symptomatic or complicated Barrett's oesophagus. Barbezat and I found 52 patients with Barrett's oesophagus from among all patients endoscoped in a medical gastroenterology unit which provided all endoscopic services for the Dunedin area in a period from January 1981 to December 1986. Ten per cent of patients endoscoped with evidence of gastro-oesophageal reflux had Barrett's oesophagus; these figures are similar to those from other centres.^{4,5} A quarter of our patients with Barrett's oesophagus did not have symptoms related to the oesophagus. I submit that our series contained a broader and perhaps more representative spectrum of Barrett's oesophagus and in such series the numbers of patients with complications such as benign oesophageal stricture will be less. The latest figures from our own unit at Dudley Road Hospital support this view; 35 cases of Barrett's oesophagus have been diagnosed from among all patients undergoing endoscopy over the last two years and only four patients had benign oesophageal stricture. Barrett's oesophagus is common and endoscopists are becoming much more aware of it, therefore it is likely that many cases diagnosed in the future will be at an earlier stage and without complications.

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Reply

SIR, - We were interested to read Dr Cooper's letter and to see that his most recent experience confirmed his earlier findings that the prevalence of oesophageal stricture in Barrett's oesophagus is substantially lower than that of 30-80% previously recorded. Barrett's oesophagus represents the end stage of severe oesophageal damage and it is understandable that many have found associated oesophageal stricture to be much commoner than in reflux oesophagitis without Barrett's. It is generally recognised that particularly in the elderly, symptomatic disability and severity of oesophagitis do not show a close correlation and it may well be that the now widespread use of fiberoptic endoscopy in gastroenterological units is bringing to light gastric epithelialisation of the lower oesophagus in patients who some years ago might well never have been endoscoped. This might well explain the apparent discrepancy between Dr Cooper's figures and our own which go back for up to 12 years.

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Age related increase of brush border enzyme activities along the small intestine

SIR, - We should briefly like to comment on the recent paper by Raul and co-workers (*Gut* 1988; **29**: 1557-63), which describes studies in which small intestinal morphology and brush border hydrolase activity was measured in three, 12, and 29 mo Wistar

rats. Overall, our findings in the chow fed Fischer 344 rat, raised under barrier reared conditions by contract with the National Institute on Aging, are similar. We also found no change in proximal intestinal brush border hydrolase activity, but only in the rate of enzyme expression in mucosal epithelial cells.¹ Furthermore, we have confirmed our initial preliminary observations² that ileal villus height and cell number are greater in 27 mo than in the four to five mo rats,³ in agreement with Raul's data. We were puzzled, however, by the somewhat discrepant observations of a fall in proximal intestinal villus height and crypt depth coupled to an increase in gut cell mass and protein content. Initially, we wondered whether the reduction in proximal intestinal villus and crypt dimensions seen in senescent rats, in the study of Raul and coworkers, was the result of reduced food intake (quoted as 14-18 g v 20-25 g per day), but we are at a loss to understand the presence of an increased gut protein content at the same time. A fall in protein degradation is commonly seen in the organs of aging rodents,⁴ but this has not been confirmed in the small intestine.⁵ We look forward to further studies that can elucidate these most interesting observations.

We are sure, however, that both of our groups agree that these changes in brush border hydrolytic enzymes are unlikely to be of nutritional significance although they clearly may suggest some fundamental age associated changes in the gut similar to those that our group have described for proliferation of the small⁶ and large⁷ intestine. We are delighted to see another experimental group pursuing studies of the aging gut.

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