Surgery for uncomplicated gastro-oesophageal reflux

Editor,—The leading article by Mr Dehn on surgery for uncomplicated gastro-oesophageal reflux (Gut 1993; 33: 293–4) is particularly timely in view of the increasing availability of omeprazole and the continued efforts on several fronts to improve the results and reduce the invasiveness and incidence of side effects following surgery.

While omeprazole is undoubtedly the most effective agent currently available for the management of gastro-oesophageal reflux, its greatest efficacy is in Savary and Miller grades 1 and 2. Most patients coming to surgical treatment have grade 3 oesophagitis, in which group the healing rate on omeprazole treatment is 67%. Furthermore, recurrence of endoscopic oesophagitis after one year's continuous omeprazole treatment is up to 33% and even assuming a uniform relapse rate between grade 3 and grades 1 and 2 oesophagitis, this means that more than 50% of patients with grade 3 oesophagitis will remain unhealed on continuous omeprazole treatment.

We recently presented to the International Society for Disease of the Oesophagus a comparison between patients referred for anti-reflux surgery in the pre and post-omeprazole era. There has been little change in the overall numbers of patients being referred, although patients in the post-omeprazole era tend to be younger, have a higher incidence of mechanical defects such as lower oesophageal sphincter failure or severe impairment of 'pump' function, a more severe grade of oesophagitis, and a higher proportion of patients with an alkaline or neutral refluxate, which omeprazole is unable to control. Interestingly, 35% of patients referred were concerned about the possibility of long term effects of continuous omeprazole treatment (as were their referring gastroenterologists) and many younger patients who were having a comfortable life could be on omeprazole prefer to opt for an anti-reflux procedure and a surgeon with a good track record rather than a lifetime of continuous medication. Furthermore, there are considerable cost benefits in favour of surgery if omeprazole treatment is continued beyond five years, even allowing for treatment of the small proportion of failures after anti-reflux surgery.

We believe that much attention has been directed in recent years towards improving efficacy and reducing side effects of anti-reflux surgery, fuelled by greater understanding of the pathophysiology of gastro-oesophageal reflux and the mode of action of anti-reflux procedures. While the floppy Nissen fundoplication is undoubtedly an effective anti-reflux procedure, it is, in the generality of its use, accompanied by a significant incidence of mechanical complications including gas bloat syndrome, inability to belch and vomit, and troublesome dysphagia, felt to be because of the production of a supra-competent lower oesophageal sphincter which relaxes imperfectly on swallowing. The best and most recent study by an advocate of the floppy Nissen procedure, with symptomatic assessment of 145 patients by an independent gastroenterologist showed an overall incidence of troublesome mechanical complications of 28%.

For these reasons, we have been evaluating over the past 15 years a procedure designed to be more physiologically sound—Nissen fundoplication, which by adopting a multifactorial view of competence enables the avoidance of a supra-competent lower oesophageal sphincter and preserves its ability to relax on swallowing. We have recently reported a clinical and objective study investigating manometry, pH monitoring, endoscopy, and symptom scoring by a non-surgical observer. Symptomatic assessment showed 92% of patients were Visick I and 11, healing of endoscopic oesophagitis occurred in 86%, and restoration to physiological pH profile in 84%. There was no incidence of gas bloat or inability to belch or vomit, although 11% had transient dysphagia and in 2% this was troublesome. Follow up of 497 patients extends up to 15 years, during which time there was one hospital death (0.2%) because of a myocardial infarction 23 days following surgery, and a necessity for revisional anti-reflux surgery in 3.8%. There was no evidence of concomitant splenectomy. Similar results were presented by Munro and colleagues at the recent meeting of the Association of Surgeons of Great Britain and Ireland using this technique.

Undoubtedly, the next step in the evolution of anti-reflux surgery will be its performance through the laparoscopic, which we and others have commenced. It is essential, however, that procedures have a functional as opposed to an anatomical end point subject to careful objective evaluation, and preferably preceded by animal studies. Perhaps the randomised controlled studies which Dehn espouses should be between omeprazole and laparoscopic anti-reflux surgery once this has been fully evaluated. It would be unfortunate if the potential for this technique and these controlled studies were jeopardised by the hasty introduction of laparoscopic anti-reflux surgery without object evaluation, with the inherent risk of bringing a promising technique into disrepute.

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2 Lundell L. Prevention of relapse of reflux oesophagitis after endoscopic healing: the efficacy and safety of omeprazole compared with ranitidine. Digestion 1990; 47: 72–5

Reply

Editor,—Professor Watson's letter concentrates on three aspects of treatment of gastro-oesophageal reflux disease: the use of omeprazole, the side effects following laparoscopic Nissen fundoplication, and the advent of laparoscopic anti-reflux surgery.

Professor Watson states that most patients undergoing anti-reflux surgery are found to have endoscopic oesophagitis of Savary and Miller grade 3. I believe that in the 'post-omeprazole era' more patients undergoing surgery will be found to have lower grades of oesophagitis because of previous long term use of omeprazole treatment. We have commended 93% patient satisfaction. Finally, Professor Watson quotes an abstract by Beauchamp et al.1 The assessment following floppy Nissen fundoplication was undertaken in only 50 of the 145 patients (not all 145) and there was an overall satisfaction rate of 82%. Severe changes in swallowing were reported in 0%. The abstract does not state how these 50 patients were selected. Recently, Walker et al.4 reported significant post-operative dysphagia in one of 26 patients following floppy Nissen fundoplication and gas bloat in three of 26 at the long term assessment.

Whatever anti-reflux operation is performed the most important factor is, as Professor Watson states, choosing a surgeon who specialises in the field and has a good record. There is no substitute for anterior oesophageal vest syndrome which was undertaken with enthusiasm. Initial reports appear encouraging.5 It is to be hoped that the results of this surgery will be audited by


LETTERS TO THE EDITOR

1 Gut 1993; 33: 293–4

Reviewer

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Letters

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Hysterectomy and the irritable bowel

EDITOR — Prior et al found (Gut 1992; 33: 814–7) irritable bowel type symptoms in 22% of women before hysterectomy and reported that such symptoms improved or disappeared in 60% of the women after operation and developed in 40% of them. They suggested that factors related to hysterectomy produced these results and they doubted that natural fluctuation of irritable bowel syndrome was responsible.

I found even higher rates of irritable bowel symptoms in 172 women having elective hysterectomy (40%) and 86 women receiving diagnostic laparoscopy for chronic pelvic pain (48%). One year after hysterectomy, symptoms criteried as bowel had disappeared in 44% of women who originally had them and had appeared de novo in 20% of patients. In relation to whether hysterectomy causes irritable bowel symptoms, it is pertinent to examine the follow up data in the women who had laparoscopy because only 15% of them had hysterectomy during the following year. Irritable bowel symptoms had disappeared in 30% of them after laparoscopy, which appeared de novo in 41% of them. The proportions of women with irritable bowel symptoms originally and at one year follow up were similar in both groups.

My findings do not implicate hysterectomy in the disappearance or the cause of irritable bowel syndrome. As irritable bowel symptoms are common and intermittent, assessment of a control group would be important in future studies of the relation of hysterectomy to bowel symptoms.

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Seum amylase levehs and acute pancreatitis

EDITOR — We fully agree with the conclusion reached by Winslet et al (Gut 1992; 33: 982–6) that serum amylase is not a sensitive test for acute pancreatitis especially when the cause is related to alcohol. In a recent study we found that a serum amylase (>3 times normal) had a sensitivity of only 55% in detecting acute alcoholic pancreatitis. This is much lower than the 96% noted by the authors.

In our study, we included only patients with image proved pancreatitis (ultrasound or computed tomography scanning) as opposed to the clinical criteria adopted by Winslet et al and this could explain the difference.

In their study Winslet et al correctly speculate that serum lipase may be a better test in these circumstances. And we have proved this in our study. We found that serum lipase (>3 times normal) had a sensitivity and specificity of 100% in detecting acute pancreatitis of alcoholic cause.

In the past, the unpopularity of serum lipase was attributed to defects inherent to its assay. With the advent of new techniques and the information obtained from our study, we feel that serum lipase should replace serum amylase as the initial test for acute pancreatitis.

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Reply

EDITOR — We are grateful for the comments by Dr V Gumaste regarding our recent publication. The paper referred to does not give the time from when symptoms first occurred to the serum tests being performed. The finding of a sensitivity of 55% for serum amylase in patients with acute pancreatitis because of alcohol suggests that this was undertaken on average 2–3 days after the start of the attack.

Although not all our patients had confirmatory evidence of the attack, it is worth pointing out that the conclusions in those that did (292 patients) differed little from those that did not (119 patients). In fact, all but four of the patients had at least one ultrasound examination in our study but in most failure to visualise the pancreas because of gas proximally implied an accurate diagnosis. Gumaste et al found ultrasound accurately diagnosed acute pancreatitis in 25 of 32 (78%) patients. It would be important to know how many patients were in fact patients with chronic pancreatitis presenting with an exacerbation. This type of patient was precluded from our study and may fundamentally affect the interpretation of results.

Nevertheless, the finding of a highly sensitive and specific diagnostic rate using the Kodak Ektachem slide lipase test in this difficult group of patients appears to be an important advance. Comparative assessment in a British group of patients is warranted.

J P NEOPTOLEMOS


Colonic tuberculosis

EDITOR — I have read with interest the report by Shah et al on colonic tuberculosis (Gut 1992; 33: 347–51). In the opening statement of the discussion, the authors state that the caecum is most affected in colonic tuberculosis. But, it is the ileocaecal region that is not a caecum, that is involved in tuberculosis. In sordid tuberculosis, involvement of the ileocaecal region is a distinct entity and should be considered separate from small bowel (ileal and jejunal) and large bowel (colonic) tuberculosis. Even in their own results, the authors state that 35 of 50 patients had involvement of the ileocaecal region. Another recent report commented the same semantical error and described 60 cases of ‘ileocaecal tuberculosis’. In a series of 49 ileocaecal tuberculosis cases included as many as 49 with ileocaecal involvement.

The histological hallmark of tuberculosis, central caseation in a granuloma, was seen in only nine patients. The diagnosis in at least some of the 24 patients with non-caseating granulomas, seven with agglomeration of epithelioid cells and five with chronic inflammation could as well be non-specific enteritis or Crohn’s disease, which though uncommon is not unknown in India.

The authors state that in most colonic (including ileocaecal) lesions, the differential diagnosis included Crohn’s disease, and then justified in advocating anti-tubercular chemotherapy even when colonicoscopic biopsy examination is negative for tuberculosis? It would be interesting to know how many patients who were clinically suspected to have tuberculosis, but had negative colonooscopic biopsy for tuberculosis, finally turned out to have other diseases like neoplasia, lymphoma, Crohn’s disease, etc. Also coexisting tuberculosis and carcinoma is not unknown. Moreover, empirical anti-tubercular chemotherapy, in the absence of definite histological diagnosis of tuberculosis, may create problems of diagnosis later, as it changes the histological picture so that differentiation from Crohn’s disease becomes difficult.

Patients with ileocaecal and colonic tuberculosis usually present with lump or features of small bowel and large bowel (ileocaecal) symptoms. In a series of 37 patients with colonic tuberculosis, a third presented with subacute intestinal obstruction and a quarter had hump. Anti-tubercular chemotherapy causes healing which is accompanied by varying amounts of fibrosis and may result in further narrowing of the already compromised lumen and increase the risk of aggravating the obstruction. The authors state that all patients responded to anti-