LETTERS TO THE EDITOR

Surgery for uncomplicated gastro-oesophageal reflux

EDITOR,—The leading article by Mr Dehn on surgery for uncomplicated gastro-oesophageal reflux (*Gut* 1992; **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 antireflux 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 oesophagitis. Most patients coming to surgical treatment have grade 3 oesophagitis, in which group the healing rate on omeprazole treatment is 67%.[1] Furthermore, recurrence of endoscopic oesophagitis after one years continuous omeprazole treatment is up to 33%,[2] 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 unlikely to influence. Interestingly, 35% of patients referred were concerned about the possible long term effects of continuous omeprazole treatment (as were their referring gastroenterologists) and many younger patients who were able to experience how comfortable life could be on omeprazole preferred 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.

As Dehn indicates, 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.[3–6] 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%.[7]

For these reasons, we have been evaluating over the past 15 years a procedure designed to be more physiological than floppy 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 incorporating manometry, pH monitoring, endoscopy, and symptom scoring by a non-surgical observer.[8] Symptomatic assessment showed 92% of patients were Visick 1 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[9] using this technique.

Undoubtedly, the next step in the evolution of anti-reflux surgery will be its performance through the laparoscope, which we and others have commenced. It is essential, however, that procedures having a functional as opposed to an anatomical end point are subjected 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 full objective evaluation, with the inherent risk of bringing a promising technique into disrepute.

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1 Hetzel DJ, Dent J, Reed WD, *et al.* Healing and relapse of severe peptic esophagitis after treatment with omeprazole. *Gastroenterology* 1988; **95**: 903–12.
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.
3 Negre JB. Post fundoplication syndromes. Do they restrict the success of Nissen fundoplication? *Ann Surg* 1983; **198**: 698–700.
4 DeMeester TR, Bonavina I, Albertucci M. Nissen fundoplication for gastro-oesophageal reflux disease. *Am J Surg* 1986; **204**: 9–20.
5 Dedinski GK, Vane DW, Black CT, *et al.* Complications and re-operation after Nissen fundoplication in childhood. *Am J Surg* 1987; **153**: 177–82.
6 Leonardi HK, Corzier RE, Ellis FH. Re-operation for complications of the Nissen fundoplication. *J Thorac Cardiovasc Surg* 1981; **81**: 50–6.
7 Beauchamp G, Dube M, Perrault J. The Post Nissen fundoplication syndrome. *Proceedings 4th World Congress International Society for Diseases of the Esophagus.* 1989; 55.
8 Watson A, Jenkinson LR, Ball CS, *et al.* A more physiological alternative to total fundoplication for the surgical correction of resistant gastro-oesophageal reflux. *Br J Surg* 1991; **78**: 1088–94.
9 Park K, Zentler-Munro P, Munro A. An evaluation of the Watson fundoplication. *Proceedings Annual Meeting of the Association of Surgeons of Great Britain and Ireland.* 1992; 144.

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 floppy Nissen fundoplication, and the advent of laparoscopic anti-reflux surgery.

Professor Watson states that most patients undergoing anti-reflux surgery are found to have 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. In the preceding six months of the 12 patients on whom I have performed anti-reflux surgery, only three were of grade 3 oesophagitis and three were endoscopically negative refluxers. All patients had been given omeprazole and five were under 35 years of age. Although different surgeons seem to treat different populations of patients with reflux disease[1] the widespread use of omeprazole is likely to be reflected in the downgrading of endoscopic findings by comparison with the data obtained in the pre-omeprazole era.

The Large Bowel Cancer Study and West Midlands Oesophagectomy Study both established that the results following major intestinal surgery are largely surgeon dependant. This is borne out by the results reported by Professor Watson following the anti-reflux operation that bears his name[2] and by the results of Bancewicz[3] and others who have employed the floppy Nissen fundoplication. In his letter Professor Watson criticises the second of these operations quoting several references. Bombeck and Donahue first reported the technique for floppy Nissen fundoplication in 1976 following its use in animals.[4] The same authors reported the results in humans in 1985.[5] Negre's paper[6] reports on patients operated on between 1970 to 1979 when the wrap was constructed over an 18 French gauge naso-oesophageal tube: a considerably tighter wrap than that constructed in the floppy procedure (50 French gauge oesophageal bougie in addition to an 18 French gauge oesophageal tube).

Dr Meester's paper[7] quoted by Professor Watson shows a reduction of persistent post-operative dysphagia from 24% to 4% by undertaking a shorter wrap and by increasing the size of the intra-oesophageal stent. The authors reported 93% patient satisfaction. Finally, Professor Watson quotes an abstract by Beauchamp *et al.*[8] 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*[9] 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. Laparoscopic anti-reflux surgery is now being undertaken with enthusiasm. Initial reports appear encouraging.[10,11] It is to be hoped that the results of this surgery will be audited by

surgeons as assiduously as those following laparoscopic cholecystectomy and that the operation will be performed by surgeons with an interest in both oesophageal disease and endoscopic surgery.

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1 Bancewicz J, Matthews HR, O'Hanrahan T, Adams I. A comparison of surgically treated reflux patients in two centres. In: Little AG, Ferguson DB, Skinner DB, eds. *Diseases of the Esophagus*. Vol II. Mount Kiscoe, New York: Futura Publishing, 1990: 177–80.
2 Watson A, Jenkinson LR, Ball CS, *et al*. A more physiological alternative to total fundoplication for the surgical correction of resistant gastro-oesophageal reflux. *Br J Surg* 1991; **78**: 1088–94.
3 Mughal MM, Bancewicz J, Marples M. Oesophageal manometry and pH recording does not predict the bad results of Nissen fundoplication. *Br J Surg* 1990; **77**: 43–5.
4 Donahue PE, Bombeck CT. The modified Nissen fundoplication: reflux prevention without gas bloat. *Chir Gastroenterol* 1977; **11**: 15–27.
5 Donahue PE, Samelson A, Nyhus LM, Bombeck CT. The floppy Nissen fundoplication. Effective long-term control of pathologic reflux. *Arch Surg* 1985; **120**: 663–7.
6 Negre JB. Post-fundoplication symptoms – Do they restrict the success of Nissen fundoplication. *Ann Surg* 1983; **198**: 698–700.
7 De Meester TR, Bonavina L, Albertucci M. Nissen fundoplication for gastro-oesophageal reflux disease. *Ann Surg* 1986; **204**: 9–20.
8 Beauchamp G, Dubé M, Joseé P. The post Nissen fundoplication syndrome. In: *4th World Congress of International Society for diseases of the oesophagus*. Chicago: 1989; 55.
9 Walker SJ, Holt S, Sanderson CJ, Stoddard CJ. Comparison of Nissen total and Lind partial transabdominal fundoplication in the treatment of gastro-oesophageal reflux. *Br J Surg* 1992; **79**: 410–4.
10 Cuche F, Pelissier EP, Girard JF. Preliminary results of laparoscopic Nissen fundoplication. *Br J Surg* 1992; **79** (suppl): 568.
11 Dallemagne B. Laparoscopic Nissen fundoplication. In: *Proceedings of Minimally Invasive Surgery and new technology*. Second European Congress. 1992.

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 de novo in 10% 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[1] 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, symptom criteria for irritable 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 the women one year after laparoscopy but had 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[2,3] and intermittent,[4] assessment of a control group would be important in future studies of the relation of hysterectomy to bowel symptoms.

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1 Longstreth GF, Preskill DB, Youkeles MS. Irritable bowel syndrome in women having diagnostic laparoscopy or hysterectomy. Relation to gynecologic features and outcome. *Dig Dis Sci* 1990; **35**: 1285–90.
2 Jones R, Lydeard S. Irritable bowel syndrome in the general population. *BMJ* 1992; **304**: 87–90.
3 Talley NJ, Zinsmeister AR, Van Dyke C, Melton III LJ. Epidemiology of colonic symptoms and the irritable bowel syndrome. *Gastroenterology* 1991; **101**: 927–34.
4 Waller SL, Misiewicz JJ. Prognosis in the irritable bowel syndrome: A prospective study. *Lancet* 1969; ii: 753–6.

Serum amylase levels 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[1] 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 86% noted by Winslet *et al*. 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 a 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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1 Gumaste V, Dave P, Sereny G. Serum lipase: a better test to diagnose acute alcoholic pancreatitis. *Am J Med* 1992; **92**: 239–42.

Reply

EDITOR,—We are grateful for the comments by Dr V Gumaste regarding our recent publication. The paper referred to[1] 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[2] had confirmatory evidence of the attack, it is worth pointing out that the conclusions in those that did (298 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 precluded an accurate diagnosis. Gumaste *et al*[1] 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[1] in this difficult group of patients appears to be an important advance. Comparative assessment in a British group of patients is warranted.

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1 Gumaste V, Dave P, Sereny G. Serum lipase: A better test to diagnose acute alcohol pancreatitis. *Am J Med* 1992; **92**: 239–42.
2 Winslet M, Hall C, London NJM, Neoptolemos JP. Relation of diagnostic serum amylase levels to aetiology and severity of acute pancreatitis. *Gut* 1992; **33**: 982–6.

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, and not the caecum, that is involved in tuberculosis. In abdominal 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.[1] Even in their own results, the authors state that 35 of 50 patients had involvement of the ileocaecal region. Another recent report committed the same semantical error and described 60 cases of 'large bowel' tuberculosis but included as many as 49 with ileocaecal involvement.[2]

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.[3]

The authors state that in most colonic (including ileocaecal) lesions, the differential diagnosis included neoplasia. Are they then justified in advocating anti-tubercular chemotherapy even when colonoscopic biopsy examination is negative for tuberculosis? It would have been interesting to know how many patients who were clinically suspected to have tuberculosis, but had negative colonoscopic 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.[4] 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.[4]

Patients with ileocaecal and colonic tuberculosis usually present with lump or features of subacute intestinal obstruction. In a series of 37 patients with colonic tuberculosis, a third presented with subacute intestinal obstruction and a quarter had lump.[5] 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-