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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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 found¹ 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.
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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 study¹ 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 to¹ 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 patients² 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 test¹ 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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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.⁵

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.⁶ 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-

tubercular chemotherapy, but the duration of follow up and details of recurrence of obstruction are not given.

Ileocaecal tuberculosis should be described as a separate entity from colonic tuberculosis. Anti-tubercular chemotherapy may be used as the primary treatment if a definite histological diagnosis of tuberculosis has been obtained, if there is no suspicion of malignancy, and if there is no intestinal obstruction; in all other cases surgical treatment should be offered.

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Reply

EDITOR,—I thank Dr Kapoor for his comments on our recent publication in which he raises several issues:

1—He contends that ileocaecal tuberculosis should be considered a distinct entity, separate from large or small bowel tuberculosis. He does not, however, provide a rationale for this suggestion.¹ In our report we included caecal lesions (with or without ileal involvement) as colonic lesions because the caecum is anatomically regarded a part of the colon and because the treatment and the response to treatment is the same whether or not the ileum is involved.

2—Caseating granulomas with acid fast bacilli are a histological hallmark of tuberculosis and we agree that in their absence distinction from Crohn's disease can be difficult. In fact, the difficulty in distinguishing Crohn's disease of the colon from tuberculosis of the colon has been repeatedly emphasised in our paper. The granulomas of tuberculosis, however, in contrast to Crohn's disease, tend to be confluent,² have a heterogeneous appearance,³ are composed of larger numbers of multinucleated giant cells,⁴ and are invariably surrounded by inflammatory cells.⁴ As with any disease, a diagnosis must be made based on the history, physical findings, and the results of all the investigations interpreted in the context of the overall clinical situation.

3—Colonic malignancy was considered in 96% of our patients, but this was based exclusively on the colonoscopic appearance. When this was taken together with the rest of the clinical picture and the histology, which showed features suggestive of tuberculosis and no indication whatever of malignancy, we felt justified in initiating treatment for tuberculosis rather than subjecting the patient to a diagnostic laparotomy. While colonic tuberculosis and malignancy of the colon are known to occur in the same patient, this is relatively uncommon, although this possibility should be borne in mind.

4—Patients with intestinal strictures and symptoms of subacute intestinal obstruction have usually been known to respond to anti-tubercular treatment without requiring surgery as part of their treatment.⁵ In general, the

response to chemotherapy, as assessed by a diminution in the intensity of the symptoms and an increased sense of wellbeing is dramatic. We maintain that a trial with antituberculous drugs is indicated in all patients with intestinal tuberculosis except those with complications such as perforation or fistula formation.

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New theories based on inappropriate application of high technology

EDITOR,—The article by Howard *et al* (*Gut* 1991; 32: 1406-11), brought to my attention, has a puzzling conclusion drawn by the authors. Based upon their ultrasound findings, Howard *et al* proposed a new theory that the emptying of the gall bladder after a meal is in three phases. I have read this article with great interest and find that this new theory may have resulted from an inappropriate use of ultrasound for measuring gall bladder emptying.

The measurement of gall bladder volume by a geometric method, called the sum of the cylinder method, was originally described in 1948 by de Silva.¹ The method requires that the gall bladder be a pear shaped organ that can be cut into a series of small cylinders and stacked one above the other. The volume is then computed by summing up the volume of each cylinder. Everson *et al*, using ultrasound, modified de Silva's method slightly by applying the correction factor to account for the change in curvature of the gall bladder.² To be accurate, this method again requires that the

gall bladder be pear shaped and change in the displacement of the longitudinal axis be uniform. Everson *et al*, however, cautioned that the shift in the longitudinal axis may not be uniform in all and that the volume would be an overestimate in the case of long narrow tapering segments at each end of the gall bladder. Also, in situations where the long axis is parallel to but displaced from the central axis, the gall bladder volume will be underestimated in the tapering regions.

The gall bladder at rest has many shapes, often with segmentations and septa. The gall bladder changes its axis and shape during its contraction.³ The mean resting pressure inside the sphincter of oddi is 15 cm of water, the common bile duct 12 cm of water, and the gall bladder 10 cm of water. If the tensed gall bladder does not generate enough pressure inside to overcome sphincter of oddi pressure, it may not empty bile despite appearing small in size. To overcome these disadvantages, we have introduced a count based non-geometric scintigraphic method where the bile emptying measurement is not influenced by the change in shape or the axis of the gall bladder. The bile emptying is measured as ejection fraction. Measurement of the absolute bile volume is not required.⁴

The main portion of the normal gall bladder filling occurs from the absorption of water through the wall and not from frequent emptying and refilling. We studied over 300 normal subjects with technetium-99m HIDA compounds by acquiring the data at a half or one minute interval for 60-120 minutes. A typical gall bladder filling phase is a smooth upgoing curve without any serrations (no emptying or refilling). On rare occasions, a gall bladder may empty spontaneously before the meal. This spontaneous emptying is rare and when it occurs it accounts for less than 5-10% ejection fraction in normal subjects. When the fatty meal is ingested, the gall bladder shows a latent period of 6-16 minutes before it begins to empty bile which is consistent with the known normal release time of endogenous cholecystokinin into circulation. In more than 50 normal subjects, which we studied with a fatty meal, an early dip of the cephalic phase was seen in less than 5%. When the normal gall bladder begins to empty following endogenous cholecystokinin release, it maintains its continuous emptying for 60-120 minutes without

Figure 1: Typical normal gall bladder bile emptying curve following fatty meal. Note a latent period of 16 minutes before bile emptying begins. Once the bile emptying begins it is continuous and sustained with no refilling during the ejection period. (Reproduced with permission from *J Nucl Med* (Ref 5).)