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.

T C B DEHN
Royal Berkshire Hospital, London Road, Reading RG1 5AN

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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 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.

G F LONGSTRETH
Southern California Permanente Medical Group, San Diego, California, USA

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 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.

V GUMASTE
Division of Gastroenterology, Mt Sinai Services/City Hospital Elmhurst, New York, USA

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.

J P NEOPTOLEMOS

1 Gumaste V, Dave P, Sereny G. Serum lipase: A better test to diagnose acute alcohol pancreatitis. *Am J Med* 1992; **92**: 239–42.
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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.[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-