cacy of combining these drugs might be of great help. Presently, the data in the three meta-analyses constitutes a sound, objective basis on which to tackle therapeutic decisions.

The statement endorsed by the working party seems to reflect the common belief shared by leading authorities in this field that these agents are ineffective in the treatment of acute pancreatitis. This may be because the drugs are believed to act through their antisecretory or protease inhibitory activity. However, other relevant properties of somatostatin and octreotide, namely the haemodynamic effects on reducing splanchnic blood flow, the stimulation of the hepatic reticuloendothelial system, and the modulation of the cytokine cascade, are usually forgotten. It has been hypothesised that the efficacy of somatostatin and octreotide in acute pancreatitis might be better explained by all these properties rather than its capability of inhibiting pancreatic secretion.³

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Colonoscopic surveillance in ulcerative colitis

EDITOR,—The study by Karlen *et al* (*Gut* 1998;42:711-14) makes the reader wonder how the authors could draw a conclusion that is suggestive of a positive outcome when the data analysis clearly shows no statistical significance.

It is well established that mortality from colorectal carcinoma is an important factor in patients with ulcerative colitis. However whether surveillance programmes have any effect on mortality is still matter of debate.¹⁻⁴ Furthermore for the practical and ethical considerations mentioned by Karlen *et al* in their introduction, it is unlikely we would ever have a randomised controlled trial to look at the value of colonoscopic surveillance in this respect. It is likely that we will come to rely on studies such as Karlen *et al*'s.

It is for this reason that we should scrutinise the methods and results of such studies closely so that we can draw appropriate conclusions. Karlen *et al* studied 4664 subjects with a definite diagnosis of ulcerative colitis from an approximate background population of 3 million people in two regions of Sweden. The patient group was identified as "all patients in the study population that had died from colorectal cancer after 1975", which amounted to 40 cases. The aim of the control population was to have three controls for each patient matched individually by age at diagnosis (± 5 years), duration of disease,

extent of disease at diagnosis, sex, as well as needing to be alive at the time of death of the patient and to have some part of the colon intact five years prior to the diagnosis of the cancer of the patient. The criteria are extensive and valid and the authors' aim was to obtain 120 individuals. However they give the impression that after the 120 individuals were chosen, tight scrutiny of the criteria confined them to 102. Surely you would select your controls based on defining your criteria initially rather than reassessing the number of individuals chosen and excluding some that do not meet the "criteria".

Finally the results give a relative risk for which the reference is not clearly identifiable. One is left to assume that it is the relative risk of survival advantage having undergone a surveillance colonoscopy. Regardless of this, the confidence interval includes 1. Karlen *et al* go on to state that of those undergoing two or more surveillance colonoscopies the relative risk was 0.22 with a 95% confidence interval that is wider than the overall confidence interval for the relative risk of ever having a surveillance colonoscopy. This confidence interval also includes 1, and they state that somehow this indicates "a protective dose response relation".

The final comment in the results alludes to 10% of controls having undergone colectomy within five years prior to diagnosis of the cancer of the patient but we are not informed of the reasons why these patients underwent colectomy. It is important to know whether their colectomy was for colorectal cancer or failure of medical treatment to control their ulcerative colitis.

One is left disconcerted that, given the criticisms outlined earlier, the authors could still conclude that, "the main finding, although not statistically significant, indicates that colonoscopic surveillance may have a protective effect against death from colorectal cancer". Furthermore that this, "protective effect is even more pronounced if the patient underwent two or more surveillance colonoscopies, indicating a protective dose response relation".

This is clearly a negative study, and the statement that there is a "protective dose response relation" must be considered to be unfounded and biased.

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Cholecystokinin provocation tests

EDITOR,—The commentary by Al-Musawi and Williamson (*Gut* 1997;43:454-5) on our paper (*Gut* 1998;43:571-4) in the same issue suggests that there is still a place for cholecystokinin (CCK) provocation tests although the authors fail to provide any evidence to support their statement. Their criticism of our

paper relates to numbers of patients in subgroups and histology of the gall bladder, and they seem to have ignored the key message on outcomes after cholecystectomy in these patients as a whole. The only study they have quoted in support of their statement is Rhodes *et al*'s paper¹ which we have discussed in detail as proving that patients who were CCK negative had perhaps better symptom relief than CCK positive patients (89 v 67%). CCK does contract the gall bladder and may cause pain but this has no bearing on symptomatic outcome after cholecystectomy. This is what makes this test useless.

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Requiem for the cholecystokinin provocation test

EDITOR,—I read with interest the paper from Professor Johnson's group (*Gut* 1998; 43:571-4) questioning the value of the cholecystokinin (CCK) provocation test as a predictor of outcome following cholecystectomy in patients suspected of having acalculous biliary pain. The results of this study are predictable as in my experience tests of gall bladder function in this situation are not much better at outcome prediction than tossing a coin unless they are combined with a quantitative assessment of sphincter of Oddi function.

It is my practice when evaluating patients with suspected acalculous biliary pain to measure the gall bladder ejection fraction (GBEF) using a CCK stimulated HIDA (hydroxyindole diaminooacetic acid) scan. Patients with two separate GBEF values of <35% are offered endoscopic biliary manometry. Patients with normal manometry (basal pressure <40 mm Hg, mean peak wave pressure <140 mm Hg and contraction rate <9/min) of both biliary and pancreatic sphincters respond extremely well to cholecystectomy. Conversely, patients with abnormal manometry must be warned that sphincter of Oddi hypertension may result in post-cholecystectomy symptoms requiring treatment with calcium channel antagonists, botulinum toxin injection into the sphincter of Oddi or endoscopic sphincterotomy.

The modern biliary manometry catheter (Lehman catheter, Wilson Cook Ltd) with a distal aspiration channel has made endoscopic manometry of the sphincter of Oddi a safe procedure with an incidence of postprocedure acute pancreatitis of <5% even for patients with confirmed sphincter hypertension. Manometry of the hypertensive pancreatic sphincter should always be accompanied by short term pancreatic duct stenting as this reduces the incidence of acute pancreatitis in these patients dramatically.

I would suggest that the study from Smythe *et al* is incomplete without sphincter of Oddi function data and that we should not discard gall bladder function tests just yet.

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