They question whether we were able to empty the balloon between inflations. Because we measured the volume inserted into the balloon on each occasion and also the volume recovered to ensure that accumulation of fluid in the balloon did not occur. They criticise the use of a fluid-filled catheter to detect intraballoon pressure (standard practice by most other workers). We agree that different absolute values could be obtained using different catheters but find it difficult to see how the varying inter and intraindividual results we obtained could be the result of using different catheters.

They also question whether our normal subject group, all of whom were symptom free and without past or current history of bowel symptoms, could have inadvertently included subjects with irritable bowel syndrome. If it is possible for an adult who feels healthy and who is not aware of any symptoms referable to the gastrointestinal tract to be suffering from irritable bowel syndrome then we have to admit guilt, but then anybody studying normal human physiologic of the gut will commit the same mistake.

Varma and Smith also make further points concerning irritable bowel syndrome, suggesting that there may be subgroups who could perhaps agree. We would not disagree with this possibility and indeed two of our patients with predominant diarrhoea (cases 4 and 24) had maximal tolerable volumes to distension which were at the bottom of the patient range. The point that we make in our paper, however, is that neither of these two patients fell outside the 95% confidence limits for our normal range and while there may indeed be a difference between irritable bowel syndrome patients who are at the extremes of symptoms, neither of these two extremes can be regarded as being ‘abnormal.’

They question the past history of the patients and their age: we also considered this and mentioned it in our discussion. It seems likely that failure to control for differences between age and sex of the two groups would have increased the differences between the groups rather than reduced them.

They question whether rectal volumes tolerated by subjects would vary with the confidence and experience of the subjects. We wholeheartedly agree with this statement and indeed this was one of the aims of our study. Our results showed that there was individual variability rather than independent measurement.

They comment that the tracings which we published of the pressure-volume relations seem to be consistent in each individual and we agree with this. Our point again, however, is that it is the sensory end points that are variable, even though the pressure-volume curve is consistent. The major problem with analysis of pressure-volume curves, however, is to adequately define in mathematical terms the pressure-volume curve itself. It is of doubtful use to talk about compliance (as do many workers in the field), since as is evident from all published data, the slope of the pressure-volume curve varies during inflation so that there are a number of compliances rather than a single value.

Sun et al also criticise our selection of patients and suggest that our sample was biased towards constipation. Thirteen of 26 patients were constipated, two were predominately diarrhoea sufferers, five had both diarrhoea and constipation, the rest were unaffected by major alterations in stool consistency. These patients therefore do not seem too different from those reported in other recent studies of irritable bowel syndrome such as that of Price et al (Sun et al, reference 1) in which 27 of 55 irritable bowel patients were constipated. We would not argue with their suggestion that patients with diarrhoea predominant symptoms may differ from those with constipation predominant symptoms and also are aware that those individuals with diarrhoea in our study had the lowest tolerated volumes of the groups. We point out again, however, that none of these individuals fell outside the range of normality as defined by our volunteer data, so that while proctometrography may differentiate between irritable bowel syndrome patients with diarrhoea and constipation, it is difficult to see how it might be used to distinguish the irritable bowel syndrome from normal.

From the interest which our article has stimulated it is evident that more data are required to distinguish between these two basic factors influencing pressure-volume relations in the rectum. We look forward to seeing more data from both correspondents to help clarify these difficulties.

Why do patients with ulcerative colitis relapse?

Sir,—Dr J W Pauley takes issue with our criticism that previous studies of psychological factors and ulcerative colitis relapse are uncontrolled (Gut 1990; 31:1419). In support of his argument he cites three studies in which radiotherapy patients, healthy siblings, and healthy volunteers are used as ‘control’ groups against which ulcerative colitis patients are compared. The data as presented by Dr Pauley are, however, misleading. In the first study radiotherapy patients were only used as controls for the purpose of personality comparisons. The data concerned colitis relapse are uncontrolled and anecdotal. The second study is a comparison of personality characteristics between 23 patients with inflammatory bowel disease (12 with ulcerative colitis) and their healthy siblings and makes no attempt to study colitis relapse. This study is considerably flawed since the decision to use sibling controls was based on previous data suggesting that siblings had different personalities. Unfortunately, we have been unable to obtain a copy of the 1957 Czech study1 as there is no United Kingdom source (British Library, personal communication). We suspect, from his limited description, that this too is a personality assessment.

Unfortunately, personality studies are of limited value in the study of ulcerative colitis as it is difficult to distinguish between symptom attributes from illness related changes. It is clearly inappropriate to use controls who do not suffer from episodic bloody diarrhoea and who are not at risk of incontinence, surgery, and cancer. We recognised that matched disease controls are not easy to find but the above studies are inappropriate controlled.

The relapsing nature of colitis offers an ideal opportunity to undertake controlled studies of the psychological factors associated with relapse. Patients in relapse may be compared with those in remission and in cohort studies patients may act as their own controls. Using such a design we found that the patients of those patients reported stressful life events in the three months before relapse. In the absence of controlled data this suggests an association, but patients in remission reported a similar number of events.

Finally, Dr Pauley believes that in forming our conclusion that psychological factors are unimportant in colitis relapse we ‘have listened to commonly recited, but uncorroborated views of others, rather than checked the original sources.’ We suggest that such criticisms are commonly voiced because others have also checked the original studies and found them lacking.

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Sedation for upper gastrointestinal endoscopy

Sir,—The paper by Daneshmand et al (Gut 1991; 32:12-5) outlines the results of a postal questionnaire sent to 665 endoscopists and provides some alarming statistics — 52 deaths, and a further 119 respiratory arrests and 37 cardiac arrests in a two year period.

I suggest that most of these incidents could have been avoided if the following precautions had been observed: (a) all patients were monitored with pulse oximetry; (b) all patients received supplemental oxygen; (c) a trained medical observer devoted total attention to the sedation and cardiorespiratory well being of the patient; (d) oxygen was continued in the postendoscopy period when indicated.

In the experience of many endoscopy units a combination of midazolam and fentanyl given in doses appropriate to the age and condition of the patient provides satisfactory sedation that is safe provided that the above precautions are observed.

It is a pity that some endoscopy units seem to be isolated from the department of anaesthesia, who may be able to help with advice and trained personnel to make the procedure safer.

R CLARK
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Sir,—I was interested in the result of the reported study of postal questionnaires inquiring about the sedation practice of endoscopy clinicians only 2% of respondents stated they did not use sedation. It has been my practice since 1976 to offer no sedation to patients but
to use local anaesthetic spray to the throat. If the patient requests sedation or if terrified a titrated dose of midazolam is given. Endoscopy without sedation is successfully practised by my registrars and has the advantage that it is possible to talk to the patients immediately after the endoscopy, and they are allowed home within the hour.

There have been no deaths or major injuries on my unit relating to patients undergoing upper gastrointestinal endoscopy since 1976. It is my impression that, apart from patient safety, instruments get damaged less. Keeping the patient’s conscious cooperation by sympathetically talking the instrument down also protects the endoscope. Many of my patients come back for several repeat endoscopies.

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Reply

Str,—Mr Kingston and Drs Clark and Goy suggest contrasting means of improving the safety of endoscopy. While most would agree that the option of not using sedation should be considered occasionally, this option is clearly not possible in all patients. Nevertheless, the avoidance of sedation when possible in ‘at risk’ patients, such as those with acute gastrointestinal bleeding, should be encouraged.

From our experience the ‘interventionist’ approach of intensive monitoring and oxygen supplements is not yet likely to find favour among most British gastroenterologists. These interventions all have opportunity costs both in resources and time and until there is some evidence of their being both effective and appropriately applied to ‘at risk’ groups, then recommendations as to their routine use seem premature. Indeed, is there evidence that patients having routine upper endoscopies with supplementary oxygen suffer significant O₂ desaturation and would therefore also need pulse oximetry?

The purpose of our survey was to make a preliminary assessment as to whether an appreciable problem existed. Our survey suggests a striking number of serious adverse outcomes occur, and studies, sponsored by the British Society of Gastroenterology, are now in progress to identify which patients are at risk and the most useful interventions.

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Palliation of malignant obstructive jaundice — surgery or stent?

Str,—I would like to comment on Hatfield’s excellent brief leader (Gut 1990; 31: 1339–40). Although he was reviewing methods for palliation, it might have been appropriate to mention the frequent difficulty in proving that a patient has an incurable lesion. Both histological confirmation of malignancy, and irrefutable evidence of unrespectability, are sometimes hard to obtain short of operation. An argument in favour of surgical palliation is that these doubts can be laid to rest; the morbidity of surgery is very low if carefully selected patients are managed by an expert surgeon and perioperative team.

The statement that ‘there is little doubt that endoscopic stenting is the treatment of choice’ (for hilar lesions) is provocative. Some primary biliary bifurcation lesions are resectable1 (a few even prove to be benign), and I am not convinced that percutaneous interventions are obsolete. The careful randomised study by Speer et al.2 certainly showed endoscopic stenting to be safer than percutaneous intervention at the Middlesex and London Hospitals — but those data have yet to be confirmed in other institutions. It may be that two expandable stents placed percutaneously via small transhepatic catheters would be more effective than one placed endoscopically from below; stenting both sides of a hilar lesion endoscopically is rarely possible. In addition, combined percutaneous-endoscopic manipulation is often necessary in managing difficult problems, as the Middlesex group have reported.3

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Reply

Str,—We read with interest the leading article in Gut (1990; 31: 1339–40) which discussed the relevant merits of surgical bypass and endoscopic stenting for the palliation of malignant biliary obstruction. While we would agree with Dr Hatfield that endoscopic stenting achieves good biliary decompression with a low procedure related mortality and morbidity, there are several points in his article which cannot remain unchallenged.

Firstly, we strongly contest the notion that cholangiocarcinoma of the proximal common bile duct or its confluence (Klatskin tumour) should automatically be managed by stenting. This tumour is characteristically slow growing and often masked by the presence of biliary features surgical resection often produces good longterm palliation and occasional cure. The resection rate of hilar cholangiocarcinoma is increasing worldwide with a corresponding fall in mortality and morbidity when performed in specialist units.1 A median survival of two years is expected, and the five year survival in recent series has reached a creditable 17%. A quality of survival for the vast majority of these patients is excellent. Another serious consequence of stenting high biliary strictures without exploratory surgery is that the diagnosis is not proved. It has been shown that, despite sophisticated imaging techniques, the diagnosis of Klatskin tumour will be incorrect in 20–30% of cases.2 Immediate endoscopic stenting will therefore lead to the mismanagement of both benign biliary strictures and some highly curable malignant lesions, such as papillary adenocarcinoma.

With regard to low bile duct obstruction due to malignancy, two points need to be made. Firstly, the best bypass procedure is a Roux-en-Y choledochoduodenostomy, not the operations mentioned in Dr Hatfield’s article. Moreover, the standard Whipple pancreaticoduodenectomy can be performed with an operative mortality of less than 5%1, and excellent palliation accompanies this procedure. Five year survival rates of 30% for cholangiocarcinoma and 15% for pancreatic cancer have been reported.3 These results far exceed the median survival of five months quoted from the Middlesex trial.

In summary, endoscopic biliary stenting is a new and exciting procedure for the palliation of malignant biliary obstruction, but its exact role has yet to be defined. We would agree with Dr Hatfield that stenting is now a possible option for high risk surgical candidate and for patients with obvious advanced malignant or metastatic disease. Resectional surgery, however, remains superior to any palliative endoscopic procedure, and appropriate patients at least deserve the opinion of a specialist surgeon before being referred for stenting. Medical nihilism should be discouraged, and physicians should remember that the decision to stent limits the patient’s survival to a few months. Routine stenting, therefore, rests very uncomfortably with us.

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Reply

Str,—In response to the letters from Professor Peter Cotton and Messrs Russell and Rees concerning this leading article we would like to comment with particular reference to hilar and low bile duct strictures.

Hilar strictures

The evidence that cholangiocarcinoma is a uniformly slow growing tumour is at variance with our own experience1 in a large group of over 100 patients with Klatskin tumours in whom the median survival is only 12 weeks. A few patients have prolonged survival admitted, but such patients survive with restorative surgery, radiotherapy, or stenting. However, while we agree that specialist units are necessary, it is important to note the median age of patients in the above series was 75 years, less than 15% being under the age of 60 years. The good results of Cotton’s review could merely be due to patient selection and age. Probably our experience and referral pattern is different and this is an excellent example of the widely quoted ‘apples and oranges’ phenomenon described by Peter Cotton.

We agree that diagnosis can be difficult, but spontaneous benign strictures in this region are extremely rare and papillary lesions are easily distinguished by their different radiological appearance. Our policy is to attempt to obtain a histological diagnosis in those patients where endoscopic retrograde cholangiopancreat-