the decreased CDAI scores, which are considered universally as unreliable and subjective assessments, remission induced in these patients by the novel treatment is probably only short lived. The high concentrations of soluble TNF receptors show, in our opinion, that these patients have not achieved a stable remission. We have found that the concentration of the soluble TNF receptors in the urine is a useful prognostic indicator for determining the efficacy of treatment and predicting a change in the status of the disease: relapse and remission.

Transfusion for variceal bleeding in cirrhotic patients

EDITOR.—We wish to comment on the article by McCormick et al (Gut 1995; 36: 100–3) suggesting that secondary haemodynamic changes in the splanchic circulation after variceal bleeding may contribute to increased risk of further haemorrhage. They suggested that the increased splanchic blood flow could result either from a reflex portal hyperpressure or to overtransfusion during resuscitation. The reflex portal overpressure could be related to neuroendocrine stimulus induced by the presence of blood in the gut. In a previous trial, we have shown that whole gut irrigation with isotonic mannitol reduced blood transfusion requirements and rebleeding rate after variceal bleeding.1 To investigate the possible deleterious influence of blood transfusions, we have compared, in a pilot randomised study, two protocols of transfusion in cirrhotic patients after recent (<24 hours) and severe variceal bleeding (packed cell volume <27%). In group 1 (n = 43), patients were transfused to reach a packed cell volume value of 25 ± 2% and in group 2 (n = 32 ± 2%). Patients were mainly Child–Pugh grades B and C without any difference between the two groups. There were six Child–Pugh A, 20 B, 17 C in group 1 and seven A, 23 B, and 17 C in group 2. In patients with active bleeding, haemotasis was obtained by balloon tamponade. The percentage of patients with active bleeding was similar (52%) in the two groups. No patient received vasoactive drugs. The follow-up period was six days after the initial endoscopy performed at admission. Recurrent bleeding was defined by a new bleeding episode, a 3% or more drop of the packed cell volume within 24 hours or the lack of correction of the initial value of packed cell volume despite transfusions, or all three. At day 6, the mean number of blood units received by the patients was 2.6 in group 1 versus 4.4 in group 2 (p < 0.05). The rebleeding rate was 40% in group 1 and 48% in group 2 (NS) and the death rate was 14% in group 1 and 12% in group 2 (NS). Complications (hepatorenal failure, sepsis, hepatic encephalopathy) occurred with a similar frequency in the two groups. In this pilot study, the absence of significant benefit with the low transfusion rate is probably due to a beta error. Nevertheless, in accordance with McCormick’s opinion, it suggests that limited blood transfusions could reduce the early rebleeding risk in cirrhotic patients with variceal bleeding without deleterious effects. A large multicentre trial is actually in progress to compare the results of the two transfusion protocols in cirrhotic patients with variceal bleeding treated by vasoactive drugs and emergency sclerotherapy.

GASTRIC METAPLASIA AND HELICOBACTER PYLORI INFECTION

EDITOR.—Dr Savarino and his colleagues (Gut 1995; 37: 445–6 [letter]) are not entirely correct.1 Their observations were published in Gut in 1989 and subsequent report.2 They quote us as ‘failing to show any reversal of gastric metaplasia in the duodenum’ following medical antisecretory treatment. However, our numbers were small, we reported a noticeable difference between the persistence of metaplasia, as shown by a light and electron microscopy scoring, at the end of one year’s maintenance treatment following duodenal ulcer healing with sucralfate 400 mg every night and one year’s maintenance treatment with sucralfate 1 g twice daily. Gastric metaplasia was absent in two of 14 and minimal in three of 14 of the cimetidine group compared to four of 14 and four of 11 respectively of the sucralfate group. It was noted that Helicobacter pylori was absent in those patients with no metaplasia. In addition, although both groups at the time of the ulcer healing showed a moderate improvement in the mucosal scoring, at the end of the maintenance year the score in four of 14 of the cimetidine group had reverted to their high initial pretreatment score compared with one of 14 in the sucralfate group. This suggests that treatment that enhances mucosal protection is more likely to enable the duodenal mucosa to revert to normal than reduction of acid secretion.

FAecal STREAM DIVERsION IN PATIENTS WITH COLLAGenous COLITIS

EDITOR.—Veress et al have published an interesting paper on the microscopic colitis syndrome (Gut 1995; 36: 88–9). Their finding on patient 16 whose increased collagen layer was normal after a temporary loop ileostomy is identical to our previous findings.3 4 We have presented experiences of faecal stream diversion in collagenous colitis at both Swedish1 and Scandinavian2 conferences on gastroenterology. Our original report included five of our own patients, but later extended with four other cases who underwent surgery in other Swedish hospitals. These four patients were included in our study after written permission had been obtained from their specific doctors.

Our main conclusion from these nine operated patients was that faecal stream diversion induced clinical and histopathological remission in collagenous colitis.5 After closure of the ileostomy and restoration of intestinal continuity clinical symptoms and the abnormal collagen layer recurred. In a patient who had a sigmoidostomy using the Hartmann procedure, the abnormal collagen layer remained thickened in the proximal colon still exposed to the faecal stream, but was normal in the excluded rectosigmoid colon. Later, the sigmoidostomy was replaced by a split ileostomy and at follow up the collagen layer was normal in the whole colon. These findings strongly indicate that a luminal noxious factor may be of pathogenetic importance.1–3 The nature of this luminal factor is unknown. Hypothetically, it may remain in the small bowel after faecal stream diversion, which could be the cause that relapse seen in some of the patients after restoration of intestinal continuity.3 Patient 16 in the study by Veress et al is the same patient IBM in our report.2

Reply

EDITOR.—Thank you for giving us the opportunity to comment on the letter from Järnerot et al. In 1977–1979 one of us (BV) was working together with Dr C Lindström, who first described collagenous colitis.1 In 1982, we presented our first patient with collagenous colitis at the Meeting of the Swedish Society of Surgeons in Karlstad.2 The patient referred to by Järnerot et al (no 16) was included in our first joint report from the Central Hospital in Karlstad and Huddinge University Hospital. Järnerot et al were unaware that this patient was originally seen by us. The findings described by Järnerot et al at the time of the preparation of our paper had only been presented as abstracts.
Faecal stream diversion in patients with collagenous colitis.

G Järnerot, J Bohr, C Tysk and S Eriksson

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