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.

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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 haemo-
dynamic 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 due to blood products. The reflex portal over-
pressure 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 isonotic man-
nitol reduced blood transfusion require-
ments and rebleding rate after varical 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 varical 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=41) to 32±2%.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, haemostasis 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. Recur-
rent bleeding was defined by a new bleeding episode, a 3% or more drop of the packed cell volume ratio 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. The rebleding 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 (hepa-
toracal failure, renal failure, hepatic encepha-
lopathy) 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 rebleding rate in cirrhotic patients with varical 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 varical bleeding treated by vasoactive drugs and emergency sclerotherapy.

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2 Tull S, Bruna T, Dao T, Duhamel C, Transvozes JL, Gueden C, et al. Influence des transfusions sur le taux de récidives précoces des hémor-

Gastric metaplasia and Helicobacter pylori infection

EDITOR.—Dr Savarino and his colleagues (Gut 1995; 37: 445–6 [letter]) are not entirely correct in their opinion that their paper published in Gut in 19891 and subsequent subsequent report.2 They quote us as 'failing to show any reversal of gastric metaplasia in the duo-

denum following medical antisecretary treatment. Although 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 of 400–500 mg every night and one year's maintenance treatment with sucralfa 1 g twice daily. Gastric metaplasia was absent in two of 14 and minimal in three of 14 of the cimetidine group compared with no one 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 only one of 14 of 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.

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1 Tovey FI, Husband EM, Yiu Chu Yiu, Baker L, McPhail G, Lewin MK, et al. Comparison of relapse rates and of mucosal abnormalities after healing of duodenal ulceration and after one year's maintenance with cimetidine or sucral-


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 find-
ing on patient 16 whose increased collagen layer was normal after a temporary loop ileostomy is identical to our previous findings.2 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 cases but was later extended with four other cases who underwent surgery in other Swedish hos-
pitals. These four patients were included in our study after written permission had been obtained from the patients' doctors.

Our main conclusion from these nine operated patients was that faecal stream diversion induced clinical and histopatho-
logical remission in collagenous colitis.3 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 resected sigmoid colon. Later, the sigmoidostomy was replaced by a split ileostomy and at follow up the col-

lumen was normal in the whole colon. These findings strongly indicate that a luminal noxious factor may be of patho-
genetic importance.1,3 The nature of this luminal factor is unknown. Hypothetically, it may remain in the small bowel after faecal stream diversion, which is then continued for the first time after the surgical end of the ileostomy is closed. Patient 16 in the study by Veress et al is the same patient IMB in our report.

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Reply

EDITOR.—Thank you for giving us the opportu-
nity to comment on the letter from Jänerot et al. In 1977–1979 one of us (BV) was work-
ing 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 Jänerot et al (no 16) was included in our first joint report from the Central Hospital in Karlstad and Huddinge Univer-
sity Hospital. Jänerot et al were unaware that this patient was originally seen by us. The findings described by Jänerot et al at the time of the preparation of our paper had only been presented as abstracts.
Gastric metaplasia and Helicobacter pylori infection.

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