Surgery was considered on days two and six. The knee-elbow position reversed the radiographic appearances, however, and changed the treatment of our patient's colitis. The use of this simple and safe manoeuvre probably spared our patient the use of the knee to decompress the bowel in toxic megacolon.

J FLETCHER  
A J LOBO  
R F HARVEY  
Frenchay Hospital,  
Bristol BS16 1LE

EDITOR.—Panos et al (Gut 1993; 34: 1726–7) describe the knee-elbow position for the relief of bowel distension in patients with toxic megacolon. They suggest that use of this therapeutic manoeuvre is new.

In 1988 Present et al 1 published their experience with ‘rolling’ 19 patients with toxic megacolon into the prone position every two to three hours for 10 to 15 minutes. They concluded this was a helpful addition to the standard treatment for this serious condition.

Both groups are describing the same phenomenon – the prone position redistributes colonic gas and fluid into the lower bowel leading to easier evacuation. We have been using this technique for several years at the Stanford Medical Center.

R R BABB  
Palo Alto Medical Clinic,  
300 Homer Ave,  
Palo Alto,  
CA 94301, USA

EDITOR.—We welcome the report by Noach et al (Gut 1993; 34: 1510–4) on the relation between duodenal gastric metaplasia and Helicobacter pylori infection. Two findings deserve particular attention in this study. Firstly, the presence and extent of gastric metaplasia in the duodenum was not significantly associated with H pylori infection, because it was found in 78% of H pylori positive and in 85% of H pylori negative patients with dyspeptic complaints. Secondly, there was no reduction in the extent and prevalence of gastric metaplasia after 12 months of eradication of H pylori.

The first finding obviously excludes the responsibility of H pylori in the formation of gastric metaplasia in the duodenum, but raises serious questions on the role of acid in this process.1 In fact, a study has already shown that patients with functional dyspepsia have a basal acid output that is comparable with that of normal subjects.2 Using ambulatory pHmetry3 we have even found that the circadian gastric acidity in H pylori negative dyspeptic patients is significantly lower (24 hour mean: SEM) pH 2.8 (0.8) v 1.8 (0.4), p<0.001 than normal (Figure). In addition, although H pylori infection results in an increase in gastric release, this hypergastrinaemia does not induce an increase in acid secretion. There is no difference, indeed, in basal and maximal acid output in patients with duodenal ulcer as compared with non-ulcer dyspepsia.4 5

Reply

We welcome the correspondence on our report, which gives us the opportunity to further discuss the use of postural manoeuvres for bowel decompression. 'Rolling', as described by Dr Present et al, entails turning the patient to the prone position, on a flat bed.1 This position is clearly different from the knee-elbow position we have reported in which the patient is positioned head down with hips, knees, and elbows flexed.5

A crucial point needs to be made with respect to the ‘rolling’ regimen, as described by Present et al in their series of 19 patients: in addition to turning the patient prone, a long enteral tube was passed for aspiration of gas and enteral fluid.1 Consequently it is difficult to discern what the individual contribution of the intubation-aspiration versus the postural manoeuvre was, in decompressing the bowel. We also note that in five of their 19 cases, a probably sparse was required to facilitate evacuation of gas that had not been forthcoming.1 The uppermost position of the rectum and anus in the knee-elbow position permits easy passage of flatus and could obviate the need for rectal catheterisation.

The early and longterm follow up results from Dr Present’s uncontrolled series are impressive. Nevertheless, the effect of bowel decompression by postural manoeuvres on the outcome of toxic megacolon should be confirmed by prospective, randomised, controlled trials.

We remain unaware of any previous reports

Acid and gastric metaplasia in the duodenum

EDITOR.—We read with great interest the paper by Noach et al (Gut 1993; 34: 1510–4) on the relation between duodenal gastric metaplasia and Helicobacter pylori infection. Two findings deserve particular attention in this study. Firstly, the presence and extent of gastric metaplasia in the duodenum was not significantly associated with H pylori infection, because it was found in 78% of H pylori positive and in 85% of H pylori negative patients with dyspeptic complaints. Secondly, there was no reduction in the extent and prevalence of gastric metaplasia after 12 months of eradication of H pylori.

The first finding obviously excludes the responsibility of H pylori in the formation of gastric metaplasia in the duodenum, but raises serious questions on the role of acid in this process.1 In fact, a study has already shown that patients with functional dyspepsia have a basal acid output that is comparable with that of normal subjects.2 Using ambulatory pHmetry3 we have even found that the circadian gastric acidity in H pylori negative dyspeptic patients is significantly lower (24 hour mean: SEM) pH 2.8 (0.8) v 1.8 (0.4), p<0.001 than normal (Figure). In addition, although H pylori infection results in an increase in gastric release, this hypergastrinaemia does not induce an increase in acid secretion. There is no difference, indeed, in basal and maximal acid output in patients with duodenal ulcer as compared with non-ulcer dyspepsia.4 5


Reply

EDITOR.—We thank Savarino et al for their interest in our study on Helicobacter pylori and gastric metaplasia in the duodenum.
Acid and gastric metaplasia in the duodenum.

V Savarino, G Mela, G Celle and S Vigneri

Gut 1994 35: 1151-1152
doi: 10.1136/gut.35.8.1151-b

Updated information and services can be found at:
http://gut.bmj.com/content/35/8/1151.3.citation

These include:

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/