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 saved our patient from surgical intervention.

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 al1 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

Acid and gastric metaplasia in the duodenum

Editor,—We read with great interest the paper by Noach et al (Gut 1993; 34: 1510–4). The relation between duodenal gastric metaplasia and Helicobacter 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 duodenal H pylori 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. 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 2.6 (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 output4 and in 24 hour intragastric acidity5 between H pylori positive and negative patients. Finally, it is well known that only some patients with duodenal ulcer disease secrete an amount of gastric acid higher than normal6 and only a subgroup of them shows a rapid gastric emptying,7 which could result in an increased acid load to the duodenum with subsequent mucosal injury.

On the basis of these findings, the common belief that gastric metaplasia in the duodenum is induced by hypersecretion of gastric acid is questionable.

The finding of the lack of regression of gastric metaplasia in the bulb after 12 months of eradication of H pylori has been explained by the authors by the fact that gastric acid secretion is not changed by treatment of the infection10 and therefore, the persistent high production of acid helps to maintain the histological duodenal alteration. As acid hypersecretion is not the rule in duodenal ulcer, however, and is not present in functional dyspepsia, it is again difficult to claim this physiological abnormality as the main cause of maintenance of gastric metaplasia in the long term.

Thus, it seems that the presence of H pylori, by itself, is the main factor in the development of duodenal ulcer, although the mechanisms of this action are far from clear. It is suggested by the authors, the mucosal production of cytokines and specific toxins released by the bacterium might be possible ulcerogenetic factors, independent of gastric metaplasia. The persistence of this epithelial change in patients where H pylori has been eradicated who show a dramatic reduction in ulcer relapse, seems to diminish further its pathogenetic relevance.

V SAVARINO
G MELA
G CELLE
Department of Internal Medicine,
University of Genova, Italy
S VIGNERI
Institute of Internal Medicine and Geriatrics,
University of Polermo, Italy


Reply

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