LETTERS TO THE EDITOR

The knee-elbow position relieves distension

EDITOR,—In their article in *Gut* 1993; 34: 1726-7 Panos et al described colon decompression after assuming a knee-chest position and state they are ‘not aware of any previous reports of its use ... in toxic megacolon’ and suggest that a prospective assessment of outcome is appropriate.

Unfortunately, for the authors, little is new in clinical medicine and a similar technique has already been well described in our paper.1 We had also cited a previous paper by Kramer and Wittenberg2 describing redistribution of gas in toxic megacolon by changing the anatomical position. Kramer and Wittenberg did not clinically apply this technique to their patients.

Panos et al also requested longterm follow up, which was presented in our paper in 19 patients in which 13 (68%) treated with this technique did well and did not require surgery (mean follow up 6-5 years). Since this report we have treated at least 15 more patients with this technique and have never had to perform emergency surgery for persistent colonic dilatation, although some patients require surgery for persistent activity after decompression.

Fortunately for inflammatory bowel disease patients, this article hopefully will reawaken interest in the comprehensive medical treatment programme required to treat fulminant colitis and toxic megacolon. Many patients will respond to intensive medical treatment and thus avoid emergency surgery. This comprehensive programme including the rolling technique is detailed in a more recent publication.3

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EDITOR,—We have recently used the intriguing knee-elbow position described by Dr Panos (*Gut* 1993; 34: 1726-7) in a 45 year old man with acute colitis and progressive dilatation of the sigmoid colon.

He presented with a seven week history of worsening diarrhoea. Over the preceding week he had passed 10 stools a day containing blood and pus. On examination, he was afebrile, with no tachycardia and his abdomen was soft with normal bowel sounds. Sigmoidoscopy showed a granular friable mucosa with spontaneous haemorrhage ahead of the instrument. Histological tests were consistent with active ulcerative colitis. Haemoglobin was 13.1 g/dl, platelets 509×10⁹/l, C reactive protein 207 mg/l, α1 acid glycoprotein 2275 (normal range <1150) and albumin 36 g/l. No pathogens were identified on stool culture. Plain abdominal x-ray showed a 8.5 cm dilated sigmoid loop (Fig (A)). He was treated with bed rest and intravenous hydrocortisone 100 mg 6 hourly with metronidazole 500 mg 8 hourly.

Two days after admission he developed abdominal discomfort and distension. Abdominal x-ray confirmed persisting sigmoid dilatation (Fig (B)). He was instructed to adopt the knee-elbow position for five minutes every half hour. In this position he passed large quantities of flatus often triggered by coughing. Four hours later he felt more comfortable with a soft abdomen. A repeat x ray showed decompression of the sigmoid loop (Fig (C)).

Six days after admission his abdomen again became distended and again responded to adoption of the knee-elbow position (Fig (D) and (E)). After nine days his bowel had returned to normal and he was discharged with a reducing course of oral prednisolone.

Acid and gastric metaplasia in the duodenum

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) than normal (4.0 ± 0.2). 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 normal5 and only a subgroup of them shows a rapid gastric emptying,6 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 seems 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. As suggested by the authors, the mucosal production of cytokines and specific toxins released by the bacterium might be possible ulcerogenic factors, independent of gastric metaplasia. The persistence of this epithelial change in patients where H pylori has been eradicated who do a dramatic reduction in ulcer relapse, seems to diminish further its pathogenetic relevance.

Reply

We welcome the correspondence on our case report, which gives us the opportunity of further discussion of 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 glass bed. 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 spare 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 of the use of the knee-elbow position to decompress the bowel in toxic megacolon.

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. 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. Using ambulatory pHmetry 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) than normal (4.0 ± 0.2). 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 and in 24 hour intragastric acidity 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 normal and only a subgroup of them shows a rapid gastric emptying, which could result in an increased acid load to the duodenum with subsequent mucosal injury.

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