find such a good correlation between motility and ulcer pain\(^8\) and although in our own studies the use of an anti-spasmodic did not affect the development of acid induced duodenal ulcer pain,\(^9\) we feel that duodenal wall tension or dysmotility deserve further study as a possible cause of duodenal ulcer pain and a possible explanation for the imperfect correlation between the pH around the ulcer crater and development of ulcer pain in patients with symptomatic duodenal ulcer.

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Malabsorption and motor dysfunction in patients with small bowel amyloidosis

EDITOR,—It was with great interest that we read the article by Tada et al (Gut 1993; 34: 1412–7). The authors confirm our findings that it is not only familial forms of amyloidosis that cause intestinal motor dysfunction. In their clearly large series of patients they showed that amyloidosis causes either myopathy or neuropathy and that chemical types of amyloid, AL and AH, and AA respectively may determine which of the two factors will predominate the pathological findings and thus have repercussions on the clinical signs.

Recently we reported two cases of a non-familial form of amyloidosis with malabsorption and motor dysfunction.\(^1\) The patient diagnosed having Waldenström disease and AL amyloidosis was investigated for both cases of AL amyloidosis on the other hand, patient diagnosed with Bechterev’s disease and AA amyloidosis suffered from untreated ferriprive anaemia but also with difficulties of swallowing, bouts of diarrhoea, and episodes of abdominal cramps. Malabsorption tests, among others the bile acid breath tests suggested malabsorption caused by motor dysfunction or bacterial overgrowth, or both. Amyloid deposits in the small intestine were shown in the submucosal blood vessels in case one (endoscopic biopsy specimens) and in the muscularis mucosae, the myenteric plexus, and submucosal blood vessels in case two (necropsy material).

Unlike Tada et al we cannot, however, confirm the difference in prognosis in the advantage of the AA amyloidosis. All their AL (n=2) and AH (n=1) cases died during follow up, while 62% of the AA cases (n=8/13) survived. The follow up of our first case (four years) was long but uneventful.

In the second case the clinical situation remained fairly stable for five years. Thereafter ischaemia induced necrosis of the terminal ileum was responsible for faecal peritonitis and death of the patient.

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Iron deficiency anaemia

EDITOR,—I enjoyed the excellent leading article by Drs Sayer and Long (Gut 1993; 34: 1297–9), but differ from them when they state that factor ‘haemuria alone seems unlikely to cause iron deficiency anaemia’. I showed that anaemia was 18 times more common in patients with hiatus hernias large enough to be seen on a plain chest x-ray, than in age and sex matched controls.\(^1\) Subsequently, Dr G Higgins and I prospectively evaluated 109 patients with large hernias, having at least one third of the stomach above the diaphragm.\(^2\)

Endoscopy in these patients showed a correlation between anaemia and the presence of superficial linear erosions of the gastric mucosal folds where the body of the stomach was constricted by the diaphragm. We suggested that these lesions result from local trauma. Anaemia did not correlate with oesophagitis or peptic ulcers. We confirmed earlier findings by Holt et al that repair of the hernia prevented recurrence of anaemia.\(^3\)

Having observed small bowel amyloidosis in which no other cause for anaemia was found, my colleagues at this institution have generally accepted that large hernias can cause iron deficiency anaemia secondary to the erosions we described.

In my experience, this is a common cause of chronic blood loss anaemia in older patients.

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Reply

EDITOR,—We are grateful to Drs Ectors, Geboes, and Rutgeerts for their comments on our paper. In patients with every type of amyloidosis, the prognosis seems to depend on the extent and degree of amyloid deposits. Pathological examination of the necropsy specimens in our AL and AH cases showed extensive infiltration and replacement of the muscularis propria by amyloid deposits throughout the gastrointestinal tract. Therefore we thought that they did not respond to any treatment and had a poor prognosis. Varying degrees of amyloid deposits in the gut, however, have been found among the patients with amyloidosis. In the patients with AL or AH, as shown in a case of Fraser et al\(^4\) some treatments may be effective and improve the life span until the amyloid deposits become extensive. As in the AA case of Ectors et al\(^5\) submucosal vascular deposits, which were commonly and notice-ably found in our AA cases, may produce life threatening symptoms. In amyloidosis patients with clinical manifestations of intestinal pseudo-obstruction, however, life treat ment may be effective and improve until the presence of AL amyloidosis than in AA amyloidosis.

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Reply

EDITOR,—We would like to thank Professor Kang for his constructive comments on our article. He suggests that the pain commonly experienced by patients with healed duodenal ulcers may be explained by erosive duodenitis producing small breaches in the mucosa. It does seem possible that such breaches may occur in severely inflamed duodenal mucosa and that they would be too small to be seen on endoscopic examination. Indeed, small and scattered breaches in the mucosa could also be missed with microscopic examination of endoscopic biopsy specimens because of the very small area of mucosa examined in this way. These small breaches would permit acidic gastric juice to penetrate the underlying inflamed mucosa and thus possibly lead to the pain. Such a process could occur both in severely inflamed gastritis as well as in peptic ulcer disease and hence we suggest that such pain could result from the interaction of inflammation and acid at either site.

Microscopic breaches in the mucosa would be more likely in severely inflamed mucosa and thus the occurrence of pain may relate to the severity of mucosal inflammation as well as the degree of hyperacidity.

Such small breaches in the stomach or dysmotility may play a part in duodenal ulcer pain. Such distorted motility probably arises from neuronal stimuli triggered by the interaction of acid with the inflamed mucosa and would thus be a secondary, rather than primary, event. We agree, however, that the pain could originate from either events at the mucosal level or from disturbed muscle function secondary to these mucosal events.