LETTERS TO THE EDITOR

Painful rib syndrome

EDITOR,—The painful rib syndrome recently described by Scott and Scott is, in my opinion, a misnomer. Over the years I have seen numerous cases similar to the ones they report, and have found that the tender spots they allude to are not in the ribs but in the muscles. They are, in fact, myofascial trigger points. Pain develops because of trauma induced activation of nociceptors at these sites in what is now called the myofascial pain syndrome.¹ These trigger points may be found in any muscle in the body. In the abdomen they commonly occur in the rectus abdominis and external oblique muscles. They do not only develop, however, at or near to their insertion into the ribs, but also in their bellies and at lower attachment sites such as the iliac crest, inguinal ligament, and pubic bones. The pain emanating from trigger points in this syndrome may be abolished by injecting a local anaesthetic into them.² Recently it has been shown that pain is also relieved by stimulating A-delta fibre nerves at these sites with dry needles; treatment that is physiologically more rational besides being simpler, safer, and equally effective.³

Gastroenterologists must learn to recognise 'trigger point pain' because it is common and can be treated. The concept of the painful rib syndrome restricts the diagnosis to pain in the lower thorax and upper abdomen, as well as implying that there is no effective treatment other than reassurance. Trigger point pain may occur anywhere in the abdomen with additional sites in the perineum and back. The pain can be recognised easily so unnecessary investigations and operations are avoided. It usually responds quickly to acupuncture; further courses can be given if relapse occurs.

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Painful colon syndrome

EDITOR,—The term hypochondriacal flatulentumque morbum¹² has been used synonymously by many authors for what we now call colic. Various conditions have been described in this syndrome.² It is therefore possible that the condition described by Scott and Scott (Letters, 1995; 34: 1006–8) is a case of this syndrome.²

It is perhaps helpful to look at history. The region at or below the cartilaginous parts of the ribs is also known as the hypochondrium. From the Greek hypo=below and chondros=cartilage. It was Galen from Pergammon (living AD 129–199) who first described a syndrome at this location consisting of pain in the region below the ribs, bloating, and anxiety. He coined the term hypochondriacal flatulentumque morbum.¹

In Graeco-Roman times, hypochondria was considered a part of melancholia—what we today call depression. Today, the meaning of the word hypochondria has changed. In the eighteenth century, hypochondria still had the antique denomination.³ As hypochondria was (or is?) particularly common in England, it has been described as the English malady.¹ I would like to suggest that Scott and Scott will find the pathway of their syndrome when they obtain a medical history looking for signs of depression.

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³ Cheryne G. The English malady: or, a treatise of nervous diseases of all kinds, as spleen, vapours, lameness of spirits, hypochondria and hysterical distempers, etc. London/Dublin: 1733.

Colonoscopic surveillance in ulcerative colitis

EDITOR,—We read with interest the article by Lynch et al (Gut 1993; 34: 1073–80), and agree that the problem of defining those patients at risk of developing colorectal cancer poses great logistical problems. Yearly surveillance colonoscopy did not detect most of the cancers in patients with colitis, but this was because nearly all patients in whom cancer eventually occurred fell outside their surveillance programme. Only three of nine patients who developed colon cancer had their disease initially assessed by colonoscopy, and in a further two patients had total colitis diagnosed by barium enema. We would suggest that ideally all patients with an initial diagnosis of colitis should have the extent of their disease assessed colonoscopically, thereby better defining those patients deemed to be at higher risk of developing cancer.

It is obviously true that colonoscopy will not prevent cancer from developing in the colitic colon because of the imperfect link between dysplasia and cancer, and because of the low proportion of the surface area of the colon biopsied during surveillance colonoscopy. We would therefore propose that surveillance colonoscopy should not be the only following programme that colitic patients receive. It is artificial to separate colonic surveillance from proper clinical care of a patient with a condition that relapses and remits, and during which medical treatment may have to be changed. A large prospective study of follow up of patients with colitis, 13 of 17 cancer patients in the surveillance programme had a Duke's A/B cancer, suggesting that this group of patients will have a better outlook than those patients presenting symptomatically.¹ The finding of dysplasia in 22 patients treated by colectomy would possibly have prevented at least seven relapses.¹ The authors have concluded that an aggressive policy of colorectal cancer prevention had a much higher incidence of early tumours compared with a non-prevention group, which translated into a survival advantage at five years that was statistically significant.¹

We feel that Lynch et al have been too pessimistic in their article on the value of follow up of colitis patients. Colonoscopy performed every two years from the time of diagnosis is clearly feasible, requiring 12 colonoscopies per 100,000 population.¹ All authors agree that the risk of developing colorectal cancer increases with duration and extent of disease.² In addition, onset of colitis is also associated with increased risk,³ although other studies suggest that older age of onset of colitis may be associated with a shorter interval to development of cancer.² We do agree, however, that there is a need for follow up of patients with colitis deemed to be at high risk of developing colorectal cancer needs further thought and study. In the meantime, there is a need for high risk patients from the 10th year after onset of colitis seems to be a sensible approach.

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Reply

EDITOR,—Thank you for the opportunity of replying to Messrs Rutter and Leicester's letter. We agree with much they say. In our paper we advocated longterm clinic follow

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