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 nerve fibres 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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- 1 Simons D. Muscular pain syndromes. In: Friction JR, Awad E, eds. *Advances in pain research and therapy*. New York: Raven Press, 1990: 1-41.
- 2 Friction JR. Management of myofascial pain syndromes. *Ibid* 325-46.
- 3 Baldry PE. Acupuncture, trigger points and musculoskeletal pain. 2nd ed. Edinburgh: Churchill Livingstone, 1993.

EDITOR,—Scott and Scott review what they call the painful rib syndrome (*Gut* 1993; 34: 1006-8), consisting of three features: pain in the lower chest or upper abdomen, a tender spot on the costal margins, and reproduction of the pain on pressing the tender spot. In the discussion, it is stated that the cause of this syndrome is not known.

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 Greek hypo=below and chondros=cartilage. It was Galen from Pergamon (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 hypochondriacum flatulentumque morbum.^{1,2}

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 denotation.³ As hypochondria was (or is?) particularly common in England, it has been described as the English malady.³ I would like to suggest that Scott and Scott will find the aetiology of their syndrome when they obtain a medical history looking for signs of depression.

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- 1 Claudii Galeni. *Opera omnia*. Kühn G, ed. Lpz. 1821-33.
- 2 Fischer-Homberger E. *Hypochondrie*. Bern: Huber, 1970.
- 3 Cheyne G. *The English malady: or, a treatise of nervous diseases of all kinds, as spleen, vapours, lowness of spirits, hypochondriacal, and hysterical distempers*, etc. London/Dublin: 1733.

Reply

EDITOR,—It is encouraging that others readily recognise the syndrome we described. It is also interesting that Dr Dyer sees this as part of a wider syndrome and it behoves all clinicians to keep this in mind when confronted with patients who have pain that does not readily fit other well defined categories. At the least it may prevent unnecessary investigations, and it may even lead to effective treatment. Our study did not look into the aetiology and Dr Dyer's concept of myofascial trigger points is plausible. Although reassurance and explanation is probably sufficient for most patients, some remain troubled and for them acupuncture is possibly appropriate. We agree with Professor Feurle that depression possibly plays a part in this syndrome, but doubt that it is an important part. Clinical depression was not a prominent feature among our patients at the time of examination although 28% gave a history of either depression or anxiety.

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Colonoscopic surveillance in ulcerative colitis

EDITOR,—We read with interest the article by Lynch *et al* (*Gut* 1993; 34: 1075-80), 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 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

colon¹ 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 follow up that colitic patients receive. It is artificial to separate colonoscopic 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. In the largest 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.² The finding of dysplasia in 22 patients treated by colectomy would possibly have prevented at least seven cancers.² A recent study has also 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.³

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 10th year after diagnosis is clearly feasible, requiring 12 colonoscopies per 100 000 population.⁴ All authors agree that the risk of developing colorectal cancer increases with duration and extent of disease.⁵ In addition, young age of onset of colitis is also associated with increased risk,⁶ although other studies suggest that older age of onset of colitis may be associated with a shorter interval to development of cancer.⁷ We do agree, however, that the strategy 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, surveillance of high risk patients from the 10th year after onset of colitis seems a sensible approach.

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- 1 Catnach SM, Rutter KRP, Bown RL. Colorectal carcinoma in patients with ulcerative colitis and recent colonoscopy. *Gut* 1993; 34: 1148-9.
- 2 Lennard-Jones JE, Melville DM, Morson BC, Ritchie JK, Williams CB. Precancer and cancer in extensive ulcerative colitis: findings among 401 patients over 22 years. *Gut* 1990; 31: 800-6.
- 3 Giardiello FM, Gurbuz AK, Bayless TM, Yardley JH. Colorectal cancer (CRC) in ulcerative colitis (UC): effect of a cancer prevention strategy on survival. *Gastroenterology* 1993; 104 (suppl): A705.
- 4 Jones HW, Grogono J, Hoare AM. Surveillance in ulcerative colitis: burdens and benefit. *Gut* 1988; 29: 325-31.
- 5 Sugita A, Sachar DB, Bodian C, Ribeiro MB, Aufses Jr AH, Greenstein AJ. Colorectal cancer in ulcerative colitis. Influence of anatomical extent and age at onset on colitis-cancer interval. *Gut* 1991; 32: 167-9.
- 6 Ekblom A, Halmick C, Zack M, Adami H-O. Ulcerative colitis and colorectal cancer. *N Engl J Med* 1990; 323: 1228-33.
- 7 Gyde SN, Prior P, Allan RN, Stevens A, Jewell DP, Truelove SG, *et al*. A cohort study of primary referrals from three centres. *Gut* 1988; 29: 206-17.

Reply

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

up of all colitic patients and at least one colonoscopic assessment for each. We disagree, however, that colonoscopy should necessarily be undertaken at initial diagnosis. The extent discovered at the outset of the disease can be misleading because colitis may extend proximally with time. For this reason we recommended colonoscopy and biopsy at eight years disease duration and clinical follow up indefinitely thereafter.

A colonoscopic surveillance programme means subjecting patients to a regular examination regardless of symptoms. This contrasts with a policy of close clinical follow up where patients are investigated when symptoms arise. This separation is not artificial. Indeed, it is important to distinguish the two forms of patient management to determine the effectiveness of each, particularly when considering the cost of colonoscopic surveillance. Messrs Rayter and Leicester state that in one study¹ 13 (the number is actually 12) of 17 cancer patients detected by surveillance had Duke's A/B cancer suggesting a better outlook for these patients. Analysis of the data shows that only one, or possibly two, of the 12 early cancers were detected by 'surveillance' colonoscopy. The remainder were identified by other, non-surveillance, means – that is, cancers found at colonoscopy performed because an abnormality was detected at barium enema or sigmoidoscopy, some found by chance after operations for 'disability', and another on screening colonoscopy on initial referral.

The number of patients found to have high grade dysplasia at colectomy is 12 (and not 22) though it is not made wholly clear how the dysplasia was detected in some patients – that is, by surveillance or non-surveillance methods. As our colleagues from St George's admit there are problems with the diagnosis of dysplasia, its imperfect link with carcinoma, and the unpredictable nature of its outcome. Though a possible four early cancers may have been prevented this does not change the poor detection rate of early cancers by colonoscopic surveillance.

We agree that an aggressive policy for colorectal cancer prevention is needed. Our experience, however, and a review of published works, suggests that colonoscopic surveillance does not work nor is it cost effective.² Furthermore, even when carried out effectively it may contribute no additional benefit to routine clinical care in reducing deaths from colorectal cancer.³

Contrary with the opinion expressed we are not pessimistic about the value of longterm follow up of colitis patients. We have shown that an expensive, widely advocated, labour intensive programme does not work. The resources saved by stopping colonoscopic surveillance are considerable and could be channelled into more effective measures of preventing people with colitis from dying of cancer.

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1 Lennard-Jones JE, Melville DM, Morson BC, Ritchie JK, Williams CB. Precancer and cancer in extensive ulcerative colitis: findings among 401 patients over 22 years. *Gut* 1990; 31: 800–6.

2 Lynch DAF, Lobo AJ, Sobala GM, Dixon MF, Axon ATR. Failure of colonoscopic surveillance in ulcerative colitis. *Gut* 1993; 34: 1075–80.

3 Lashner BA, Kane SV, Hanauer SB. Colon cancer surveillance in chronic ulcerative colitis: historical cohort study. *Am J Gastroenterol* 1990; 85: 1083–7.

BOOK REVIEWS

Cancer of the Esophagus: approaches to etiology. By Valda M Craddock. (Pp 282; illustrated; £60.00.) Cambridge, Cambridge University Press, 1993.

Despite the spelling, this book is written by the senior scientist of the MRC Toxicology Unit in the UK about cellular molecular biology and the effects of *N*-nitroso compounds on the basal cell layer of the oesophagus. The author worked with Barnes and Magee who discovered in 1956 that the simple inert chemical compound dimethyl nitrosamine was carcinogenic. Since then, more than half of the 300 *N*-nitroso compounds have been found to be carcinogenic. This book gives an updated description of the extrinsic factors possibly related to the aetiology of oesophageal cancer and shows the experience and wide range of knowledge of the author that is essential for epidemiological research. At first sight, the oesophagus is an unsuitable organ for studying carcinogenesis especially to those who consider it as a hollow tube about 25 cm long with no other function than to convey nutrition into a much more useful organ – the stomach. That this requires a journey of less than 10 seconds may seem to be hardly enough time for causing cancer even if you do swallow 1–2000 times a day. Yet, worldwide, the incidence of oesophageal cancer ranges from less than 1 per 100 000 to over 270 per 100 000. This discrepancy has stimulated the search for extrinsic factors. Do alcohol, smoking, opium, betel nut, and pickled chinese cabbage all mediate their effects through *N*-nitrosamine?

Here, you can read all about it. The applied biochemistry will explain much and is described intelligibly for non-specialists. Humble clinicians are very aware of the danger of alcohol, its relation to strength and quantity (the whisky distillery workers around Aberdeen), its multiplier effect with smoking (the UK), the South African alcoholic beverages brewed in old tar barrels, and the high incidence in Normandy related to Calvados. This last point shows the need for some biochemistry. Apple brandy has 152–308 mg/100 ml of fusel alcohols and high concentrations of tannin. The apples for cider making have even higher concentrations of tannin and are highly resistant to birds but cider does not seem to cause oesophageal cancer. The tannins, black and green tea, are discussed and the tannin in the betel leaf, which surrounds the crushed betel nut for chewing seems protective. Red wine contains more tannin than white wine and this is related to the grape skins. But the high concentration in Scotch whisky may result from the tannin in the oak of the barrels in which the whisky is matured. Such is the detail of the possible aetiologies of oesophageal cancer discussed in this book.

The epidemiology is not, however, complete. There is further detail about geographical variation of the disease in gastroenterological and surgical works. So there should be, as a new textbook on the oesophagus appears each year and clinicians invade the 'cosa nostra' territory of the epidemiologists.

Some of the clinical findings and reports of gastroenterological research are also not com-

plete. Carcinogenesis in Barrett's oesophagus, the risk thereof, the epidemiology (Cameron is not mentioned in the book), the mechanism and the aetiological agents have all been the subject of intense discussion among clinicians because factors are important for the aetiology of cancer both in squamous and columnar epithelium. There is hardly a mention of this work here. The final criticism is of the abysmal index, which is so incomplete as to be dangerous and certainly does not do justice to the text. Each chapter has numerous up to date references and the book is a modern compendium of knowledge essential for discussing the aetiology of oesophageal cancer. The data are locked away in the text but inaccessible because the index is incomplete. This book is to be recommended for clinicians and our next steps are to merge this database with the epidemiological and clinical databank, index it for retrieval, and make it widely accessible. Such steps will lead clinicians, with experts such as Dr Craddock by their side to the aetiology of oesophageal gastric and colonic cancer.

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Hepatic Transport and Bile Secretion: Physiology and Pathophysiology. Edited by Nicola Tavoloni and Pawl D Berk. (Pp 791; illustrated; \$249.) New York: Raven Press, 1993.

Tavoloni and Berk set themselves the task of 'highlighting the recent advances made in our understanding of hepatic physiology and its related diseases. With the help of 99 experts, they achieve their aim in 49 chapters.

Therein lies the importance of this book, each chapter is comprehensive in its cover, duplication is rare, and partisanship not glaringly obvious. Each bibliography has a fair sprinkling of 1990 and 1991 references – even 1992 is there, at least twice. The book begins with the 'Functional morphology of the liver with emphasis on microvasculature'. For a biochemist (like myself) this is an essential and exciting chapter, its high standard of illustration is maintained throughout the book. While the mathematical expertise demanded by the two chapters on the modelling of hepatic transport is daunting perhaps even 'chaotic', the critical appraisal of the usually tacit assumptions is easily understood and provides an evaluation not readily available elsewhere.

In addition to traditional topics like bilirubin, bile acids, bile flow, fatty acid binding proteins, there are surprise chapters on such diverse subjects as 'Fluid-phase endocytosis', the 'Use of membrane vesicles to study hepatic transport', and the 'Molecular biology of the uridine diphosphate glucuronyltransferases'.

Unfortunately, the index is not as comprehensive as the text. For example cholehepatic shunting is not in the index but of course is discussed in the chapter on 'Hepatic secretion of bile acids'. But surprisingly there is nothing in this book on ursodeoxycholic acid therapy for cholestatic liver disease. Was this a chapter too late?

This book is for the academically minded clinician as well as the researcher who while focusing on one small aspect wonders what the rest of the liver does and how. It will provide excellent material for the medical school teacher for many years to come. A snip at \$249 – well what can you get for £168 nowadays?

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