LETTERS TO THE EDITOR

Colorectal adenoma–carcinoma sequence

EDITOR,—I read with interest the article by Fernandez-Baneres et al (Gut 1996; 38: 254–9). These authors demonstrated novel significant differences in tissue fatty acid profiles when they compared diseased and paired normal mucosa of adenoma and carcinoma patients. My one concern about this study is the author’s comparison between tissue fatty acid profiles with plasma fatty acid concentrations that only reflect recent intake and give no information on the longterm dietary intake of n3 fatty acids. I feel that it would be more appropriate to compare their tissue fatty acid profiles with red cell fatty acid levels.

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Reply

EDITOR,—Mr Khosraviani points to the necessity of assessing longterm fatty acid intake in our patients by measuring the red cell fatty acid profile. However, the observed changes in the fatty acid profile in the diseased mucosa from both adenoma and carcinoma cannot be attributed to different dietary intake, because we compared it with the fatty acid profile of the normal mucosa surrounding both adenomas and carcinomas—that is, the comparison was made between two tissues obtained from the same patient. In any case, the fatty acid profile in the normal colonic mucosa probably gives better information about longterm dietary intake than the fatty acid profile in red cells, which is more influenced by plasma fatty acid concentrations.

It should be also emphasised that we did not compare, as Mr Khosraviani states, plasma versus tissue fatty acid profile. We merely described the fatty acid pattern in plasma phospholipids, which is a reflection of recent fatty intake and also of tissue fatty acid values and metabolism.

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Proximal colonic motility

EDITOR,—We were most interested by the manometric method developed by Lemann et al for studying proximal colonic motility reported in the journal (Gut 1995; 37: 649–53). We must take issue however with their statement that ‘the placement of recording probes introduced through the anus with the aid of colonoscopy requires premedication, air insufflation, and prior preparation to ensure vacuity of the colon’. The method used in this department for over 10 years for studying distal colonic motility has been the placement of four perfused manometry catheters 15 to 50 cm into the colon by flexible sigmoidoscopy without sedation or bowel preparation. Following placement the position of the catheters is checked using fluoroscopy. This method is associated with a high rate of success and produces little discomfort. Studies using this method have been published in this journal1–3 and elsewhere.4

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BOOK REVIEWS


The medical and scientific communities are already well served with original articles, reviews, book chapters, and monographs on gall stone disease. That being the case, when a new volume on this topic appears, one must ask: is it needed, what is new, and who will benefit from it? Despite some undoubted virtues, the answers to these rhetorical questions are far from clear.

The title of the book may be misleading as it deals with much more than ‘techniques’. Although it is written predominantly by surgeons, it is not just about operative methods. Rather, in its 23 chapters, it covers a broad range of topics written by many distinguished contributors. Although two of the editors are now working in the United Kingdom, their FRCSI diplomas suggest that they have a common training in Ireland. Indeed, no less than 14 of the 41 contributors have degrees and diplomas that suggest a background in the Emerald Isle, which may well explain the matching green colour of the book.

The volume begins with a chapter on pathogenesis, which is well written and liberally documented with references—albeit with a surgical, rather than a medical or basic scientific, bias. The second chapter is entitled ‘Natural History’ but it comes to a stop midway through this term of reference with a rather superficial repetition of pathogenesis, which, arguably, is not relevant to natural history. It also contains unusual statements, which, sadly, are not referenced—such as the suggestion that gall stones may fragment spontaneously, and that the stress of surgery is a ‘tissue-promoting’ factor.

Once again there is a surgical bias that ignores, for example, data on natural history gained from more than 300 patients given a placebo in the National Co-operative Gallstone study (which cost the US taxpayer $12 million). Contrast this with near anecdotal accounts cited in the chapter of the natural history of stones based on four ‘series’ of 11, 17, 23, and 25 patients, or with information about intravenous cholangiography in 11 000 patients studied by the author of the chapter himself. From this position of undisputed surgical expertise, the author returns to rather the red cell fatty acid profile. However, the observed changes in the fatty acid profile in the diseased mucosa from both adenoma and carcinoma cannot be attributed to different dietary intake, because we compared it with the fatty acid profile of the normal mucosa surrounding both adenomas and carcinomas—that is, the comparison was made between two tissues obtained from the same patient. In any case, the fatty acid profile in the normal colonic mucosa probably gives better information about longterm dietary intake than the fatty acid profile in red cells, which is more influenced by plasma fatty acid concentrations.

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