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?

G M MURPHY