really needed to confirm that this is good clinical practice?

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Re-epithelialisation of Barrett's oesophagus

EDITOR,—We were interested to read the case report by Van Laethem and colleagues of a carcinoma arising under a re-epithelialised segment of Barrett's oesophagus (*Gut* 2000;**46**:574–577). This raises issues in the debate over ablation of Barrett's epithelium. There has been interest in ablating the columnar epithelium to encourage squamous regrowth which may reduce the risk of progression to adenocarcinoma. However, there have been numerous reports of buried glands under the regenerated mucosa.[1–3]

While we accept that columnar glands may persist under the squamous epithelium and that this may represent a continuing carcinoma risk, this is difficult to quantify. Indeed, this is the first report of such a malignant change. It may be that as any buried glands are no longer exposed to potential carcinogens in the form of acid or bile reflux, the risk is reduced. Although the ultimate aim of treatment is to eliminate the risk of potential malignant change, any means of reducing such risk, for example by diminution of the volume of metaplastic tissue, would be worthwhile. This whole issue needs further evaluation by appropriately designed clinical trials.[4]

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Outcome of lamivudine resistant hepatitis B virus infection in liver transplant recipients in Singapore

EDITOR,—We read with interest the article by Mutimer and colleagues (*Gut* 2000;**46**:107–113). The Birmingham group described the clinical course of four liver transplant patients who developed graft infection with lamivudine resistant virus. Lamivudine resistant hepatitis B developed after a mean duration of nine months (range 8–11) after the transplant. Liver function abnormalities occurred at a mean duration of six months (range 3–12) after the emergence of lamivudine resistant virus and three of the four patients died 5–20 months later. The authors concluded that the lamivudine resistant phenotype can cause severe graft damage.

In our liver transplant centre, 12 patients with chronic hepatitis B (four with hepatocellular carcinoma) underwent liver transplantation over a five year period. All were given lamivudine before and after transplant. Lamivudine resistant hepatitis B developed in six of the nine survivors at a mean duration of 60 weeks (range 1–127) after liver transplant. Apart from weaning off immunosuppression aggressively, no further antiviral treatment was added. All six had normal liver function at their last follow up (mean 28, range 0–123 weeks after emergence of lamivudine resistant virus).

Contrary to what the Birmingham group experienced, all of our patients with lamivudine resistant virus were well, with no evidence of graft dysfunction. Long term outcome of such patients remains unknown and it may be premature to conclude that the lamivudine resistant phenotype causes severe graft damage.

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Gastric cancer in patients with benign dyspepsia

EDITOR,—There is an ongoing debate regarding the value of endoscopy in younger patients presenting with dyspepsia. One important consideration is the likelihood of detecting an underlying cancer which might be cured by early treatment. The large retrospective study by Breslin and colleagues in the January issue of *Gut* (*Gut* 2000;**46**:93–97) indicates that underlying cancer will be diagnosed in about 1 in 1000 patients presenting with uncomplicated dyspepsia under 45 years of age. However, the calculated 95% confidence intervals for this are wide (1 in 2963 to 1 in 300).

An important question in considering the significance of this finding is whether the prevalence of cancer in these patients with benign dyspepsia is any different from that in the general population. In our own country, Scotland, the chance of a patient presenting with gastro-oesophageal cancer before the age of 50 is 1 in 909 (ISD Scotland Cancer Surveillance Group Data Request and Analysis Service) and half of those have presented with the cancer within the age band 45–49. Most of these patients will have had the tumour present in their stomach for a considerable time prior to clinical presentation, which would have been detected by screening endoscopy five years earlier. Even allowing for the fact that population based rates of gastro-oesophageal cancer are higher in Scotland than Alberta,[1] this suggests that the prevalence of underlying cancer in patients presenting with uncomplicated dyspepsia may not be different from that in the general population. Consequently, offering endoscopy to patients with simple uncomplicated dyspepsia to detect cancer may merely represent screening of the general population.

There has been a general assumption that a tumour growing in the stomach will produce dyspeptic symptoms. However, there is no evidence for this. Tumours developing in the colon or other parts of the gastrointestinal tract rarely, if ever, cause symptoms until they produce complications such as bleeding or obstruction.

A very small proportion of patients presenting with uncomplicated dyspepsia will have underlying cancers but this finding may be unrelated to their symptoms. Unless uncomplicated dyspepsia is confirmed to be a symptom of underlying malignancy, then one would be as well to recommend offering endoscopy to patients presenting with a sprained ankle in order to pick up underlying gastro-oesophageal cancer.

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BOOK REVIEW

Colonic Microbiota, Nutrition and Health. Edited by G R Gibson, M B Roberfroid (Pp 304; illustrated; £93) The Netherlands: Kluwer Academic Publishers, 1999. ISBN 0412798808.

I was taught as a medical student that the major function of the colon was that of a storage organ. Since then, premises about the colon have evolved and the complexities of colon function is much better understood, as described by Gibson and Roberfroid's multi-authored book *Colonic Microbiota, Nutrition and Health*.

Although the authors state that the purpose of the book is to overview current knowledge of the activities and functions of the gut microflora, the scope goes beyond these boundaries and takes us on an ecological journey into the exciting life of gut microflora and their impact on colon function in health and disease, and the intimate critical relationship between diet, bacteria, and quality of life.

Gastroenterologists are still recovering from the impact that a bacterium, *Helicobacter pylori*, has had on upper gastrointestinal tract pathology. In this context, it is interesting to note that the large bowel is the most heavily colonised part of the gastrointestinal tract yielding up to 10^{12} bacteria per gram of intestinal contents in healthy human subjects. It is a complex ecosystem in which the numerous