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Duodenal ulcer and carbohydrate

SIR,—We have read with interest the results of the study from Nottingham by Katschinski *et al* (*Gut* 1990; **31**: 993–6) concerning the association between duodenal ulceration and fibre and refined carbohydrate intake. The findings suggested that relative risks were reduced by a high vegetable fibre and low refined sugar intake but not a high intake of cereal fibre.

We have continued to gather information about the geographical distribution of duodenal ulceration and the staple diets of high and low incidence areas, and we find no correlation between duodenal ulcer incidence and fibre intake alone. There are high incidence areas in Ethiopia, Rwanda, and Burundi and in sorghum eating areas of India where the fibre intake is high.

The overall picture suggests that areas where polished rice, yams, or cassava are the staple foods the duodenal ulcer incidence is high. Where unrefined wheat, soya, some pulses or millets, or certain green vegetables form a large part of the staple diet the incidence is low.[1,2]

Experimental work on several animal models of peptic ulceration shows that the food substances mentioned above from low incidence areas contain a protective fraction which is liposoluble. The fraction is present in wheat bran but to a less degree in wheat germ.[3] We think that it is a protective factor present in certain high fibre foods and not the fibre itself that protects against duodenal ulceration.

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Gastric acid and urinary acid excretion

SIR,—Johnson *et al* (*Gut* 1990; **31**: 826–6) could not find a significant rise of urinary pH two hours after the start of a standard meal in normal subjects (despite a reduction in urine acid output) nor in patients after vagotomy. They concluded that changes in the rate of urinary acid output after a meal could not be detected by measuring pH because of the presence of buffers in normal urine. Their findings may reflect the inferiority of a standard meal to pentagastrin for maximal stimulation of gastric acid secretion.

We measured urinary pH in 14 duodenal ulcer patients with no vagotomy (group A) and in 14 patients after vagotomy (group B), before and two hours after pentagastrin 6 μg/kg subcutaneously. Median pH of basal urine in group A was 4·9 (range 3·9–5·7). Two hours after the meal the corresponding values were 6·2 (5·2–7·0). In group B preprandial and postprandial urine pH was 5·3 (4·7–7·0) and 5·3 (4·5–7·0).

Thus the conclusion drawn by Johnson *et al* is correct for pentagastrin stimulation after vagotomy, but not for duodenal ulcer patients who have not had a vagotomy.

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Reply

SIR,—Thank you for the opportunity to reply to these comments. Like us, Dr Niv and colleagues were unable to show a true 'alkaline' tide in the urine after gastric stimulation because their pH values were all 7 or less. Nevertheless, they showed an alkalinisation of the urine, as measured by rising pH in duodenal ulcer patients after pentagastrin stimulation, and were unable to show this effect in duodenal ulcer patients after vagotomy.

It would be interesting to know whether these measurements were taken during a standard gastric secretory study, with aspiration of gastric contents. This manoeuvre will clearly render the body's acid-base balance more alkaline by the removal of the gastric acid secretion. Perhaps this explains why they were able to show a change in pH in non-vagotomised ulcer patients. We maintain, however, that pH is a much less sensitive indicator of the amount of acid excreted in the urine than is direct titration of the acid content of the urine.

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Lipid pattern and plasma insulin in diabetics with gall stones

SIR,—We read with great interest the paper by Laakso and colleagues (*Gut* 1990; **31**: 344–7) regarding the relation between serum lipids, plasma insulin, and gall stones in non-insulin dependent diabetic women. The authors suggest that diabetics with gall stone disease have higher fasting insulin concentrations and lower total and low density lipoprotein cholesterol than diabetics without gall stones.

In the introduction they state that no studies have been published comparing lipids and lipoproteins in diabetics with or without gall stones. Some years ago we reported different results on the relations between gall stones and serum lipids in non-insulin dependent diabetic patients. We studied total cholesterol, serum triglycerides, and apolipoproteins A I and B in 81 subjects with non-insulin dependent diabetes mellitus affected by gall stones and 305 diabetics without gall stone disease.[1] We documented increased concentrations of triglycerides and decreased values of apolipoproteins A I in diabetic women with gall stones compared with those without, while no difference was shown in men. Total cholesterol and apolipoprotein B concentrations did not differ between groups. The observation of high concentrations of triglycerides in gall stones has been reported by most authors; our experience is in agreement with published papers and conflicts with the data reported by Laakso. Our finding of such an association only in women agrees with the observation of more severe lipid alteration in women with diabetes[2] and of an association of gall stones, low concentrations of high density lipoprotein cholesterol, and coronary disease found only in women.[3]

The serum lipid pattern in our patients might be related to increased serum insulin concentrations, as suggested by Laakso *et al* in another paper.[4] In this regard, in a case-control study (34 patients with gall stones and non-insulin dependent diabetes mellitus *v* 30 controls without gall stones, comparable for sex, age, body mass index, and duration and metabolic control of diabetes) we also documented increased values of C peptide in subjects with gall stones compared with controls.[5]

Concerning the possible mechanism by which hyperinsulinaemia could enhance gall stone formation, Laakso *et al* report that high insulin concentrations could activate low density lipoprotein receptors with an increased plasma-bile clearance of low density lipoprotein cholesterol. It, however, has also been reported that insulin is able to enhance the activity of ß-hydroxy ß-methylglutaryl coenzyme A reductase[6] and to suppress 7 alpha-hydroxylase[7] with consequent increased cholesterol and decreased bile acid secretion in bile. According to this finding Bennion and Grundy[8] showed that insulin administration in non-insulin dependent diabetics could increase cholesterol saturation of bile. In a preliminary retrospective evaluation of 386 subjects with non-insulin dependent diabetes mellitus we showed a significantly higher frequency of gall stones in patients treated with insulin compared with those being managed by diet or oral hypoglycaemic agents.[9] This finding seems to support the hypothesis of an increased risk of gall stones in diabetics treated with insulin, but prospective investigations on this topic are necessary.

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Oesophagography and AIDS

SIR,—In a leading article on gastrointestinal tract involvement by AIDS Gazzard contends that oesophagography has such a poor sensitivity and specificity for diagnosing opportunistic – that is, fungal and viral – oesophagitis that it is an unsatisfactory technique for investigating oesophageal symptoms in HIV-positive patients.[1] Recent evidence, however, suggests that double contrast oesophagography is in fact a valuable diagnostic test in these patients. In two separate studies double contrast oesophagography had a sensitivity of approximately 90% in diagnosing *Candida* oesophagitis.[2,3] The major advantage of this technique over conventional single contrast barium studies is its ability to show mucosal plaques that cannot easily be seen with single contrast techniques. As a result, only mild cases of *Candida* oesophagitis are likely to be missed on double contrast examinations. Patients with AIDS often have a more fulminant form of candidiasis in which the oesophagus has an easily recognisable 'shaggy' appearance on oesophagography due to multiple plaques, pseudomembranes, and ulcers.[4] In contrast, herpes oesophagitis is typically seen on double contrast radiographs by discrete, superficial ulcers without evidence of plaques.[5] Recently, cytomegalovirus (CMV) has also been recognised as a cause of viral oesophagitis in HIV-positive patients. Unlike herpes, CMV may be shown radiographically as large, relatively flat ulcers one or more centimetres in size.[4,6] Because herpetic ulcers rarely become this large, the presence of a giant ulcer should be highly suggestive of CMV oesophagitis in patients with AIDS.

A recent study of HIV-positive patients confirmed that these various types of opportunistic oesophagitis can usually be differentiated by their characteristic features on double contrast oesophagrams, eliminating the need for endoscopic intervention in many cases.[4] We therefore believe that double contrast oesophagography is a valuable technique for investigating AIDS patients with oesophageal symptoms. Nevertheless, endoscopy may be required for a more definitive diagnosis if the radiographic findings are equivocal or if the patient fails to respond to appropriate treatment with antifungal or antiviral agents.

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Reply

SIR,—Thank you for giving me the opportunity to reply to the helpful letter of Levine and Herlinger discussing the merits of radiology *v* endoscopy in HIV antibody positive patients. Double contrast barium swallow in expert hands undoubtedly produces fine mucosal detail of the oesophagus, but at the expense of some false positives (5 out of 34) in the study quoted by Levine and Herlinger.[1]

Any assessment of sensitivity only applies to a particular population. Thus in one of the references that they have cited[1] only patients with oesophageal candidiasis were studied and in the other[2] only two of 18 cases had an alternative opportunistic infection (herpes). Had a wider spectrum of patients been chosen the sensitivity for radiology might well have been considerably reduced.

The preference for endoscopy in the diagnosis of HIV-related oesophageal symptoms is pragmatic. The sensitivity is likely to be higher than that for radiology in most hospitals and the capacity to biopsy dubious lesions is a major advantage. The major theoretical disadvantage is the transmission of HIV to staff or other patients – this has not been described.

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BOOK REVIEWS

Hepatology for the clinician. A problem-orientated approach. By S Beker. (Pp 369; illustrated; $96.) New York: Alan R Liss, 1989.

Hepatology for the Clinician, edited by Dr Beker, uses the problem oriented approach and is designed to be read by clinicians caring for patients with liver disease. Its 13 chapters come from authors in the United States, Venezuela, and Japan.

The first thing to be said about this book is that it was a pleasure to read. Its size and the length of the chapters mean that a topic can be read in a short space of time. Most chapters give an insight into how the individual authors tackle a clinical problem rather than give an exhaustive review of the literature on the subject.

Most of the common problems in hepatology are covered with chapters devoted to jaundice, gastrointestinal haemorrhage, ascites, hepatic encephalopathy, acute hepatitis, and chronic liver disease. There are excellent chapters on pregnancy and the liver and on space-occupying lesions of the liver. In a book of this kind some chapters inevitably tend to become a little imbalanced. This is particularly evident in the chapter on ascites where the subject of spontaneous bacterial peritonitis is allocated twice as much space as the discussion of ascites and the hepatorenal syndrome combined. The chapter on the febrile patient and the liver is disappointing as it consists mainly of a listing of infectious disorders involving the liver with descriptions of each. A more problem oriented approach here with perhaps discussion of the management of the patient with established liver disease presenting with pyrexia would, in my view, enhance this book.

Despite these criticisms this is an enjoyable book to read. It gives straightforwrd advice about the approach to and management of problems in patients with liver disease. I think it succeeds in its aim of providing practical guidelines for patient management. It is not a substitute for, nor is it intended to be a substitute for, the more comprehensive textbooks in hepatology. I think that it will be read with enjoyment by gastroenterologists, internists, and fellows in training.

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Radiology of the small intestine. By P Bret, C Cuche, and G Schmutz. (Pp 400; 550 figs; DM398.) Paris: Springer-Verlag, 1989.

This is a welcome translation of one of France's leading gastrointestinal radiologist's work on the small bowel. One of the first impressions of this book is of the outstanding quality of the radiographs. As Igor Laufer mentions in his preface, it is a beautiful work and a pleasure to look through. The book also has a very practical emphasis. Each section finishes with a short paragraph on 'practical conclusions' and the text is full of helpful points on technique, interpretation, and differential diagnosis that reflect the immense experience of the authors. Ultrasound, computed tomography, and arteriography are discussed, though most of the book concentrates on barium studies. It was refreshing not to find a total insistence on the small bowel enema as the only method for examination. I agree with its recommendation for use in obstruction and was particularly pleased to see the insistence on routine compression during examination with 'each loop separated from the neighbouring one by the Holzknecht device.' This is a useful compression device unfortunately not in common use in the United Kingdom. The index is slightly limited with one page references only to main topics, but, as this is not a reference tome but a book to read through and learn the practical aspects of small bowel radiology, I do not consider this a real disadvantage. I liked this book. There is competition in the field, but this is a particularly good account of the everyday problems encountered when examining the small bowel.

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The development of American gastroenterology. By J B Kirsner. (Pp 480; illustrated; $71.) New York: Raven Press, 1990.

Digestive Disease Week is the annual magnet that lures increasing numbers of gastroenterologists from all over the world to the United States; it is now the undisputed 'unofficial world congress.' The reason for this is meticulously displayed in this considerable work of scholarship; the Americans' contribution to gastroenterology is a rich scientific and clinical heritage which lends a lustre to their meetings. Joe Kirsner is, at 81, some 12 years