


Duodenal ulcer and carbohydrate

Sr.,—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 authors 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, 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. 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. 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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3 Tovey FI, Jayaraj AP, Clark CG. Fibre and duodenal ulcer. Lancet 1982; ii: 878.

Gastric acid and urinary acid excretion

Sr.,—Johnson et al (Gut 1990; 31: 826–6) could not find a significant rise in urinary pH two hours after the start of a standard meal in normal volunteers or in patients with duodenal ulcers (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 (4–7–0) and 5.3 (4–5–7). 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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Lipid pattern and plasma insulin in diabetics with gall stones

Sr.,—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. We documented increased concentrations of triglycerides and decreased levels 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 of the authors, and this 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 alterations in women with gall stone disease than in an association of gall stones, low concentrations of high density lipoprotein cholesterol, and coronary disease found only in women.

The serum lipid pattern in our patients might be related to insulin concentrations, as suggested by Laakso et al in another paper. 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 age, sex, body mass index, and duration and metabolic control of diabetes) we also documented increased levels of C peptide in subjects with gall stones compared with controls.

Concerning the possible mechanism by which hyperinsulinemia could enhance gall stone formation, Laakso et al report that high insulin concentrations could activate low density lipoprotein receptors and reduce plasma bile clearance of low density lipoprotein cholesterol. It, however, has also been reported that insulin is able to enhance the activity of b-hydroxy b-methylglutaryl coenzyme A reductase and to suppress b-hydroxybile acid synthesis with consequent increased cholesterol and decreased bile acid secretion in bile. According to this finding Bennion and Grundy 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. 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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Letters, Book reviews

Oesophagography and AIDS

Stir.—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 un satisfactory 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.1,2 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 by this contrast examination. Patients with AIDS often have a more fulminating form of candidiasis in which the oesophagus has an easily recognizable 'shaggy' appearance on oesophagography due to multiple plaques, pseudomembranes, and ulcers. In contrast, herpes oesophagitis is typically seen on double contrast radiographs by discrete, superficial ulcers without evidence of plaques.1 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 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 oesophagograms, 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.

Rebecca T. Yardley


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 straightforward 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.

P A McCormick


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 Lauer 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 compres sion work of scholarship 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.

CIB TRAMT


Digestive Disease Week is the annual meeting that draws 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 thoughtful and readable work; the Americans' contribution to gastroenterology is a rich scientific and clinical heritage which lends a lustre to their meetings. Joe Kirshner is, at 81, some 12 years