

PostScript

LETTERS

Immunosuppression, IBD, and risk of lymphoma

We read with interest the two recent reports of lymphoma in patients with inflammatory bowel disease (IBD) (Farrell *et al*, *Gut* 2000;47:514-19 and Palli *et al*, *Gastroenterology* 2000;119:647-53). We believe the report from Farrell of four cases of lymphoma in a cohort of 782 patients (of whom 238 had received immunosuppression) considerably overestimates the relative risk of lymphoma in IBD patients. They calculate a relative risk of lymphoma as 31 for the whole cohort and 59 for the group treated with immunosuppressives (compared with the general population).

Immunosuppressive therapy is well recognised as increasing the risk of developing non-Hodgkin's lymphoma (NHL) in organ transplant patients.¹ The risk of NHL is increased in other inflammatory conditions, such as rheumatoid arthritis² and psoriasis, although how much is attributable to the underlying disease and how much is due to the drug is unclear.

For IBD, if the incidence of lymphoma is indeed increased, is this due to drug or disease? Two recent reviews^{3,4} have examined this question in detail.

Several large well designed population based studies have been performed specifically to examine the baseline risk of lymphoma in IBD. In none of these studies does the relative risk for NHL significantly exceed one, while only one study (Palli *et al*) has shown an excess risk of Hodgkin's disease (relative risk 9.3; 95% confidence interval 2.5-23.8).

A number of smaller case series have been published which show an increased incidence of NHL. This type of study, although interesting, should not be regarded as evidence of increased risk as case ascertainment bias is likely to exist.

Several studies have specifically addressed the question of immunosuppression in IBD. In total, only 11 cases of lymphoma were described in more than 4000 patients who had received immunotherapy, with over 17 000 patient years of follow up. Extrapolating these data to lymphoma rates in the general population may be unreliable, particularly as lymphoma rates vary widely geographically, by sex and age.⁵

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We believe that compared with the other known risks of immunosuppression, such as myelosuppression and infection, the risk of developing lymphoma (if it does exist) is likely to be of minor clinical significance and to be outweighed by the potential benefit of these treatments in patients with IBD.⁶

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References

- Opelz G, Henderson R. Incidence of non-Hodgkin lymphoma in kidney and heart transplant recipients. *Lancet* 1993;342:1514-16.
- Kinlen LJ. Incidence of cancer in rheumatoid arthritis and other disorders after immunosuppressive treatment. *Am J Med* 1985;78:44-9.
- Aithal GP, Mansfield JC. Review article: the risk of lymphoma associated with inflammatory bowel disease and immunosuppressive treatment. *Aliment Pharmacol Ther* 2001;15:1101-8.
- Bebb JR, Logan RPH. Review article: Does the use of immunosuppressive therapy in IBD increase the subsequent risk of developing lymphoma? *Aliment Pharmacol Ther* 2001;15:1843-9.
- Cancer incidence in five continents*, vol. 5. IARC Scientific Publications No 88. Oxford: Oxford University Press, 1987.
- Lewis JD, Schwartz JS, Lichtenstein GR. Azathioprine for maintenance of remission in Crohn's disease: Benefits outweigh the risk of lymphoma. *Gastroenterology* 2000;118:1018-1024.

Current guidelines fail young patients with oesophagogastric cancer

Wallace *et al* (*Gut* 2001;49:29-34) clearly describe the difficulties with AGA guidelines for endoscopy in the light of their study in identifying young oesophagogastric pathology. They concluded that better clinical prediction strategies are needed. In the UK we have found similar failings in our own guidelines for endoscopy¹ for identification of young patients with oesophagogastric carcinoma. The national data suggesting oesophagogastric carcinoma is rare in patients under 55 years seemed at odds with the large number of patients we had seen in this age group in recent years.

We reviewed our oesophagogastric cancers over three years and found 76 patients under the age of 55 years between January 1997 and December 1999. The hospital records of these patients were examined with the general practice records where possible. A detailed review was undertaken in patients under 50 years old, looking at their presenting symptoms, stage, location, nature, and size of tumour. Timings were noted from the date symptoms first developed, referral date, date of diagnosis, and date of death. Kaplan-Meier methods were used to estimate survival.

Of the 460 patients with oesophagogastric carcinoma, 74 (16%) were found to be less than 55 years of age at diagnosis. Thirty eight patients (8.3%) were less than 50 years of age, and 19 (4.1%) were less than 45 years.

Detailed analysis of patients under 50 years revealed that 29 (76%) of 38 patients had dysphagia at the time of presentation (mean duration four months (range 1-9)). Three patients recalled food occasionally sticking after swallowing for 2, 14, and 20 years, respectively. Reflux or heartburn was recalled prior to presentation by 57%, with a mean duration of 6.8 years (3 weeks to 20 years). Seventeen patients (45%) had been prescribed acid suppression or antacid therapy prior to referral. Seventy one per cent were adenocarcinomas, 16% squamous, and 13% others, and average length was 6.8 cm (range 1-16). Fourteen involved the oesophagogastric junction, 15 the oesophagus, and nine the stomach alone. Staging of 37 patients revealed: one T1 tumour, nine T2, 12 T3, and 15 T4 tumours. Forty per cent had metastasis at presentation and 34% were tertiary referrals.

Twenty three patients had a resection, nine of whom had adjuvant chemotherapy and two had radiotherapy. There were 11 palliative, nine chemotherapy, and two radiotherapy treatments. Nine patients were stented. Only four of 23 resections had no lymph node spread. There was no perioperative mortality but median survival was only one year (fig 1). Currently, only eight patients are alive.

The current UK government guidelines specifically state that "The chance of a dyspeptic patient under the age of 55 having gastric cancer is one in a million". This cannot be true given that one unit saw 38 cases of carcinoma in the oesophagus and stomach under the age of 50 years over a three year period. Twenty three had disease involving the stomach; almost all had some prodromal dyspepsia and a high proportion had reflux disease.

Gastro-oesophageal cancer presents late; reliable indicators for the presence of disease are still to be found, although the association with reflux disease has been confirmed recently.² The young present with advanced disease and, as reported by Bowry and colleagues,³ quickly progress to end stage disease. Our referral bias does not explain this effect. We chose to look at patients under 50 years in detail, particularly as this is the proposed increase to the BSG guidelines. The BSG guidelines use 45 years to restrict access, suggesting endoscopy in those patients older than 45 years with new onset dyspepsia and only in younger patients with alarm

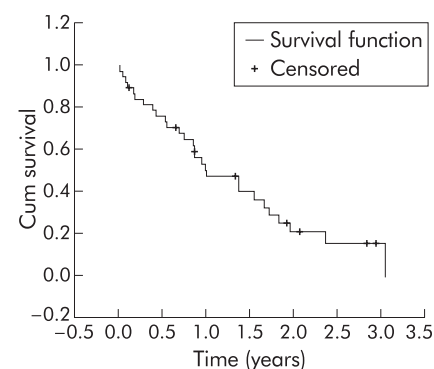


Figure 1 Kaplan-Meier curve of survival of oesophagogastric cancer patients in those less than 50 years of age.

symptoms. Guidelines issued by the UK Department of Health⁴ state that 55 years of age is the cut off for referral, with at least one high risk factor or new onset dyspepsia. Local Cancer Networks can allow 50 or 45 years as a cut off. In our unit, increasing the age to 50 years for patients with new dyspepsia will clearly disadvantage patients when the potential life saving window is made even smaller.

Reflux often precedes the development of oesophageal carcinoma. Is relief of symptoms using medical treatment enough to prevent malignant change or may it even mask a developing tumour? Access to endoscopy needs to be improved in young patients who present with intermittent dyspepsia if early diagnosis is to be made. The development of more sophisticated endoscopic modalities will also make inroads into achieving earlier diagnosis. Without prevention or early diagnosis in this group there is unlikely to be any progress made to extending the lives of these patients.

Our data do not prove that early endoscopy would actually save these patients from a certain death. What it clearly demonstrates is that using the current guidelines, our small region allows 30 of 38 patients under 50 years in a three year period to die of oesophageal or gastric carcinoma. Nationally, this translates into a major failure to adequately diagnose and treat oesophagogastric cancer in young people. The guidelines of diagnosis fail. A screening method is urgently needed for this rapidly increasing cancer problem. Standard endoscopy is too cumbersome and expensive; new technologies may change our whole approach.

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References

- 1 **British Society of Gastroenterology.** *Dyspepsia Management Guidelines*. London: BSG, 1996.
- 2 **Lagergren, J, Bergstrom R, Lindgren A, et al.** Symptomatic gastroesophageal reflux as a risk factor for esophageal adenocarcinoma. *N Engl J Med* 1999;**340**:825–31.
- 3 **Bowry DJ, Clark GWB, Rees BI, et al.** Outcome of oesophagogastric carcinoma in young patients. *Postgrad Med J* 1999;**75**:22–6.
- 4 **Department of Health.** *Referral Guidelines for Suspected Cancer*. London: Department of Health, April 2000 (<http://www.doh.gov.uk/pub/docs/doh/guidelines.pdf> taken Nov 2000).

Acute appendicitis in Japanese soldiers in Burma: support for the "fibre" theory

In the last three months of 1946 and the first quarter of 1947, the late Anand Pardhy and I were the graded surgeon and graded physician in a CCS (Casualty Clearing Station) which was acting as a static hospital in the small town of Prome, on the Irrawaddy, in South Burma. In the same area was a camp of 1077 Japanese soldiers awaiting repatriation. They had their own medical officers and sick bay but cases requiring surgery and the more severely ill medical cases were admitted to the CCS.

During this period, we admitted from the Japanese camp 10 cases of acute appendicitis, or one case every 2–3 weeks. At the beginning I assisted Pardhy but became so familiar with the technique that I was allowed to do the operation, with Anand Pardhy assisting.

We were intrigued by the high incidence of appendicitis in the Japanese soldiers and thought it might be because the camp was receiving mainly British rations which had a lower fibre content than the normal diet of a Japanese soldier. The fact that their own medical officers were surprised at the number of cases suggested that appendicitis was normally rare in Japanese troops.

Fortunately, in the Prome area there were large concentrations of Indian, Gurkha, and Burmese troops, and a battalion of irregulars from the Chin Hills, on the border between Burma and what is now Bangladesh. The total number of these troops greatly exceeded the number of Japanese soldiers in the camp, yet we admitted no cases of appendicitis from these various nationalities.

In discussing the aetiology of appendicitis, Burkitt and Trowell point out that communities with a high fibre diet have a low incidence of appendicitis, while those consuming a Western style diet, low in fibre and high in refined carbohydrates, have a higher incidence.¹ Confirmation of their theory is provided by the difference in the incidence of appendicitis between British and Indian troops in India during the period 1936–1947. Appendicitis was 4–6 times more common in the British than in the Indian troops.² In the same period, the basic ration for Indian troops contained one third the amount of animal protein and three times as much high fibre foods (parboiled rice, atta (unrefined wheat flour), and pulses (dal and peas)) as that of British soldiers in India.³

The effect of a change in intake of fibre was also discussed by Burkitt and Trowell who cited a report that the incidence of appendicitis in Japanese immigrants in Hawaii, where they presumably ate an American style low fibre diet, was higher than in Japan.¹ The same authors referred to reports that Sudanese troops in North Africa and West African troops in Singapore had an increased incidence of appendicitis when they were given British rations.¹ These reports are analogous to our experience with the Japanese prisoners of war but do not discuss whether the effect of a sudden change in diet produced an incidence higher than that in countries consuming a Western type of diet. A rate of 10 cases in six months would produce 20 cases a year in a population of 1000 men, or 200 cases per 10 000 population. The incidence of appendicitis among adults in England and Wales for the years 1931–1935 was estimated at 45 per 10 000⁴ while the annual discharge rate for appendicitis for 1959 was 27 per 10 000,⁵ suggesting a rate of approximately 35 per 10 000 for the 1940s, or about one sixth of the incidence in the Japanese camp. Our values certainly suggest that a sudden change in diet, in this case a reduction in the intake of fibre, produces an exceptionally high incidence of appendicitis.

Van Ouwerkerk⁶ gave an example of the opposite effect. In the Dutch internment camps in Indonesia during the 1939–45 war, appendicitis was practically unknown; the diet consisted of "rice in insufficient quantities, unprocessed vegetables, and practically without meat and fat".

In conclusion, our experience with the Japanese camp tends to confirm Burkitt and Trowell's theory that a low fibre diet causes a

high incidence of appendicitis. However, recent evidence from South Africa⁷ has shown that urban Black Africans continue to have a very low incidence of appendicitis in spite of the fact that their dietary intake of fibre is lower than that of the urban white population. Clearly then, there may be factors other than the level of fibre intake in determining the incidence of acute appendicitis.

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References

- 1 **Burkitt DP, Trowell HC.** *Refined carbohydrate foods and disease*. London: Academic Press, 1975.
- 2 **Medical Directorate, India.** *Reports on the health of the Army in India 1936–1947*. New Delhi: Medical Directorate.
- 3 **Medical Directorate, India.** *Field service hygiene notes, India*. New Delhi: Medical Directorate, 1945;111–114.
- 4 **Young M, Russell WT.** *Appendicitis: a statistical study*. MRC Special Reports Series No. 233. London: HMSO, 1933.
- 5 *Report on the Hospital In-Patient Enquiry*. London: HMSO, 1959.
- 6 **Van Ouwerkerk LW.** Diet and appendicitis. *Arch Chir Neerl* 1951;**3**:164–78.
- 7 **Paterson-Brown S.** In: Burnand KG, Young AE, eds. *The New Aid's Companion in Surgical Studies*, 2nd edn. London: Churchill Livingstone, 1998; 722.

GRP and stimulation of acid secretion

We read with interest the article by Hildebrand *et al* on the effect of gastrin releasing peptide (GRP) on acid secretion in healthy individuals (*Gut* 2001;**49**:23–8). This study shows that a GRP antagonist (BIM 26226) inhibits acid secretion with no effect on plasma gastrin. The authors concluded that GRP stimulated acid secretion by a non-gastrin mechanism. Their results are in agreement with previous studies from our laboratory which indicate that GRP in the rat stimulates acid secretion by a mechanism other than by gastrin release.¹ We also found that GRP releases somatostatin and inhibits histamine release from the oxyntic mucosa, an effect which viewed in isolation should counteract acid secretory stimulation.¹

These studies and others demonstrate the complexity of neuropeptide mechanisms in the regulation of gastric acid secretion. In our opinion, great care should be shown when interpreting the results from experiments using neuropeptides in the study of gastric physiology and pharmacology.

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References

- 1 **Sandvik AK, Holst JJ, Waldum HL.** The effect of gastrin-releasing peptide on acid secretion and the release of gastrin, somatostatin, and histamine in the totally isolated, vascularly perfused rat stomach. *Scand J Gastroenterol* 1989;**24**:9–15.

Authors' reply

Our conclusion that gastrin releasing peptide (GRP) is not a physiological regulator of gastrin secretion in humans stands against an

impressive body of evidence collected in the past 30 years in different animal species. Even in humans, several lines of evidence would support a role for GRP as a regulator of the G cell: infusion of exogenous GRP stimulates gastrin release in humans but GRP also releases gastrin from isolated human g cells in vitro.¹⁻³ In several laboratory animals, GRP antagonists or GRP antibodies inhibit the release of gastrin to a variety of stimulants.^{4,5} Our results obtained in healthy male subjects could therefore be due to species differences with respect to the physiological role of GRP as a gastrin secretagogue. As pointed out by Waldum and Sandvik, some studies in rats have also shown that GRP can stimulate acid secretion independent of gastrin release. Along the same lines, gastrin concentrations are normal in mice lacking the GRP receptor.⁶ As gastrin secretion is regulated by various factors, including nutrients, G cell responses to GRP reflect the balance of direct stimulatory effects and indirect inhibitory factors. We hope that our data will generate new interest in studying the role of this interesting peptide in regulating gastrointestinal functions.

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References

- 1 Hildebrand P, Werth B, Beglinger C, *et al*. Human gastrin-releasing peptide: biological potency in humans. *Regul Pept* 1991;**36**:423-33.
- 2 Squires PE, Meloche RM, Buchan AM. Bombesin-evoked gastrin release and calcium signaling in human antral G cells in culture. *Am J Physiol* 1999;**276**:G227-37.
- 3 Moore ED, Ring M, Scriven DR, *et al*. The role of protein kinase C isozymes in bombesin-stimulated gastrin release from human antral gastrin cells. *J Biol Chem* 1999;**274**:22493-501.
- 4 Dockray GJ. Topical review. Gastrin and gastric epithelial physiology. *J Physiol* 1999;**518**:315-24.
- 5 Dockray GJ, Varro A, Dimaline R, *et al*. The gastrins: their production and biological activities. *Annu Rev Physiol* 2001;**63**:119-39.
- 6 Wada E, Watase K, Yamada K, *et al*. Generation and characterization of mice lacking gastrin-releasing peptide receptor. *Biochem Biophys Res Commun* 1997;**239**:28-33.

BOOK REVIEWS



Textbook of Gastrointestinal Radiology, 2nd edn, vols 1 and 2

Edited by R M Gore, M S Levine. UK: Harcourt Publishers Ltd, 2000, £265, illustrated, pp 2261. ISBN 072167836

With Margulis and Burhenne's *Alimentary Tract Radiology* currently out of print, the *Textbook of Gastrointestinal Radiology* is the only large all encompassing reference text for

gastrointestinal radiologists and interested gastroenterologists. This is the second edition and its predecessor was never far from my hand, especially when a particularly difficult or obscure differential diagnosis was required. While reviewing a book can sometimes be a necessary chore, this is a delight, not least because it is an essential purchase for any aspiring gastrointestinal radiologist.

This large two volume set attempts to cover the whole gamut of gastrointestinal radiology, both luminal and solid organ, in 131 chapters. The editors state their aim "to provide complete, up to date coverage of the state of knowledge in gastrointestinal radiology in a practical and useable way." While large books such as this can sometimes be criticised as cumbersome, the editor's ambitions demand a format capable of presenting the necessary breadth and depth required of a reference text, and I would argue that the book is indeed practical and useable; I certainly found the predecessor so. The plethora of chapters is sensibly divided into those dealing with hollow and solid viscera, each preceded by sections dealing with general techniques and principles of interpretation. There are also sections on paediatric disease and common clinical problems. The respective organ sections are generally exhaustive; for example, separate chapters are entirely devoted to postoperative appearances.

I have only one criticism, albeit major. The editors claim to have assembled "an outstanding group of internationally recognised authors". While this is probably true, they are certainly not international! Although not overtly stated, this is essentially a North American text; a paltry three of the 124 authors hail from outside this continent. I cannot help feeling this prevents the text from being truly definitive. Gastrointestinal radiology is a well defined subspecialty and the movers and shakers are by no means exclusively North American. I suspect the editor's parochial approach is partly responsible for some glaring errors of omission. For example, anal endosonography, a clinically important and well disseminated technique, is rarely practised by North American radiologists and not even mentioned in passing. Indeed, the relative dearth of ultrasound in general probably reflects transatlantic practice. I hope other omissions, for example colonic stenting and the use of magnetic resonance imaging to assess and classify perineal fistulae, merely reflect the time taken to get a large book like this into the shops rather than spectrum bias. The absence of virtual colonoscopy is almost certainly due to publication lag; it is mentioned in the preface but absent in the book!

However, there is little to criticise when straying into more conventional territory and here the North American authorship brings undoubted benefits for a reference text; it would be difficult to find more extensive references to computed tomography imaging of gastrointestinal pathology elsewhere. From personal experience, most chapters dealing with mainstream topics can be used as well referenced starting points for indepth analysis of the particular field described. So, with the caveat mentioned, the second edition of this book remains as invaluable as the first and is certainly a must-buy for any gastrointestinal radiologist who needs a reference text to hand in his or her office, or for more leisurely inspection at home.

S Halligan

Core Curriculum in Adult Primary Care Series: Gastroenterology

The Core Curriculum Committee HealthStream, Nashville, TN, USA. ISBN 1-57276 943-2

This CD, which was extremely difficult to unwrap, is aimed at primary care physicians in the USA, and is part of a fairly comprehensive set of learning materials produced by an organisation called Core Curriculum. The CDs are all based on lectures given by clinicians from in and around Boston and each consists of an audio recording of a four hour session of four lectures, backed up by a transcript of the lectures and by graphics. The gastroenterology CD covers irritable bowel syndrome (IBS), coeliac disease and lactose intolerance, and advances in upper and lower gastrointestinal endoscopy and in the treatment of chronic viral hepatitis. The graphics change at the rate of about one per minute and the entire CD is readily navigable and also searchable. The programme concludes with a CME quiz for accreditation purposes.

I found the material quite engaging, although I could not resist skipping about between and within the lectures. An hour is a long time, even in gastroenterology, so that the quality of the lecturer's voice gradually assumes greater importance than the quality of the material being presented. Unfortunately, the moderator of this programme speaks in a hypnotic register, and the IBS lecturer would not keep most of us on the edge of our seats. In contrast, the coeliac disease expert (Irish) had a most engaging audio persona. The sound quality of two of these lectures, particularly the enthusiastic endoscopist, was not, I thought, of a high enough quality for publication, and seriously interfered with my concentration.

The content of all four lectures was however excellent and represents a useful refresher course, although not for primary care physicians as we know them because much of this material goes beyond the needs and experiences of general practitioners in Europe. I imagine that trainees in gastroenterology would find this a useful source of reference and revision and, although this material is not quite as cutting edge as the publishers claim, it may well be of interest to more senior gastroenterologists wishing to be updated in areas outside their own specialism.

I have no idea whether the combination of a talk, without the talking head, with good quality graphics, and a readily accessible transcript is likely to lead to more concentrated or deeper learning than its component parts. I can almost feel a randomised controlled trial coming on. I tried to find out more about the lecture series by visiting HealthStream's website at www.corecurriculum.com, and eventually found my way to a programme about gastroenterology for family physicians, consisting of a one hour presentation on gastro-oesophageal reflux disease funded by AstraZeneca. The website was difficult to use and I got stuck in the middle of a course on craniopharyngeal embryology.

R Jones

Recent Advances in Gastrointestinal Endoscopy

Edited by M S Bhutani, R K Tandon. Jaypee Brothers, pp 678. ISBN 81 7179-810-1

This is an extremely comprehensive and well illustrated reference book for experienced endoscopists wishing to extend their area of

practice to include the very latest diagnostic and therapeutic procedures. It comprises seven sections covering areas that the average UK gastroenterologist would not normally enter. As a reference book for physicians, surgeons, and radiologists, I have not come across a more complete presentation of up to date techniques. The seven sections comprise upper and lower gastrointestinal endoscopy as well as small bowel, pancreatobiliary, laparoscopy, endoscopic ultrasound, and developing imaging modalities. The editors have enlisted contributions from many well known names from around the world and the target audience is clearly global.

Who should buy this book? The answer must be any large multidisciplinary endoscopy unit already offering a full and comprehensive service to patients but with aspirations of providing leading edge endoscopic facilities and practice. All of the significant recent advances are covered in the seven sections and it is a timely reference book for clinicians wanting to be at the forefront of gastrointestinal endoscopy. It is also a book which will educate the experienced endoscopist about the current frontiers and future direction of endoscopy, as viewed by the editors. Clearly, some sections will not be relevant to UK endoscopists which is why a department should purchase this book rather than an individual clinician.

The book has many strengths, including the written detail of procedures that clinicians aspiring to be advanced endoscopists need to know in order to safely perform the latest techniques. It is not a substitute for attending workshops and courses but it does give the reader more insight into the nuances of a new procedure, which often cannot be obtained from reading scientific journals.

Although the list of contributors is formidable, I found it slightly surprising that some areas of expertise were not covered by those most qualified to write about it. For instance, the chapter covering endoscopic mucosal resection was not written by any of the Japanese endoscopists who have pioneered this procedure but by three authors from Germany. The same is true of other areas, but this must not detract from the fact that, as a reference book with multiple authors, the editors have done a magnificent job of making it easy to read and relevant to the practise of present day endoscopists. Such a book fulfils a valuable role and I am sure that I will be referring to it many times during the next few years. It will of course need to be kept up to date and I look forward to the next edition in five years time.

M Bramble

Hepatobiliary Diseases: Pathophysiology and Imaging

Edited by K Okuda, D G Mitchell, Y Itai, *et al.* Oxford: Blackwell Science, 2001, £150, pp 764. ISBN 0-632-05542-1

Professor Okuda sets out the aim of this book which is to describe advances in various imaging modalities to facilitate the use of different techniques and contribute to the understanding of hepatobiliary diseases and pathophysiology. The book is edited by three Japanese professors of medicine and radiology, and one American professor of radiology who is a magnetic resonance imaging (MRI) specialist. The contributing authors are Asian and American with the majority from Japan. The authors include physicians, gastroenterologists, pathologists, endoscopists, and radiologists.

This book celebrates the progress in imaging over the last two decades or so such that gross pathology is now fairly well shown by accurate and non-invasive or minimally invasive imaging methods. Microscopic histological changes are incompletely shown by imaging techniques but there has also been significant progress in characterising various processes, diseases, and tumours by imaging without biopsy.

The first section entitled "progress in imaging" considers computed tomography (CT), MRI, and developments in ultrasound. Modern CT and MR techniques are explained with suitable illustrations. Most impressive are the three dimensional images constructed from CT and MR data.

The chapter on ultrasound introduces all of the new ideas of tissue harmonic imaging (printing error on page 33, tissue harmonica imaging!), tissue characterisation, and ultrasound contrast agents. Unfortunately, some of the images in this short chapter are disappointing and the concepts described are not completely clear.

The next section concerns anatomy and gross changes in the liver with illustrations of pathology and imaging examples. These chapters are descriptive down to the level of electron microscopy with a lot of useful diagrams and imaging illustrations. There is a further short chapter describing the relationship and pathology of the diaphragm with the liver.

Longer sections then deal with diffuse hepatic diseases with chapters on acute and chronic viral hepatitis and other causes of chronic hepatitis. There is a chapter on pathology, physical signs, and imaging in cirrhosis and further chapters on fatty liver, alcoholic liver disease, iron overload, and other diffuse changes, including metabolic disease and drug induced liver damage.

The next section deals with vascular disease including portal hypertension, non-cirrhotic portal hypertension, portal vein thrombosis, and Budd-Chiari syndrome. Included in this section is a chapter on haemodynamics. The sometimes complex relationship between the various hepatic circulatory systems are illustrated by CT, arterial portography, and CT hepatic angiography, and also by balloon occlusion of hepatic arteries and veins. Many observational studies of this type have been published in the radiological literature, usually from Japanese centres. This type of work is useful for the understanding of odd lesions or pseudo lesions demonstrated in more conventional CT or MR examinations but they are by no means routine procedures in UK practice.

The chapter on portal hypertension and non-cirrhotic portal hypertension has many fascinating illustrations with a large number of "invasive" studies, including transhepatic portal vein catheterisation and transumbilical portal vein catheterisation. As diagnostic procedures, these would be regarded as somewhat invasive in current UK and European practice when much of this information is obtainable by CT, ultrasound, or MR. We see similar images during procedures such as TIPS but not usually for diagnosis alone.

Naturally, there is considerable discussion of hepatocellular carcinoma, a major problem in Japan and possibly an increasing problem in the western world.

All types of focal liver lesions are described with considerable emphasis on the haemodynamics contributing to contrast enhancement at CT, MRI, and ultrasound. Pathology and imaging of potential premalignant cirrhotic nodules are also reviewed. Later chapters

cover other liver disorders including trauma, parasites, and liver transplants followed by extensive discussion of biliary diseases.

This is not an ordinary textbook but a collection of explanations of the pathophysiology illustrated by imaging. This leads to emphasis on CT, hepatic angiography, and arterial portography. In the UK and USA, current emphasis is more on the use of multislice multiphase CT with intravenous contrast and MRI with various, more specific, contrast agents.

Some of the images included are remarkably over the top. For example, a cystic arteriogram is shown in a case of gall bladder carcinoma. Overall, I enjoyed the book, particularly for the imaging minutiae. However, mixed up with this is an attempt to cover all types of liver and biliary disease as well as imaging and pathology. The treatment and management of many conditions are also discussed in a limited way. This gives an overall broad but uneven coverage. Although entirely satisfactory in themselves, I am not certain that it was necessary to include chapters on trauma and liver transplantation with the other material.

This is not a book for every gastroenterologist but will be of more interest to those specialising in liver diseases, particularly if they wish to delve into the complexities of imaging.

S P Olliv

NOTICES

Sir Francis Avery Jones BSG Research Award 2003

Applications are invited by the Education Committee of the British Society of Gastroenterology who will recommend to Council the recipient of the 2003 Award. Applications (TWENTY COPIES) should include:

- A manuscript (2 A4 pages ONLY) describing the work conducted
- A bibliography of relevant personal publications
- An outline of the proposed content of the lecture, including title
- A written statement confirming that all or a substantial part of the work has been personally conducted in the UK or Eire.

Entrants must be 40 years or less on 31 December 2002 but need not be a member of the Society. The recipient will be required to deliver a 30 minute lecture at the Annual meeting of the Society in Birmingham in March 2003. Applications (TWENTY COPIES) should be made to the Honorary Secretary, British Society of Gastroenterology, 3 St Andrews Place, London NW1 4LB by 1 December 2002.

Hopkins Endoscopy Prize 2003

Applications are invited by the Endoscopy Committee of the British Society of Gastroenterology who will recommend to Council the recipient of the 2003 Award. Applications (TEN COPIES) should include:

- A manuscript (2 A4 pages ONLY) describing the work conducted
- A bibliography of relevant personal publications
- An outline of the proposed content of the lecture, including title

- A written statement confirming that all or a substantial part of the work has been personally conducted in the UK or Eire.

An applicant need not be a member of the Society. The recipient will be required to deliver a 30 minute lecture at the Annual meeting of the Society in Birmingham in March 2003. Applications (TWENTY COPIES) should be made to the Honorary Secretary, British Society of Gastroenterology, 3 St Andrews Place, London NW1 4LB by 1 December 2002.

Broad Medical Research Program—Inflammatory Bowel Disease Grants

Funds for inflammatory bowel disease (IBD) research are available immediately from the Broad Medical Research Program of The Eli and Edythe L Broad Foundation for innovative projects regarding etiology, therapy, or prevention. Grants totalling approximately US\$100,000 per year are available for basic or clinical projects. Larger requests may be considered. Initial letter of interest (no submission deadline), simple application, rapid (60 day) peer review, and funding. Criteria for funding includes new ideas or directions, scientific excellence, and originality. Early exploratory projects, scientists not currently working in IBD, and/or interdisciplinary efforts are encouraged. Further information: Marciana Poland, Research Administrator, Broad Medical Research Program, 10900 Wilshire Blvd., 12th Floor, Los Angeles, CA 90024-6532, USA. Tel: +1 310 954 5091; email: info@broadmedical.org; website: www.broadmedical.org

ESPEN 2002

The European Society for Parenteral and Enteral Nutrition will be hosting its annual meeting on 31 August to 4 September 2002 in Glasgow, UK. The organisers anticipate 300 delegates, principally from Europe but also from the USA and the Far East. Further information: Mrs Pat Howard, Honorary Secretary, BAPEN, Head of Nutrition and Dietetic Services, Bristol Royal Infirmary, Bristol BS2 8HW. Tel: +44 (0)117 928 2049; fax: +44 (0)117 928 3005; email: pat.howard@ubht.swest.nh

Recent Developments in Gastric MALT Lymphoma

This is a one day meeting organised jointly by the European Gastrointestinal Lymphoma Study Group and British Society of Gastroenterology, Gastro-duodenal Section. The meeting will be held on 20 December 2002 in London, UK. Further information and registration details: Dr A Dogan, Department of Histopathology, Royal Free and University College, Medical School, Rockefeller Building, University Street, London WC1E 6JJ, UK. Tel: +44 (0)20 7679 6015; fax: +44 (0)20 7387 3764; email: a.dogan@ucl.ac.uk; website: www.ucl.ac.uk/histopathology/egils

Postgraduate Gastroenterology

This course will be held on 15–18 September 2002 in Oxford, UK. The course has been designed for consultants and registrars, including those who do not specialise in gastroenterology. Topics will include: Barrett's Oesophagus; The Case for Endoscopic Surveillance Debate; Liver Disease; Bacteria and the Gut; IBD Therapeutics, Gastrointestinal Bleeding, Endoscopic Training. Further information: Professor Derek P Jewell, University of Oxford, Nuffield Department of Medicine, Gastroenterology Unit, Gibson Laboratories, 2nd Floor, Radcliffe Infirmary, Block 21, Woodstock Road, Oxford OX2 6HE. Tel: +44 (0)1865 224829; fax: +44 (0)1865 790792; email: derek.jewell@ndm.ox.ac.uk; website: www.medicine.ox.ac.uk/gastro

British Association for the Study of the Liver

The 2002 BASL meeting will be held on 11–12 September in Newcastle, UK. Further information: Mrs Jackie Carter, Centre for Liver Research, University of Newcastle, Floor 4, William Leech Building, Medical School, Framlington Place, Newcastle upon Tyne, NE2 4HH, UK. Tel: +44 (0)191 222 5640; fax: +44 (0)191 222 0723; email: j.a.carter@ncl.ac.uk

3rd World Chinese Congress of Digestology

This congress will take place on 23–25 September 2002 in Beijing, China. Further

information: Lian-Sheng Ma, President of WCCD, PO Box 2345 Beijing 100230, China. Fax: +86 6589 1893; email: wcjd@public.bta.net.cn

EPGS Second Update on Coloproctology

The European Postgraduate Gastro-Surgical School presents this course on 10–11 October 2002 in Amsterdam, the Netherlands. Further information: visit the website www.epgs.nl, or email epgs@amc.uva.nl. Tel: +31 20566 3926/4386.

Xth European Course on Therapeutic Digestive Endoscopy and Radiology

This course will take place on 24–25 October 2002 in Rome, Italy. Further information: SC Studio Congressi, Via Francesco Ferrara 40, 00191 Roma, Italy. Tel: +39 06 3290150; fax: +39 06 36306897; email: sc.congressi@stm.it; website: www.scstudiocongressi.it

Advances in the Inflammatory Bowel Diseases

This conference will take place on 6–7 December 2002 in New York, USA. Further information: Heather Drew, Imedex, 70 Technology Drive, Alpharetta, GA 30005-3969, USA. Tel: +1 770 751 7332; fax: +1 770 751 7334; email: h.drew@imedex.com; website: www.imedex.com

15th European Intensive Course (SMIER) Digestive Endoscopy

This course will take place on 16–17 December 2002 in Strasbourg, France. Further information: Michele Centonze Conseil, 6 bis Rue des Cendriers, 75020 Paris, France. Tel: +33 1 44 62 68 80; fax: +33 1 43 49 68 58.