target biopsy specimens can be obtained from an abnormal area.

It is true that a small proportion of patients develop carcinoma in colitis without dysplasia elsewhere in the colon and that dysplasia, when present, is usually patchy. The paper by Rasonhoff et al., however, is quoted as evidence that dysplasia at a distance from the cancerous lesion occurs in only 50% of colicky specimens. This paper was based on histological blocks from only 22 specimens with cancer in colitis, complete resections were performed in only 11 of the patients, and more than five blocks were available from only 13 of the specimens. Some dysplasia at a distance from the tumor was found in 73% of specimens and high grade dysplasia in 50%. The authors suggest that one reason why the incidence of dysplasia was less than in other reports may have been due to limited sampling. Careful examination of 62 colicky specimens with cancer in colitis at St Mark's Hospital has shown dysplasia at a distance from the tumour in 87%.

Dr Mide selected three clinical studies for comment and we appreciate her remarks about the importance of high levels of follow up and of clinical care in regard to our surveillance programme. It is a pity that she made no reference to two excellent Swedish series, 'one reasonable' in which there has been cancer death and in which the three tumours treated surgically were at an early stage (Duke's A).

Like Dr Gyde, we would welcome the development of a new marker of neoplastic potential in colitis. Apart possibly from analysis of aneuploidy, which is complex and expensive, no such marker has been identified. Despite its limitations, dysplasia is still the only thoroughly tested marker we have and it should not be rejected.

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Reply

Str, — Professor Blum et al report the superiority of sucralafate over placebo in the prevention of gastric ulcer recurrence. They speculate that continued treatment with sucralafate may have theoretical advantages over long-term maintenance therapy with antisecretory drugs, but fail to discuss the potential risk of aluminium accumulation. Administration of sucralafate (1 g twice a day) will result in a daily intake of 414 mg aluminium. The aluminium moiety of sucralafate can dissociate at a low pH, and short-term administration of 4 g daily might lead to appreciable increases in serum and urine aluminium concentrations.1,2 Long-term administration of sucralafate (1 g twice a day) did not result in statistically significant increases in plasma aluminium in either of two small maintenance studies3,4 but animal experiments indicate that bony accumulation of aluminium may occur in the absence of raised serum concentrations.5 Sucralfate may prove to be effective and safe for the prevention of gastric ulcer recurrence. However, further studies of aluminium accumulation from long-term use are likely to be needed before this drug can be widely recommended as an alternative to antisecretory agents.

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Reply

Str, — MC Allinson mentions in his letter experiments in rats which showed an increase of bone aluminium concentrations. This finding, however, could not be confirmed in patients receiving 4 g sucralafate daily for eight to ten weeks before total hip replacement. There was no increase of bone aluminium concentration compared with a control group.1 Aluminium toxicity is mainly discussed in connection with urological patients and the application of high dose aluminium hydroxide as a phosphate binding agent. There is no evidence that long-term maintenance treatment with sucralafate in the recommended dosage in patients with normal kidney function will lead to unwanted side effects.

It should finally be mentioned that sucralafate is approved for the maintenance treatment of duodenal ulcer in several countries; recently it was approved for this indication also by the Federal Drug Administration in the USA.

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BOOK REVIEWS


This monograph written by Starzl and Demetris is an up to date summary of liver
transplantation seen through the enormous experience of the Pittsburgh group. It is refreshing to read a text by two authors, rather than the more common multiple authorship usually found with clinical volumes.

Dr Starzl has always written clearly and well with forthright views. The surgical procedure and pathological findings are the main concern of this book, although there is a chapter on the more hepatological aspects and selection of patients.

Dr Starzl together with Dr Moore, independently, worked out methods of experimental liver transplantation in animals and this led directly to the clinical application of liver transplantation by Dr Starzl when he was in Denver in 1963. After his move to Pittsburgh the pace of work on liver transplantation increased and the introduction of cyclosporin permitted better results, fewer steroids, and a surge of interest throughout the world. Many surgeons are now interested in this field and this book will be a useful start for them. There are nearly 700 references listed. Although the contributions in liver transplantation from Dr Starzl’s group have been exceedingly important, transplantation has developed in other centres and other countries, and in Europe there have been many developments. These do not figure prominently in this monograph which is well produced and easy to read.

R Y CALNE


Diarrhoeal diseases are a major problem in all countries. An estimated 5 to 10 million diarrhoeal-associated deaths occur annually in humans in Asia, Africa, and Latin America with the highest mortality in children. Up to 1988, new pigs and calves succumb to acute diarrhoea. Increasing use of day care centres and intensive farm management practices has increased the risk of infantile gastroenteritis in humans and animals in developed countries. The majority of cases of infectious diarrhoea are due to viruses, but knowledge has been hampered by the lack of suitable cell culture systems or other diagnostic tests.

This is an excellent book. Each chapter is carefully prepared and presents a comprehensive review (up to 1988) of all aspects of the virology, pathogenesis, and immunology of the known diarrhoeal viruses of man and animals. So much can be learnt about the human diseases by study of the animal pathogens and vice versa that it is essential to cover all aspects, as in this volume. There are, in addition, two useful chapters on intestinal immunity and enteric virus vaccines.

The book is well presented with clear and useful illustrations and a good index. The comprehensive nature of the chapters is illustrated by the section on group A rotaviruses, of which the human representative was first discovered in 1973; a total of 446 references are listed on this topic.

Saif and Theil’s book should provide a standard reference work for gastroenterologists, virologists, immunologists, and veterinarians.

D J JEFFRIES

Helicobacter pylori, gastritis and peptic ulcer. Edited by P Malfertheiner and H Ditschuneit. (Pp 478; 141 figures; 94 tables; DM 168.) Berlin: Springer-Verlag, 1990.

An interesting and useful book, which represents the (partial) proceedings of a conference (abstracts in Klin Woch 1989; 67, suppl 18). The full articles do provide some technical details and references to techniques not available in the abstracts.

The book is divided into sections on the taxonomy and biology of Helicobacter pylori; pathogenic mechanisms; immune reactions to H pylori; gastritis; the involvement of H pylori in duodenal ulcer and non-ulcer dyspepsia; and the treatment of infection with H pylori. Most of the sections end with commentaries, which vary in both quality and objectives, since some review the preceding papers, some the whole topic, and some are genuinely critical and provide pointers to the future.

Some of the papers make more impact than others. For example, a good systematic analysis of the microbiological aspects of H pylori infection is spoiled by discussion of how it manages to switch off parietal cells – a finding not compatible with its supposed involvement in the pathogenesis of duodenal ulcers. An interesting article by Labigne and colleagues discusses the relation of the urease of H pylori and plants. Similarly, an analysis of the possible clinical uses of its serology in reducing the need for endoscopy is worth mention. The section on gastritis reinforces the view that the topic requires study and analysis without preconceived ideas. The decision of the German Society of Pathology to abandon the mention of mucosal atrophy when considering ‘gastritis’ is surely correct. It is also refreshing to read, in the sensible review of Price and Stolle, that ‘histopathologists are not swept along and diagnose HP-associated gastritis at the merest sight of the organism.’ They also point out that other bacteria can be found on the gastric surface — the relevance of which to gastric mucosal disease is often ignored.

The section on the connection between H pylori and duodenal ulceration is comprehen- sive and shows how H pylori is thought to produce gastric hypersecretion, metaplasia of the duodenal mucosa, and abnormalities of upper alimentary motility. Although there are several interesting hypotheses, most are biologically not coherent. For example, H pylori neutralises antral gastric juice so as to promote hypergastrinaemia, which in turn results in gastric hypersecretion, resulting in gastric metaplasia of the duodenal mucosa. Odd, how the gastric juice passes through the antrum in sufficient amounts to produce duodenal metaplasia but not in amounts sufficient to acidify the antral mucosa. And so on. Unfortunately, there is not much critical examination of the many hypothetical factors and conditions men-

tioned in the hypotheses; nor even of the facts, since infection with H pylori is considered to produce (epidemic) hypochlorhydria in normal individuals, increased peak acid output in patients with duodenal ulcer, and decreased peak acid output in patients with the Zollinger-Ellison syndrome.

Under the circumstances, it is perhaps a little hard on agnostics to be told that failure to appreciate the significance of H pylori infection is similar to the rejection of Semmelweis and Lister in the last century.

K G WORMSLEY

Notes

Leeds Course in Clinical Nutrition

The Leeds Course in Clinical Nutrition will be held on 10–13 September 1991. Further details are available from Mrs Hilary L Helme, Department of Continuing Professional Education, Continuing Education Building, Springfield Mount, Leeds LS2 9NG. Tel: (0532) 333233.

5th International Symposium on Colorectal Cancer

The 5th International Symposium on Colorectal Cancer will be held on 24–26 September 1991 at Torino, Italy. Further information is available from the Organising Secretariat, Francochboro Health Congress, c/o Jet Viaggi, Corso Matteotti 1, 10121 Torino, Italy. Tel: 039 11599529; fax: 039 11548222.

International Conference of Gastroenterology

An International Conference of Gastroenterology will take place on 31 October–3 November 1991 (Hong Kong), 3 November–6 November 1991 (Beijing). Information and application forms are available from the Conference Secretariat, 12/F Kaiyeng Commercial Centre, 4–6 Hankow Road, Kowloon, Hong Kong. Tel: (852) 3679372; fax: (852) 7218823.

Live endoscopy demonstration in Hong Kong

The Chinese University of Hong Kong and the Hong Kong Society of Digestive Endoscopy will hold the Sixth International Workshop on Therapeutic Endoscopy, 3–5 December 1991. Further details can be obtained from Dr Joseph Leung, Department of Medicine, Prince of Wales Hospital, Shatin, NT, Hong Kong. Tel: (852) 63631285; fax: (852) 6350075.