8 Dwivedi M, Misra SP, Misra V, Kumar R. Value of adenosine deaminase estimation in the diagnosis of tuberculous ascites. *Am J Gastroenterol* 1990; 85: 1123–5.

Reply

SIR,—We acknowledge the pertinent points raised by Lingenfelser and Marks. When our paper was submitted we thought that the role of ascitic adenosine deaminase in the diagnosis of tuberculous peritonitis[1 2] needed further investigation. Subsequent evaluations of ascitic adenosine deaminase in tuberculous peritonitis[3 4] have shown high diagnostic sensitivities.

We therefore accept that this determination is an important investigatory option in the diagnosis of peritoneal tuberculosis. The role of peritoneoscopy therefore seems to be limited to equivocal adenosine deaminase results where there is high clinical suspicion of tuberculous peritonitis or cases where malignant ascites is suspected.

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1 Martinez-Vazquez JM, Ocana I, Ribera E, Sequra RM, Pascual C. Adenosine deaminase activity in the diagnosis of tuberculosis peritonitis. *Gut* 1986; 27: 1049–53.
2 Voigt MD, Kalvaria I, Trey C, Berman P, Lombard C, Kirsch RE. Diagnostic value of ascites adenosine deaminase in tuberculous peritonitis. *Lancet* 1989; i: 751–4.
3 Dwivedi M, Misra SP, Misra V, Kumar R. Value of adenosine deaminase estimation in the diagnosis of tuberculous ascites. *Am J Gastroenterol* 1990; 85: 1123–5.
4 Bhargava DK, Gupta M, Nijhawan S, Dasarathy S, Kushwaha AKS. Adenosine deaminase (ADA) in peritoneal tuberculosis: diagnostic value in ascitic fluid and serum. *Tubercle* 1990; 71: 121–6.

Zinc deficiency in cirrhosis

SIR,—We read with interest the report by Goode *et al* (*Gut* 1990; 31: 694–9) on zinc status and hepatic functional reserve in patients with liver disease. We have reported hepatic zinc, copper, and magnesium concentrations in childhood cirrhosis, indicating that the serum concentrations do not always reflect the tissue concentrations of these trace elements. We have already indicated that the tissue zinc concentration decreases not only in cirrhotic children but also in patients with idiopathic portal hypertension.[1]

We have also reported that zinc is decreased in leucocytes of patients with cirrhosis, which affects their chemotactic function.[2] This function would be corrected when leucocytes are incubated with zinc (0·05 ml neutrophil mixture and 1 μg zinc) or control serum samples. Bactericidal function was shown to be appreciably decreased, as was nitro blue tetrazolium reduction and stimulated hexose monophosphate shunt activity of these cells.[3]

We have also shown (unpublished results) that zinc absorption is not greatly depressed but urinary zinc excretion is increased in patients with cirrhosis, which seems to be the basic cause of their zinc depletion.

Therefore we are in favour of zinc supplementation in these patients as suggested previously.[3 4]

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1 Göksu N, Özsoylu Ş. Hepatic and serum levels of zinc, copper and magnesium in childhood cirrhosis. *J Pediatr Gastroenterol Nutr* 1986; 5: 459–62.
2 Özsoylu Ş, Akgün N. Defective neutrophil motility. *Am J Dis Child* 1985; 139: 10.
3 Özsoylu Ş, Akgün N, Yetgin S, *et al.* Neutrophil functions in children with cirrhosis. *Turkish Journal of Research in Medical Sciences* 1987; 5: 403–6.
4 Reding P, Dechauteau J, Bataille C. Oral zinc supplementation improves hepatic encephalopathy. Results of randomised controlled trial. *Lancet* 1984; ii: 493–5.

BOOK REVIEWS

Morson and Dawson's gastrointestinal pathology. By B C Morson, I M P Dawson, D W Day, J R Jass, A B Price, and G T Williams. (Pp 748; illustrated; £19.) Oxford: Blackwell Scientific, 1990.

The new edition of Morson and Dawson will undoubtedly prove to be one of the 'best sellers' in the fields of gastrointestinal pathology, gastroenterology, and endoscopy. The volume is larger than its predecessors and there are now six authors, although individual responsibility for chapters is not specified. Documentation of disease entities is comprehensive and informative and the text is consolidated with a wealth of references and many new larger illustrations which clarify interpretation. The sections on inflammatory bowel disease, neoplasia, and infective colitis are superb. Vascular disorders of the small and large intestine are described in two separate chapters. A new section covering pathology of the peritoneum has been introduced towards the end of the text and this should be useful.

Lynch syndromes do not appear in the index, although they are discussed with references in the text. The role of antigliadin, antiendomysial, and antireticulin antibodies in the diagnosis and control of coeliac disease has probably been too recent to be included in view of the publication time. With reference to gastric dysplasia, the statement that 'severe dysplasia on its own is not an indication for surgical intervention' does not accord with current gastroenterological opinion. The entity of staphylococcal enteritis is challenged on the basis that *Clostridium difficile* infection cannot be excluded in documented cases but the authors overlook the fact that *C difficile* is predominantly a pathogen of the large intestine whereas staphylococcal pseudomembranous colitis involves the small intestine with predisposing factors such as hypovolaemia. Occasional other controversial views may stimulate publications in the relevant areas. The presentation, style, and content of this new multiauthor book are greatly improved, and I have no hesitation in recommending that this edition should be on the shelves of every department of histopathology.

H THOMPSON

Current hepatology (volume 10). Edited by Gary Gitnick. (Pp 357; illustrated; £60.) Chicago: Year Book Medical Publishers, 1990.

This is the 10th volume in this annual series edited by Gary Gitnick. The series aims to provide the reader with an overview of the world's published reports of the previous year in each of the major areas of liver disease. The need for annual reviews can be gauged from the introductory comments in the chapter on cirrhosis and portal hypertension, in which the authors refer to 500 articles pertaining to their topic, of which 139 were selected for review.

The reviews are generally well balanced, with a good mixture of science and clinical studies. Of particular note is the chapter reviewing Japanese reports. The remarkably increased incidence of hepatocellular carcinoma in that country and the prevalence of cryptogenic cirrhosis, or cirrhosis due to non-A, non-B chronic hepatitis, is a particular stimulus to workers in that country. So much of their data and the important scientific advances which have been made by them are published only in the Japanese journals. Many new advances are based on molecular biology, and for those who, like the author, have a struggle to keep up with the rapidly advancing techniques, the chapter on the molecular biology of liver disease can be thoroughly recommended. In recent years there has been a resurgence in the anatomy and pathophysiology of the liver, and the chapter reviewing recent advances here again makes very worthwhile reading.

This is a volume to be recommended to both clinical and research hepatologists.

ROGER WILLIAMS

Inflammatory bowel disease and coeliac disease in children. Edited by F Madziselimovic, B Herzog, A Burgin-Wolff. (Pp 199; illustrated; price not given.) Dordrecht, Netherlands: Kluwer Academic Publishers, 1990.

This slim volume records the proceedings of the International Falk Symposium on Paediatric and Surgical Gastroenterology held in Basel in 1989. The theme of the meeting, however, was restricted to inflammatory diseases of the gut and particularly Crohn's disease, ulcerative colitis, and coeliac disease. The book is divided into two sections. Section I is almost entirely devoted to Crohn's disease, and to a lesser extent ulcerative colitis, and section II is concerned with coeliac disease.

Section I makes a good start with a very readable summary of current understanding of the immune function of the gut and its postnatal development. The last part of the first chapter comments on the functional interaction between the immune system and nervous and endocrine systems. Potentially the most interesting area, it is disappointing in its

Gut: first published as 10.1136/gut.32.4.458-a on 1 April 1991. Downloaded from http://gut.bmj.com/ on December 16, 2022 by guest. Protected by copyright.