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***Helicobacter pylori* eradication and serum pepsinogens**

EDITOR,—We wish to comment on the article by Parente *et al* (*Gut* 1995; 37: 210–5) regarding clinical usefulness of functional parameters in duodenal ulcer patients cured for *Helicobacter pylori* infection.

We agree with the authors about the finding of a fall in pepsinogen I serum concentrations three months after the eradication of the infection and we would like to underline the relevance of such a non-invasive parameter already defined by Samloff as 'gastric mucosa serological biopsy'.¹ In our experience on 259 (197 M; mean age 52.2 years; range 18–79) duodenal ulcer patients tested two months after *H. pylori* eradication treatment pepsinogen I decreased from mean (SD) 147.7(78.3) to 90.7 (49.2) µg/ml ($p < 0.005$). On the contrary the authors found an increase in pepsinogen I serum concentrations in patients who were not cured, where we found, in the same group, a slight decrease: 145.6(80.7) *v* 124.6(63.4) ($p = \text{NS}$). We believe that this decrease, although not statistically significant, could be explained by the fact that even in the group who were not cured the antibiotic treatment reduces *H. pylori* concentration and consequently the gastric mucosa inflammation.²

We also would like to point out that in our experience on the same patient's sample, pepsinogen II (RIA Method, Sorin Biomedica, Saluggia, Italy; normal values 2.6–13 µg/dl) decreased dramatically after *H. pylori* infection treatment. Indeed in duodenal ulcer cured patients tested two months after the end of the treatment pepsinogen II decreased from 21.6(11.8) to 8.2(4.1) ($p < 0.001$). In agreement with our finding on pepsinogen I even pepsinogen II slightly decreased in patients not cured for *H. pylori* infection (22.7(13.6) *v* 16(7.2)). The possible explanation for these findings is that pepsinogen II is produced mainly by the prepyloric glands where usually the degree of inflammation is higher, because the infection is supposed to start in this gastric area as suggested by the bacterial name.

Using the ROC curves statistical analysis we showed that a decrease in pepsinogen II serum concentrations higher than 38% was the most sensitive (75.5%) and specific (74.5%) value in predicting *H. pylori* eradication with diagnostic accuracy of 75.2% among four parameters (gastrin, pepsinogen I, pepsinogen II, and IgG anti-Hp).³

Pepsinogen I and II were useful not only to verify the success of an eradication treatment but even in the follow up of cured patients. Indeed in a sample of 70 duodenal ulcer patients tested basally, at two, six, and 12 months after the end of a successful eradication therapy we found: pepsinogen I 151.7(71.3), 89.9(46.2), 85.6(40.7), 77.3(30.6); pepsinogen II 21.4(12.7), 8.7(4.9), 7.7(3.6), 7.3(3.4).

In conclusion we suggest the use of pepsinogens, especially pepsinogen II, in the clinical monitoring of patients treated for

H. pylori infection, as indirect and non-invasive parameters of gastric mucosa inflammation.

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Reply

EDITOR,—We are grateful to Di Mario *et al* for their letter concerning our article. We are completely in agreement with these authors concerning the fact that serum pepsinogen I and pepsinogen II may be regarded as markers of mucosal gastric inflammation in patients with *H. pylori* related gastritis, as their concentrations dramatically decrease after eradication of this bacterium and the resolution of gastritis, as recently shown by ourselves and other authors.^{1,2}

However, we wish to emphasise that serum pepsinogen I and pepsinogen II concentrations may also decline (although not dramatically) after antibiotic treatment in patients where *H. pylori* infection persists, probably as a consequence of reduced *H. pylori* density and gastric mucosal inflammation. We did not see such behaviour in our control group simply because 'our controls' consisted of patients with persistent *H. pylori* infection not receiving any antibiotic regimen but who had been given a short-term regimen with lansoprazole only. Indeed, the gastritis score of these patients, both at three and six months from the end of treatment, unlike cured patients, did not change significantly compared with the pretreatment value.

In contrast, we are not in agreement with Di Mario *et al* about the usefulness of serum pepsinogen I and pepsinogen II concentrations in clinical practice as reliable indirect markers of successful *H. pylori* eradication, especially in the short-term, for two major reasons: (a) as mentioned above, pepsinogen I and pepsinogen II concentrations may decrease after antibiotic treatment even in patients with persistent *H. pylori* infection, as a consequence of reduced *H. pylori* density and gastric mucosal inflammation. Therefore, the finding of a decline in concentrations of serum pepsinogens immediately after treatment has a low specificity in terms of predicting *H. pylori* eradication; indeed, the diagnostic accuracy of this test reported by Di Mario *et al* in their own experience is fairly low (75%); (b) multiple samplings are necessary to record a consistent and persistent decrease in serum pepsinogen I and pepsinogen II concentrations related to *H. pylori* eradication (at least the baseline value and those at two and six months after the end of the treatment should be determined). The

test is therefore not competitive in terms of cost/accuracy with other non-invasive tests (for example, C13-UBT or C14-UBT), if we consider that, in Italy, each pepsinogen I determination costs roughly the equivalent of £11 (with a double assay for each sample) and pepsinogen II determination is even more expensive, whereas C13-UBT (which is undoubtedly the most accurate non-invasive test for diagnosing *H. pylori* infection) costs £18–25 depending on the number of tests performed.

To summarise, we believe that serum pepsinogen I and pepsinogen II concentrations are indices of gastric mucosal inflammation in patients with *H. pylori* related gastritis, but, like serum Ig-anti *H. pylori*, they cannot be used in clinical practice as reliable markers of successful *H. pylori* eradication.

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BOOK REVIEWS

Hepatobiliary and Pancreatic Disease. Edited by H A Pitt, D L Carr-Locke, J T Ferrucci. (Pp 496; illustrated; £95.) Boston: Little, Brown, 1995. ISBN 031-6709-158.

The editors of this comprehensive text, which concerns the diagnosis and management of 42 major hepatic and pancreatic problems, point out in their preface that subspecialisation has now occurred in all of the disciplines involved in the care of patients with hepatobiliary disorders, including surgery, gastroenterology, radiology, and pathology. In this book they have attempted to integrate the opinions of these specialists in discussions of current management and treatment options. The invited authors were asked not to avoid controversies in their chapters but to provide a balanced view of the available options and to arrive at a consensus view of the preferred approaches to patient care. The editors stated in the preface that they believed that with this format the book 'should present a unique contribution to the literature'.

The editors invited 132 specialists to produce a text that is only 486 pages in length. The hepatic, biliary, and pancreatic topics are very comprehensive in their range although they mostly exclude reference to paediatric liver disease, which was probably regarded as a subspecialty in its own right. The exception is the inclusion of a chapter on biliary atresia. The care of patients after liver transplantation, which would surely have been of great interest to any aspiring hepatologist, has been omitted as a topic.

The first chapter, which is devoted to 'Hepatitis', contains some very low key statements such as '... the surgeon plays a critical role when medical therapy is no longer possible ...' and 'The clinical immunologist is responsible for performing the many immunologic assays required to identify and characterise the different forms of chronic hepatitis.' This chapter might be satisfactory for undergraduate teaching but is not detailed enough for trainee specialists. In this and in some of the subsequent chapters it was not always clear which readership the editors had in mind when they started the project.

Chapter two, on hepatic failure, is also rather disappointing. A discussion of pathogenesis is confined to just one column and the text includes unhelpful phrases such as 'Transaminases are usually elevated, often dramatically', and 'Thus the transplant team needs to be involved in a timely way for those patients deemed to be candidates'. These comments again suggested that the editors had not defined the level of expertise they expected from their readership and this was again evident in chapter 4 on portal hypertension. In this chapter there is considerable emphasis on the technique and indications for transjugular intrahepatic portosystemic shunt (TIPS) procedures but little on the details of more conventional surgical procedures. There is no analysis of the problems of extrahepatic portal hypertension in which neither TIPS or transplantation are options, and no discussion on the value of devascularisation procedures in the bleeding patient.

The concept of inviting such a large number of contributors was very ambitious but this may have been the cause of some of the inconsistencies. Drug dosages, for example, are detailed in some chapters (hepatic failure), but are completely absent in others (portal hypertension). Surgical techniques are described extensively in some sections (hydatid disease) but are absent in others. Most of the important inconsistencies are found in the first few chapters and if the book was intended for fellow hepatologists then in my opinion the data (and the references) should have been much more complete.

However, the later chapters, especially those concerning pancreatic disease, are very good overviews on management and I particularly liked those on pancreatitis. Therefore despite my reservations I think that the book can be recommended as a useful addition to the library of the trainee gastroenterologist or surgeon.

E R HOWARD

Longitudinal Muscle in Esophageal Disease. By O Arthur Stiennon. (Pp 300; illustrated; \$150.) Maddison, Wisconsin: WRS Press, 1995. ISBN 0-9644 594-0-X.

This is an interesting book that I enjoyed reading, because unlike 99% of medical textbooks, the author challenges conventional views and makes the reader think. I am certainly persuaded that the function of the oesophageal muscle has been largely ignored, compared with other components thought to play a part in oesophageal disease and the radiological evidence of longitudinal muscle function, seems quite compelling.

The book is written in dogmatic style, rather like a lawyer making a case for the prosecution. He chooses to ignore the

'defence' evidence and this is both the strength and weakness of the text. The basis of Dr Stiennon's hypothesis is that the longitudinal muscle of the oesophagus is somehow responsible for just about every benign oesophageal disorder. I suspect that he is right about longitudinal muscle function and the subsequent effects of oesophageal shortening. Everything seems fine until the 'lawyer' has to sum up for the jury at the end of each chapter. It is here that the author strays from the evidence and in particular that which does not fit with his theory. The chapter on Barrett's oesophagus is particularly short sighted and uses the most tenuous literature review to support the author's notion that this is yet another condition explained entirely by longitudinal muscle action and that the Barrett's segment is no more than a cylinder of stomach, pulled up by the longitudinal muscle of the oesophagus.

While I agree with the author's scepticism regarding current theories of the pathogenesis of achalasia, I find them no more bizarre than his interpretation that diffuse oesophageal spasm and achalasia are manifestations of undiagnosed obstruction due to incarceration of the gastric fundus. I would be interested to know how patients with diffuse oesophageal spasm get their symptoms at night, when motor function is largely quiescent. It is when the author strays into the field of surgery, that he really gets my back up. Most of his comments about anti-reflux surgery and surgery for achalasia, are so misplaced that one wonders if he has ever read any of the surgical literature on the oesophagus in the past 40 years! There is no shortage of literature for instance, testifying to the poor performance of the Allison repair in the long term and by Dr Stiennon's theories, all Nissen funduplications should result in gas-bloat syndrome. No explanation is offered why over 95% of these operations do not produce the syndrome.

This book should be read by 'oesophagologists', if only to challenge their existing views. I cannot recommend it, however, to a wider audience as a balanced account of the role of the longitudinal muscle in the pathogenesis of oesophageal disease. The author claims that many of our beliefs about oesophageal function and disease, stem from preconceived notions. He uses the analogy of 'The Flat Earth Society' to condemn these views as two dimensional in a three dimensional organ. I would remind readers, however, of T H Huxley's aphorism, that 'The great tragedy of science is the slaying of a beautiful hypothesis by an ugly fact'. At the moment, there are too many ugly facts to support Dr Stiennon's hypothesis.

D A ALDERSON

17, B-1180 Brussels, Belgium. Tel: (32) 2 375 16 48; fax: (32) 2 375 32 99.

Abdominal imaging

A course on Abdominal Imaging will be held on 1-5 July 1996 in Wyoming, USA. Further information from: Janice Ford Benner, University of Pennsylvania Medical Center, 3400 Spruce Street, 1 Silverstein Building, Philadelphia, PA 19104, USA. Tel: (215) 662-6904; fax: (215) 349-5925.

Gastrointestinal pharmacology

A meeting sponsored by the Gastrointestinal (GI) Pharmacology Section of IUPHAR (International Union of Pharmacology) will be held 3-7 September 1996 in Sperlonga, Italy. The meeting is entitled New Approaches to Pharmacotherapy for Gastrointestinal Ulcerative and Inflammatory Disorders. Further information from: Dr Antonio Guglietta, Parke-Davis Research, 2800 Plymouth Road, Ann Arbor, MI, 48105, USA. Tel: (313)-996-2911; fax: (313)-996-4333; e-mail: guglietta@aa.wl.com.

Clinical nutrition

The Leeds Course in Clinical Nutrition will be held on 3-6 September 1996. Further details from: Mrs Hilary L Thackray, Department of Continuing Professional Education, Continuing Education Building, Springfield Mount, Leeds LS2 9NG. Tel: 0113 233 3233; fax: 0113 233 3240.

Inflammatory bowel disease

An International Meeting on Inflammatory Bowel Disease will be held in Capri, Italy, on 19-21 September 1996. Further information from: Professor Renzo Caprilli, Cattedra di Gastroenterologia, Università de L'Aquila, Via S Sisto 22E 67100 L'Aquila, Italy. Tel: 39 862 433407; fax: 39 862 433383.

CORRECTION

Correction

An authors' error occurred in the paper by Dr R J Scott and others (*Gut* 1995; **36**: 731-6). The mutation described at nucleotide 5934, codon 1978 should be nucleotide 5960, codon 1987 causing a stop codon 61 codons later as shown in the accompanying Figure.

5934	5960	5973
-GAC-CAA-GAA-AAC-AAC-AAT-AAA-GAA-AAT-GAA-		
	↓	
5934		5973
-GAC-CAA-GAA-AAC-AAC-ATA-AAG-AAA-ATG-AAC-		

Figure 3: Sequence analysis of exon 15n showed a base pair deletion at position 5960, which was identified in patients who presented with colonic disease alone or extracolonic disease with or without colonic disease.

NOTES

Digestive endotherapy

The XIVth European Workshop on Digestive Endotherapy will be held on 18-20 June, 1996 in Brussels. Further information from: Administrative Secretariat, Conference Services sa, Avenue de l'Observatoire, 3, Box