became available in seven of the DU patients. No specific mention was made of corrections for pyloric losses or duodenal reflux. Their chief findings were statistically significant falls in basal, maximal, and GRP stimulated secretion in the seven patients with dual tests. They suggest that *H pylori*, by increasing sensitivity to gastrin, increases parietal cell mass and that this is the cause of the well known increased maximal acid output in DU patients compared with controls.

This suggestion is at odds with the paper¹ from this department that demonstrated that *H pylori* positive subjects, both patients with non-ulcer dyspepsia and patients with DU, had lower maximal gastric secretion (in response to continuous intravenous histamine) than *H pylori* negative patients of the corresponding diagnostic groups. Our study was performed in 64 subjects. Corrections were made for pyloric losses and duodenogastric reflux (both especially important when low secretion rates or small differences in secretion rates are being measured).

The GRP stimulated responses seen by Harris *et al* were clearly submaximal, because they were only 40–70% of the pentagastrin responses. The latter were also possibly submaximal (intramuscular route, one shot injection). Therefore it is possible that the phenomenon they have described (if it is confirmed in a larger number of patients with correction of collection errors) represents an effect of *H pylori* that primarily reduces parietal cell mass, and thereby increases sensitivity to gastrin (GRP or pentagastrin). This alternative hypothesis was discussed and referenced in our article.¹ The important point is that the findings described by Harris *et al* do not necessarily indicate that *H pylori* increases the parietal cell mass.

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- 1 Chandrakumaran K, Vaira D, Hobsley M. Duodenal ulcer, *Helicobacter pylori* and gastric secretion. *Gut* 1994; 35: 1033–6.

Reply

EDITOR,—We thank Dr Oshowo and Professor Hobsley for their interesting comments.

We fully accept that *H pylori* may produce gastric hyposecretion from atrophic changes in body mucosa with loss of parietal cells. We believe that *H pylori* can also produce gastric hypersecretion leading to duodenal ulcer disease, but it is unclear which host or microbial factor is responsible for the one or other pathology.

Six months after eradication of *H pylori*, pentagastrin-stimulated peak acid output (PAO_{PG}) significantly decreased in all our eight patients, and not in seven as stated in the letter. It was the basal (BAO) and gastrin releasing peptide-stimulated (PAO_{GRP}) acid outputs that were available in only seven of eight patients. We did not make Hobsley's corrections for pyloric losses or duodenogastric reflux, but such corrections would probably have been slight at the high rates of secretion in patients with duodenal ulcer.¹ There are reasonable data that measurement of PAO after one injection of 6 µg/kg of pentagastrin is comparable with intravenous infusion.¹

Oshowo and Hobsley state that we 'suggest that *H pylori*, by increasing sensitivity to gastrin, increases parietal cell mass'. We actually suggested that 'the increased PAO_{PG} (an indirect measure of the parietal cell mass) in *H pylori* positive patients with duodenal ulcer may be caused by a combination of exaggerated gastrin release and acid response to gastrin' and not purely as a result of a change in sensitivity to gastrin.

Our finding of reduction of PAO_{PG} after eradication of *H pylori* has since been confirmed^{2,3} and we look forward to Professor Hobsley's study of his patients with duodenal ulcer after eradication of *H pylori* with 'a large number of patients with correction of collection errors'.

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- 1 Baron JH. *Clinical tests of gastric secretion*. London: Macmillan Press, 1978.
- 2 Jacobson K, Chiba N, James C, Armstrong D, Barrientos M, Hunt RH. Protracted gastric acid secretion in *H pylori* positive DU patients following eradication. *Gastroenterology* 1995; 108: A122.
- 3 Parente F, Maconi G, Sangaletti O, Minguzzi M, Vago L, Bianchi Porro G. Behaviour of acid secretion, gastrin release, serum pepsinogen I, and gastric emptying of liquids over six months from eradication of *Helicobacter pylori* in duodenal ulcer patients. A controlled study. *Gut* 1995; 37: 210–5.

Ultrasound for gastroenterologists

EDITOR,—I read with interest the leading article by Dr Derrick Martin (*Gut* 1996; 38: 479–80) in which I believe he makes a very good case for not training gastroenterologists in ultrasound.

The argument in favour of training gastroenterologists is, I believe, a weak one, based on an increasing waiting time for ultrasound in radiology departments, the requirement of the European Union of Medical Specialities, and the perceived attractiveness of an 'instant' diagnosis at the first outpatient clinic attendance.

These arguments are far outweighed by many reasons in favour of maintaining and constantly improving radiology services. As a radiologist, Dr Martin concedes that ultrasound is 'a difficult technique, technical artefacts in the image abound and can create diagnostic confusion'. What defence is there when a renal tumour is missed, an aortic aneurysm unrecognised, an ovarian cyst undetected or pleural effusions not looked for? The high level of training of the radiologist enables him or her to search routinely for all of these potential pitfalls. Dr Martin states that many radiologists are keen to train others. This is certainly true but there is scant evidence that they are keen to train gastroenterologists. Without doubt, training would require to be thorough and systematic.

The leading article also deals with equipment and with the need to make efficient use of expensive resources. 'You get what you pay for' is certainly true and high quality ultrasound requires the appropriate level of investment in equipment. How is it possible therefore to justify the purchase of expensive ultrasound equipment for the gastroenterology clinic based on a throughput of perhaps 20 cases per week (or less!), which is equivalent to two radiology ultrasound sessions per

week. Dr Martin readily acknowledges the unpredictable nature of the gastroenterology workload and the variable commitments of those intending to use ultrasound. This is a further good reason for focusing ultrasound in the radiology department with its dedicated centralised staff and resources. There have been obvious precedents in which hospitals have accumulated expensive ultrasound equipment outwith the radiology department in renal units, urology departments, cardiology units and stroke units, which stands unused for most of each week. Furthermore equipment located outwith the radiology department runs the risk of infrequent or inadequate maintenance. Excellent hard copy facilities are a key factor in image quality and it is unlikely that the high cost of expensive laser printers could be justified outwith the radiology department.

Formal reporting of ultrasound images has many pitfalls and the gastroenterologist would be at particular risk of meeting one or more of these.

The issue of waiting lists in radiology departments is an important one. Whenever a Trust makes a clinical appointment, it needs to ask a question "What service department (including radiology) consequences will this appointment inevitably bring with it?"

There is an increasing tendency within radiology departments towards subspecialisation, particularly in teaching hospitals. Properly controlled, this is a beneficial development, because it encourages improved skills and concentrates experience in a focused way, which benefits patient care, education and research, for example in interventional radiology, magnetic resonance, computed tomography, and not least ultrasound where biopsy techniques, power Doppler, and tissue characterisation are most likely to flourish in the hands of dedicated radiologists. It would be completely inappropriate for me to have any involvement in breast screening mammography without extensive training in it and a major ongoing commitment to it. Training physicians and surgeons in ultrasound operates in the opposite direction to this focused specialised approach. It is not sufficient to simply say 'there will be some enthusiasts with sufficient workload to support this service and they will need guidance and training'.

The decree of the European Board of Gastroenterology that training in ultrasound should be included is not necessarily a wise one and in any case is probably relevant to comparatively few UK gastroenterologists.

Elsewhere¹ Dr Martin highlights the benefits of the patient focused approach to diagnosis and therapy, but surely the way forward is to focus ultrasound services within the imaging department ensuring that the highest possible standard is established and maintained with respect to rapid, accurate and complete diagnosis, instant reporting, education, and research.

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- 1 Martin DF. Ultrasound training for non-radiologists. *Clin Radiol* 1995; 50: 589–92.

Reply

EDITOR,—I am grateful to Dr Vallance for his comments, as this is clearly a topic where debate is essential if we are to reach a logical conclusion that allows the fulfilment of ambi-

tion for both radiologist and gastroenterologist, as well as providing a prompt service with appropriate quality.

In my heart I know that the comments by Dr Vallance are correct when he highlights the potential difficulties and drawbacks that may result from ultrasound examinations being undertaken by practitioners other than skilled trained radiologists. In my head, however, I recognise that there are other arguments that must be resolved. Some gastroenterologists and indeed clinicians in other specialties, do genuinely wish to provide an ultrasound service and this wish has been given validity by the declaration of the European Board of Gastroenterology that ultrasound should be part of the training programme for gastroenterologists. Neither radiologists individually nor collectively through their Royal College, are in a position to prevent non-radiologists from using ultrasound for diagnosis and therefore we find ourselves in a position in which we can adopt one of three responses.

Radiologists can ignore the needs of non-radiologists to train in ultrasound and decline to be involved in such training. In doing this we will deny to non-radiologists an understanding of our own skills, which contribute to a quality service and we may thus contribute to the poor quality non-radiological service which we fear.

Radiologists may believe that because non-radiological ultrasound training is in vogue, that we should simply keep our heads down for a while in the hope that the fashion will pass, but again we risk the development of a second rate service without our contribution.

Thirdly, we could permit our non-radiologist colleagues free access to our skills. Of course we do this already by providing a service for them and their patients, but we should extend this to the provision of training, so that the service which non-radiologists provide is of sufficient standard to fulfil radiologists' definition of quality. Additionally the links forged between radiologist and non-radiologist during training should contribute to more open communication, which must be to the ultimate benefit of our patients.

I believe that the responsible and logical reaction of radiologists to the wishes of some non-radiologists to train in ultrasound, is to provide them with sensible achievable guidelines for training so that radiologist and non-radiologist can work together to provide what the patient needs, a rapid ultrasound service of appropriate quality.

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Peptide YY and electrolyte homeostasis

EDITOR,—We wish to comment on the article by Nightingale and coworkers on the possible importance of peptide YY as the mediator of the colonic brake to gastric emptying (*Gut* 1996; 39: 267–72). Their studies suggest that the lower concentrations of peptide YY seen in patients without a colon are associated with an increased rate of gastric emptying. This may well contribute to the increased jejuno-ostomy output seen in these patients. We have previously shown in humans that peptide YY in volunteers with jejuno-ostomies (infused at physiological postprandial values) causes

significant reduction in water and electrolyte secretion and slows small intestinal transit.¹

The high output of jejuno-ostomy fluid postprandially is therefore likely to be due to a combination of increased gastric emptying, decreased small intestinal fluid absorption, and increased small bowel motility. The development of orally active peptide YY agonists and antagonists will provide further insight into the physiological role of this peptide and could provide a novel approach to the water and electrolyte disturbances associated with high ileostomy outputs. Physiological concentrations of peptide YY do, however, cause significant increase in sodium excretion by the kidney and the relative benefits of electrolyte conservation by the intestine might be counterbalanced by the increased electrolyte lost by the kidney.²

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1 Playford RJ, Domin J, Beecham J, Parmar KI, Tatemoto K, Bloom SR, *et al.* Peptide YY: a natural defence against diarrhoea. *Lancet* 1990; 335: 1555–7.

2 Playford RJ, Moss SM, Calam J, Unwin R. Effect of Peptide YY (PYY) on renal function. *Am J Physiol* 1995; 268: F754–9.

Reply

EDITOR,—We are grateful to Professor R J Playford and Dr C E MacDonald for drawing our attention to this research. It supports the view that peptide YY is important in the short bowel syndrome. While the main reason for a high output from a jejuno-ostomy is the loss of the normal daily secretions produced in response to food, low plasma concentrations of peptide YY may exacerbate the situation by increasing the rate of liquid gastric emptying and small bowel transit,¹ and may increase water and electrolyte secretion. High plasma concentrations of peptide YY occur in patients with a short bowel and a retained colon, thus it is beneficial to preserve some colon when a major bowel resection is performed.

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1 Nightingale JMD, Kamm MA, van der Sijp JRM, Morris GP, Walker ER, Mather SJ, *et al.* Disturbed gastric emptying in the short bowel syndrome. Evidence for a 'colonic brake'. *Gut* 1993; 34: 1171–6.

DM 248.) Stuttgart: Georg Thieme Verlag, 1996. ISBN 3-13-104331-8.

Books reporting proceedings of meetings are often hurried out with little editorial control and patchy quality. This book is an exception. To begin with the book was a pleasure to handle; an attractive cover, solidly bound and on high quality paper. A glance through it revealed abundant illustrations, tables, and uniformity of style. Dipping into the book and reading in more depth revealed a systematic authoritative and in depth coverage of the molecular biology of pancreatic diseases, acute pancreatitis, chronic pancreatitis, and pancreatic cancer.

On the basis that it is a reviewer's privilege to select items for commendation or condemnation (I have nothing to offer here) I will select a few gems from the text. Weber and Jensen have given a masterly insight into the genetic and molecular biological aspects of pancreatic endocrine and carcinoid tumours. No more will one be baffled by the intricacies of the MEN 2 gene mapping nor genetic alterations in carcinoid tumours. They were simply illuminated in this excellent contribution. Acute pancreatitis was tackled by physicians, surgeons, gastroenterologists and radiologists in a comprehensive manner. The more recent controversies of artificial nutrition and antibiotics were completely up to date with a total coverage of the world literature and reasoned, balanced discussions. The surgeons perhaps were liberal with dogma, painting a picture of surgical options with little room for manoeuvre, which in the individual case is often not tenable.

This reviewer learnt a lot from reading the various sections on chronic pancreatitis and any student of this condition callow or seasoned should read it thoroughly for its comprehensive coverage of the literature and discussions.

The Japanese contributors at last gained exposure contributing an exhaustive analysis of the European versus the Japanese approach to surgery in this condition. In my view the star chapter was that of Hermance describing the pathology of pancreatic cancer culminating in the new WHO classification.

Who should buy this book? It is relevant to the practice of any serious pancreatologist, it is an essential reference for any researcher in the field and the editor is to be congratulated.

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NOTE

Abdominal imaging

A course on abdominal imaging will be held at Jackson Hole, Wyoming, Grand Teton National Park, USA, on 30 June–4 July 1997. 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.

BOOK REVIEW

Advances in Pancreatic Disease, Molecular Biology, Diagnosis and Treatment. Edited by C G Dervis. (Pp 401; illustrated;